UNIVERSITAT ROVIRA I VIRGILI

THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.

Estefania Aparicio Llopis

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INTERNATIONAL DOCTORAL THESIS

Supervised by Dr. **Victoria Arija Val** and
Dr. **Josefa Canals Sans**

Department of Basic Medical Sciences

UNIVERSITAT ROVIRA I VIRGILI

Reus, 2015

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I STATE that the present study, entitled “The effect of emotional and genetic factors on nutritional status in a school-based population” presented by Estefania Aparicio Llopis for the award of the degree of Doctor, has been carried out under my supervision at the Department of Basic Medical Sciences of this university.

Reus, 25th June 2015

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A mis padres

A Adrián

A Runa

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Abstract

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ABSTRACT

Background: Emotional psychopathology and obesity pose a major threat to public health, especially among children and adolescents. Although previous studies have suggested that early emotional psychopathology may lead to weight gain or obesity, the potential role of psychological and emotional distress in obesity development has been less extensively researched. In this vein, the relation between emotional symptoms and dietary pattern is quite clear, especially in population-based studies during early adolescence. A few studies have also suggested that some genetic factors could moderate this relation.

Main objective: To assess the effect of emotional psychopathology on dietary intake and adiposity in a school-based population from preadolescence to adolescence, according to the gender. We examine the influence of genetic factors on this relationship.

Methodology: A three-year longitudinal study was conducted with a baseline sample of 1,514 schoolchildren. We selected a risk sample of emotional symptoms and a free-risk sample of emotional symptoms. Of these subjects, 242 adolescents were followed-up and classified as showing emotional symptoms during the study or in the control group. Depression and anxiety, as well as anthropometric (BMI and waist circumference, (WC)) and body composition parameters by bioelectrical impedance (percentage of body fat (%BF)) were assessed at baseline and follow-up phase. In the follow-up phase, food consumption was recorded and dietary patterns were created by principal component analysis. Tests of quality of diet (Mediterranean diet) and physical activity were also administered, and a saliva sample was also collected from the participants, and DNA was extracted for subsequent analyses of the Monoamine A (MAOA), classified as low-activity MAOA (MAOA-L) high-activity MAOA (MAOA-H); and serotonin transporter polymorphisms (5-HTTLPR) classified as long 5-HTTLPR (LL) and short

5-HTTLPR (SS/SL). Multiple linear and adjusted logistic models were performed to assess the effect of emotional symptoms on dietary intake and weight status, and several mediational and interaction models were developed.

In addition, we developed a conceptual framework of the role of emotion regulation on the prevention and treatment of childhood obesity. To that end, a narrative review was conducted by means of an electronic database search (MEDLINE, Web of Knowledge and Scopus) of the observational and interventional/experimental literature on emotion regulation and obesity and the underlying concepts, and emotional regulation intervention techniques.

Results: 39.7% of girls with emotional symptoms during early adolescence showed high adherence to a sweet and fat dietary pattern. Based on adjusted logistic regression, girls with emotional symptoms were four times as likely to have a high adherence to a dietary pattern of sweet and fatty foods (OR: 4.79, 95%CI(1.55-15.10). No differences were observed among boys. In addition, depressive symptoms were linked to a risk of low adherence to the Mediterranean diet (OR=1.069, p=0.021) in adolescence. However, low Mediterranean diet adherence was not a mediator between depressive symptoms and overweight/obesity.

Regarding weight status, the results of analysis with adjusted multiple regression models indicated that symptoms of depression and separation anxiety were significantly associated with increased WC and BMI in boys, and that somatic symptoms were associated with increased WC and %BF in girls. A diagnosis of social phobia, panic disorder or dysthymia led to significantly increased WC and/or BMI in boys, and dysthymia increased WC in girls.

As for genetic factors, in girls the presence of MAOA-H polymorphism, together with emotional symptoms, was associated with increase sweet

and fatty food pattern adherence, reduced Mediterranean diet adherence and physical activity, as well as a higher percentage of body fat. However, in boys the presence of MAOA-H together with emotional symptoms was associated with lower BMI and WC values. Furthermore, and only in girls, the SS/SL variant of the 5-HTTLPR polymorphism was associated with a higher adherence of sweet and fatty dietary pattern in adolescence. This association was observed in the presence of emotional symptoms and without emotional symptoms.

In addition, our conceptual framework model indicates that childhood emotional regulation is a link between stress and obesity. Stress and ineffective emotion regulation leads to abnormal cortisol patterns, emotional eating, a sedentary lifestyle and sleep problems. By contrast, effective emotion regulation skills reduce obesity-related unhealthy behaviour patterns and enhance protective factors, which boost health. The literature contains some observational studies of children but very few intervention studies.

Conclusions: During adolescence, the presence of emotional symptoms and genetic factors, together with socioeconomic status, has an influence on nutritional status, mainly among girls, pushing them towards unhealthy behaviors related to obesity. Emotional psychopathology in preadolescence is associated with increased weight gain and abdominal fat in adolescence, albeit with some differences in the precise relationship with each anxiety and depression disorder according to gender. Encouraging an emotion regulation could therefore be an effective new approach, as well as a nutritional and physical activity intervention, in the early prevention and treatment of childhood obesity.

UNIVERSITAT ROVIRA I VIRGILI
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Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

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THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Index

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

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THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

INDEX

ABBREVIATIONS 9

INTRODUCTION 13

1. CHILDHOOD: ADOLESCENCE CRITICAL STAGE OF THE DEVELOPMENT 13

2. EMOTIONAL PROBLEMS 15

2.1 Definition 15

2.1.1 Depression 15

2.1.2. Anxiety 17

2.2 Epidemiology of emotional disorders in childhood 21

2.3 Health consequences and effect on eating behaviour 24

3. FOOD CONSUMPTION AND DIETARY PATTERNS 27

3.1 Food consumption and physical activity in adolescence 27

3.2 Methods of food consumption assessment 31

3.2.1 Food frequency questionnaire 32

3.2.2 24-hour dietary recall 35

3.2.3 Dietary record 37

3.2.4 Diet history 39

3.2.5 Screener and brief assessment methods 41

3.3. Dietary assessment in children and adolescents 43

3.4 Dietary patterns 45

3.4.1 Score-based methods *a priori* 46

3.4.2 Data driven approaches *a posteriori* 47

3.5 Dietary reference intake 49

4. OBESITY AND OVERWEIGHT 54

4.1 Definition 54

4.2 Epidemiology of obesity and overweight in childhood 54

4.3 Methods of obesity and overweight assessment 55

4.4 Consequences of obesity 59

4.5 Risk factors 61

Index

5. RELATION BETWEEN EMOTIONAL SYMPTOMS AND NUTRITIONAL STATUS 62
5.1 Emotional symptoms and food consumption 62
5.2 Emotional symptoms and weight gain 63
5.3 Physiological mechanisms linking emotional symptoms to obesity 69
6. GENETIC FACTORS, EMOTIONAL SYMPTOMS AND NUTRITIONAL STATUS 70
6.1 Monoamine oxidase-A polymorphism: MAOA 71
6.2 Serotonin transporter polymorphism: 5-HTTLPR 75
6.3 Interaction of genetic factors and emotional symptoms and nutritional status 78

HYPOTHESIS AND OBJECTIVES 83

MATERIAL AND METHODS 87
1. STUDY DESIGN 87
2. PARTICIPANTS 88
3. PROCEDURE 89
4. INSTRUMENTS 91
4.1 Psychopathology assessment 91
4.2. Food consumption, diet quality and physical activity 93
4.3 Obesity assessment 94
4.4 Socio-economic status 96
4.5 DNA extraction and genotyping 97
4.6 Summarize of variables in the study 100
5. NARRATIVE REVIEW 102
6. STATISTICAL ANALYSIS 103

RESULTS 111
1. DESCRIPTIVE CHARACTERISTICS OF PARTICIPANTS 111
1.1 Descriptive characteristics of the baseline sample 111
1.1.1 Socio-demographic characteristics 111
1.1.2 Psychological characteristics 112
1.1.3 Anthropometric and body composition characteristics 113
1.2 Descriptive characteristics of the follow-up sample 115

Index

1.2.1 Socio-demographic characteristics 115
1.2.2 Psychological characteristics 116
1.2.3 Anthropometric and body composition characteristics 119
2. EFFECT OF EMOTIONAL SYMPTOMS ON DIETARY INTAKE AND PHYSICAL ACTIVITY 121
2.1 Food consumption and energy and nutrient intake 121
2.1.1 Description of food consumption, and energy and nutrient intake 121
2.1.2 Association between emotional symptoms and food consumption, energy and nutrient intake 126
2.2 Dietary patterns 131
2.2.1 Description of dietary patterns 131
2.2.2 Association between emotional symptoms and dietary patterns 134
2.3 Mediterranean diet adherence and physical activity 137
2.3.1 General characteristics of Mediterranean diet adherence and physical activity 137
2.3.2 Association between emotional symptoms and Mediterranean diet adherence and physical activity 137
2.3.3 Psychological factors influence on Mediterranean diet adherence 139
2.3.4 Mediational analyses between socioeconomic status, depressive symptoms, Mediterranean diet and overweight/ obesity 142
3. EFFECT OF EMOTIONAL SYMPTOMS ON ANTHROPOMETRY AND BODY COMPOSITION 145
3.1 Cross-sectional association between emotional symptoms and anthropometric and body composition parameters in the baseline sample 145
3.2 Association between emotional symptoms and anthropometric and body composition parameters in the follow-up sample 148
3.3 Psychopathological predictors of adiposity 152
4. EFFECT EMOTIONAL SYMPTOMS ON DIETARY PATTERNS AND ANTHROPOMETRY AND BODY COMPOSITION ACCORDING TO GENETIC FACTORS 157
4.1 Allele and genotype distribution of MAOA and 5-HTTLPR polymorphism 157
4.2 Genetic factors and emotional symptoms 159
4.3 Genetic factors and dietary patterns 161
4.3.1 Genetic factors and dietary patterns 161

4.3.2 Association between emotional symptoms and dietary patterns according to genetic factors 163
4.3.3 Effect of emotional symptoms on dietary patterns according to genetic factors 168
4.4 Genetic factors and anthropometric and body composition parameters 172
4.4.1 Genetic factors and anthropometric and body composition parameters 172
4.4.2 Association between emotional symptoms and anthropometric and body composition parameters according to genetic factors 176
4.4.3 Effect of emotional symptoms on the change in anthropometric and body composition parameters according to genetic factors 178
5. NARRATIVE REVIEW: THE ROLE OF EMOTION REGULATION IN THE PREVENTION AND TREATMENT OF CHILDHOOD OBESITY 182

DISCUSSION 191
1. DESIGN, PARTICIPANTS AND METHODS 191
2. MAIN CHARACTERISTICS OF PARTICIPANTS IN BASELINE AND FOLLOW-UP PHASE 192
3. EFFECT OF EMOTIONAL SYMPTOMS ON DIETARY INTAKE AND PHYSICAL ACTIVITY 197
4. EFFECT OF EMOTIONAL SYMPTOMS ON ANTHROPOMETRIC AND BODY COMPOSITION PARAMETERS 203
5. EFFECT OF GENETIC FACTORS AND EMOTIONAL SYMPTOMS ON NUTRITIONAL STATUS 208
5.1 MAOA polymorphism 208
5.2 5-HTTLPR polymorphism 212
6. ROLE OF EMOTION REGULATION IN PREVENTION AND TREATMENT CHILDHOOD OBESITY 217
7. LIMITATIONS 220
7.1 Follow-up study limitations 220
7.2 Narrative review limitations 222
8. IMPLICATIONS AND FUTURE RESEARCH LINE 223

CONCLUSIONS 227

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

REFERENCES 233

SCIENTIFIC CONTRIBUTIONS 275

Scientific contributions related to the thesis 275

Other scientific contributions 303

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

UNIVERSITAT ROVIRA I VIRGILI
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Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Abbreviations

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

ABBREVIATIONS

APA: American Psychiatric Association

AI: Adequate Intake

BASS: Body Areas Satisfaction Scale

BMI: Body Mass Index

CDI: Children's Depression Inventory

DRI: Dietary Reference Intake

DSM: Diagnosis Statistical Manual of Mental Disorders

EAR: Estimated Average Requirement

EDI-2: Eating Disorder Inventory-2

EER: Estimated Energy Requirements

FAO: Food and Agriculture Organization

HWE: Hardy–Weinberg equilibrium

ICD: International Classification of Diseases

INA: Index of nutritional adequacy

IOM: Institute of Medicine

MAOA: Monoamine oxidase-A

MAOA-uVNRT (or MAOA polymorphism): MAOA variable number of tandem repeats polymorphism

MAOA-L: Low-activity MAOA polymorphism

MAOA-H: High-activity MAOA polymorphism

MINA: Mean of index of nutritional adequacy

MINI-Kid: MINI-International Neuropsychiatric Interview for Kids

PCR: Polymerase chain reaction

RDA: Recommended Dietary Allowance

SCARED: Screen for Childhood Anxiety and Related Emotional Disorders

SES: Socioeconomic status

SLC6A4: Serotonin transporter gene

UL: Tolerable Upper Intake Level

WHO: World Health Organization

YI-4: Youth's Inventory-4

5-HT: Serotonin

5-HTTP: 5-Hydroxytryptophan

5-HTT: Serotonin transporter

5-HTTLPR: 5-HTT-Linked Polymorphic Region

5-HTTLPR SS/SL: 5-HTTLPR with short alleles or short/long alleles

5-HTTLPR LL: 5-HTTLPR with long alleles

UNIVERSITAT ROVIRA I VIRGILI
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Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

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Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

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Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Introduction

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Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

INTRODUCTION

1. CHILDHOOD: ADOLESCENCE CRITICAL STAGE OF THE DEVELOPMENT

Childhood is the age lifetime ranging from birth to adulthood. This period is characterized by a physical growth, changes in body size and body composition, brain maturation, and psychological development. The period of growth and human development could be divided up into the developmental stages of infancy and toddlerhood (from birth to 2 years old), early childhood, middle childhood (school age), and adolescence (puberty through post-puberty) (Ballabriga and Carrascosa, 2006).

Adolescence is a crucial period in life and implies multiple physiological, psychological as well as social changes. This is a transition period from childhood to adulthood, which encompass ages from 10 to 20 year old (World Health Organization (WHO), 2001; Muñoz *et al.*, 2014). However, the last stage of adolescence is sometimes undefined. It would finish when individuals achieve certain social behaviour and the social, familiar and labour responsibilities of an adult (Rodríguez, 2003).

Traditionally, adolescence may be roughly divided into three stages (Muñoz *et al.*, 2014):

- Early adolescence (10-13years): preadolescence period in which they rapidly begin to develop secondary sexual characters.
- Medium adolescence (14-16): this period corresponds with height growth and body shape and body composition changes.
- Later adolescence (17-20 years): It is characterized by a lower growth along with a consolidation of sexual identity.

This period of life is characterized by physiological growth and development that correspond with puberty. Puberty involves a physical growth, brain maturation and development of the reproductive capacity by somatic and psychological changes (Euling *et al.*, 2008). In addition, adolescence is defined as second stage of accelerated growth and an intense anabolic stage. For this reason, nutrient requirements are higher (Ballabriga and Carrascosa, 2006; Muñoz *et al.*, 2014) and the nutrition in adolescence received special attention to provide necessary requirements and to reach an optimal growth and development.

Apart from physical development, adolescents experience several sharp psychological and social changes. Adolescence is a critical period to develop behaviour and emotional regulation skills. Although some self-regulation process begins to develop during infancy and mid childhood, the ability to develop behavioural strategies to manage their own moods increases in adolescence (Zeman *et al.*, 2006; Bariola *et al.*, 2011; Perez-Pereira *et al.*, 2013). Also, body and puberty changes influence on the psychological development since they stop being children to become adults. Especially children who show an early puberty are more vulnerable to develop psychological problem (Kaltiala-Heino and Marttunen, 2003; Zehr *et al.*, 2007). It seems to affect more girls than boys (Oldehinkel *et al.*, 2011).

Furthermore, an increase of social influences occurs in adolescence. While children are strongly influenced by attitudes and behaviour of parents as primary socialisation agents, in adolescents, the influence of secondary socialisation agents such as peer, school and media increases their behaviour. Also, adolescence is a critical period during the entire life in which adolescents adopt and establish healthy or unhealthy lifestyle behaviour which may carry into adulthood (Lake *et al.*, 2004).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

The fundamental purpose of all these changes and actions is to form one's own identity and to prepare for adulthood (Sawyer *et al.*, 2012). At the same time, these physical, psychological and social changes make adolescents more vulnerable to develop behavioural or emotional problems as well as health problems related to nutrition and obesity. Understanding the association between the psychopathological states, nutritional diseases and/or unhealthy behaviour is becoming urgent to boost mental and physical healthy as well as well-being in adolescents.

2. EMOTIONAL PROBLEMS

2.1 DEFINITION

Emotional disorders such as anxiety and depression are also known as interiorized disorders and they are frequent among child and adolescent population.

2.1.1 Depression

Depression is a common and serious medical disease. Depression symptoms can vary from mild to severe and can include: feeling sad or having a depressed mood, apathy, anhedonia (i.e loss of interest or pleasure in activities once enjoyed), feeling worthless or guilty, pessimistic feelings, difficulty in thinking or making decisions, decreased concentration, thoughts of death or suicide, and difficulty in social relation (interpersonal problems). Also it could cause changes in psychomotor activity, appetite and weight, sleeping problems, loss of energy or increased fatigue (American Psychiatric Association (APA),2000;APA, 2013).

Depression in children and adolescents includes the clinical picture described above but the expression of symptoms is specific to the child's age. Thus, the manifestations are: decrease academic task and school performance; low motivation to go to school, shouting outbursts, complaining, unexplained irritability, guilty feelings; sadness (expressed by crying or distress); tiredness and boredom, expressions of fear or anxiety; aggression, refusal to cooperate, antisocial behaviour; use of alcohol or other drugs; somatizations (constant complaints of headache, aching arms, legs, or stomach with no apparent cause) (Jané, 2001; Yorbik *et al.*, 2004; Khalil *et al.*, 2010).

According to current classification systems two diagnoses of depression can be mainly distinguished: Major Depressive Disorder, which it is an acute and severe depression; and Dysthymia, a persistent depression disorder which is a mild but chronic depression (APA, 2000). In the current classification system of Diagnosis Statistical Manual of Mental Disorders 5 (DSM-V) dysthymia is named Persistent Depression (APA, 2013). Similarity, the International Classification of Diseases 10th version (ICD) developed by the World Health Organisation (1992) distinguishes the Depressive Episode which include severity levels (mild, moderate and sever) and Persistent Mood Disorders which include Dysthymia.

The diagnostic criteria in children and adolescents are the same as those in adults (APA, 2013):

• Major Depressive Disorder requires one or more major depressive episodes. It is defined as depressed mood and/or loss of interest or pleasure in life activities and at least five of the following symptoms that cause clinically significant impairment in social, work, or other important areas of functioning almost every day:

1. Depressed mood most of the day.
2. Diminished interest or pleasure in all or most activities.

3. Significant unintentional weight loss or gain.
4. Insomnia or sleeping too much.
5. Agitation or psychomotor retardation noticed by others.
6. Fatigue or loss of energy.
7. Feelings of worthlessness or excessive guilt.
8. Diminished ability to think or concentrate, or indecisiveness.
9. Recurrent thoughts of death.

• The Dysthymic Disorder or Persistent Depression is a depressed mood most of the day, quite frequently, for at least 2 years (one year in children and adolescents), and the presence of two or more of the following symptoms that cause clinically significant impairment in social, work, or other important areas of functioning:

1. Poor appetite or overeating.
2. Insomnia or sleeping too much.
3. Low energy or fatigue.
4. Low self-esteem.
5. Poor concentration or difficulty making decisions.
6. Feelings of hopelessness.

2.1.2 Anxiety

Anxiety disorders are the most common mental disorders and its symptomatology cause physic and psychic distress. Anxiety is a set of physiologic and behavioural manifestations in front of stimuli that is considered potentially dangerous despite actually it is not. Many different symptoms affecting the cognitive, affective, somatic, relational and motor areas include: overwhelming feelings of panic and fear, uncontrollable obsessive thoughts, painful, intrusive memories, decreased attention, recurring nightmares, physical symptoms such as feeling sick to your stomach, tachycardia, palpitation, breath problems, hyperventilation, dizziness, cold sweat, flushed face, trembling,

gastrointestinal problems, headache, and muscle tension. In children and adolescents, the anxiety manifestations could also affect the school performance and relation with their peer (APA, 2013).

During developmental stages, fears and transitory anxiety symptoms often appear due to several physiological, psychological and social changes typical of this stage (Cartwright-Hatton *et al.*, 2006; Reardon *et al.*, 2009). Due to this fact some authors believe that anxiety disorders are one of the early forms of psychopathology since infancy and adolescence are critical stages of the development (Van Oort *et al.*, 2009; Merikangas *et al.*, 2010b; Beesdo *et al.*, 2011; Vicente *et al.*, 2012). Nevertheless, it has been pointed that the course of childhood anxiety is often chronic-recurrent since despite of having applied a treatment, anxiety disorders are usually chronic (Bruce *et al.*, 2005; Ramsawh *et al.*, 2009).

Different anxiety diagnoses could be classified into (APA, 2000; APA, 2013):

• Panic Disorder: is characterized by panic attacks and persistent worry and vigilance about prospective symptoms of another panic attack. The panic attacks are recurrently and unexpectedly.

Panic attack: is an episode of overwhelming fear of being in danger for no apparent reason with a combination of physical and psychological distress. The onset of symptoms is sharp, reaching a pick of intensity in 10 minutes and it is accompanied by somatic and cognitive symptoms. During an attack several of these symptoms occur in combination: pounding heart or chest pain, sweating, trembling or shaking, chills or hot flashes shaking, difficulty breathing, sensation of choking, nausea or abdominal pain, dizziness, feeling unreal or disconnected (or depersonalization) and fear of losing control, going crazy or dying. The symptoms are quiet severe and fear of having another attack and avoidance of stimuli, which are believed to serve as triggers, are

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

characteristic of panic disorders (Keeley and Storch, 2009). Such avoidance can lead to agoraphobia, with severe anxiety in public places (Rockhill *et al.*, 2010). Panic disorder is less common in childhood than in adolescence, and the clinical presentation of panic disorders varies across the developmental period (Diler *et al.*, 2004).

• Phobias: A phobia is an excessive and persistent fear of a specific object, situation, or activity. These fears cause a high level of distress. Mainly, there are two types of phobias.

- *Specific phobia*: an extreme or excessive fear of an object or situation that is not harmful. Phobic fear is clearly out of proportion to the threat and it is persistent by the presence or anticipation of a specific object or situation. This phobia results in significant functional impairment. Despite, people are aware of their fear is excessive, but they cannot avoid it. In children, their fears can often be inferred in the context of avoidant responses and somatic complaints. Children may exhibit more tearful behaviours, or may alternatively exhibit externalizing characteristics with tantrums and irritability (Rockhill *et al.*, 2010).

- *Social phobia or social anxiety disorder*: it is a significant anxiety and discomfort about being embarrassed in social situations or negative evaluation by others. For instance, public speaking, meeting people, or using public restrooms, eat or writing in public or social situation. These fears result in avoidance of situations when the child fears act in a humiliating or embarrassing manner. Somatic symptoms of blushing or trembling are also common. It is described as a vicious cycle, in which anticipatory anxiety of a perceived threatening social situation leads to negatively biased cognitions and anxiety symptoms, which, in turn, consequently leads to poor performance in the feared situations, and then leads to embarrassment and increased anticipatory anxiety about the feared situations and further avoidance (Keeley and Storch, 2009). It is common to onset during adolescence. Children

and adolescents with social phobia have been found to experience greater sensitivity to rejection, report fewer friendships and close relationships, and perceive less social support and acceptance from peers (Rockhill *et al.*, 2010).

• *Generalized Anxiety Disorder:* It is a severe and persistent pattern of tension and worry over a wide variety of daily activities (such as school performance, social concerns, or family interaction) and that interferes with the daily activity. It is characterized by selective attention to negative and threat-related information and by negative, uncontrollable, or catastrophic thoughts. They are constantly worried and feel inefficient and helpless to control these worries such as job or school responsibilities, family health, appointments, meeting (Rockhill *et al.*, 2010). Children and adolescents with generalized anxiety disorders usually show intense anxiety during or before a different of daily routine activities and also their worries are excessive about academic task or relation with their peer. Somatic symptoms could include trembling, muscle tension, headaches, and fatigue irritability, problems to concentrate and work efficiently (Rockhill *et al.*, 2010). Children with generalized anxiety may have a chronic inability to relax and with few coping skills to effectively handle concerns (Keeley and Storch, 2009).

• *Separation Anxiety Disorder*: While it has been a specific disorder in children in DSM-IV-TR, it can now be diagnosed in adulthood (APA, 2013). The manifestations of anxiety and distress appear when they are separated from who represents safety for the affected child, typically a parent or primary caregiver. Although separation anxiety is a normal state during early years of development (until 2 years), sometimes it could continue after this age or appears later in front of a stressful situation. When separated from the person representing safety, significant worry about self or the person representing safety results in both distress and interference with functioning (APA, 2000). The common symptoms are: excessive distress when separated from the

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

primary caregiver, fear of getting lost, kidnapped, or dying and feel worry about losing their primary caregiver or that something harmful occurs; also they could experiment nightmares, avoidance to go to school or other places because of fear of separation of their primary caregiver, to go to sleep without the primary caregiver nearby, repeated somatic symptoms (e.g., stomach-ache) (Keeley and Storch, 2009; Rockhill *et al.*, 2010). The separation anxiety could be manifested differently with age, with younger children showing excessive crying and bad temper and older children displaying social withdrawal and manipulative behaviour to avoid school or separation (Keeley and Storch, 2009).

In addition, it is important to bear in mind that each subtype of anxiety is associated more frequently with different stages of the development. In general, it describes that separation anxiety and certain specific phobias appear in earlier ages, as long as social phobia usually appears between last childhood and early adolescence, and panic disorder and generalized anxiety disorder start to mid or late adolescence (Kessler *et al.*, 2005; Becker *et al.*, 2007; Beesdo *et al.*, 2007, 2011; Costello *et al.*, 2011).

2.2 EPIDEMIOLOGY OF EMOTIONAL DISORDERS IN CHILDHOOD

Mental disorders affect 10–20% of children and adolescents worldwide (Kieling *et al.*, 2011). It is estimated 1 in 5 young people between the ages of 12 and 19 years experiencing a mental disorder at any time (Costello *et al.*, 2011).

Merikangas et al. (2010a) studied the twelve-month prevalence of the main mental disorders in children from United Stated; and found that mood disorders, panic disorder along with anxiety disorder were 3.7% and 0.7% respectively. The same author in another study conducted on a representative sample of adolescents found that anxiety disorders are

more prevalent (31.9%) in adolescent population; followed by mood disorders in third place (14.3%) (Merikangas, He, Burstein, et al. 2010b). The main findings of these studies reveal that one in every four to five youngsters in the United State meets criteria for a mental disorder with severe impairment across their lifetime.

In Europe, an Irish study observed that the prevalence of any mental disorder was between 27.4% and 36.8% in children and adolescent (Coughlan *et al.*, 2014). The lifetime prevalence in children aged 11-13 of mood disorder was 14.9%, being diagnosis of major depression disorder of 6.9% and diagnosis of dysthymia of 0.4%. Likewise, the prevalence rate of anxiety disorders was 22.6% (Coughlan *et al.*, 2014). Another study conducted on Italian children and adolescents showed that the prevalence of emotional disorders, a 6.5%, was higher than those externalizing disorders, a 1.2% (Frigerio *et al.*, 2009). A multicentre European study showed that 47.7% of the children and adolescents presented psychosomatic problems (Vanaelst *et al.*, 2012). Particularly, in Spain, symptoms of anxiety and depression affect around 47% and 9% respectively of school population (Canals *et al.*, 1995; Romero *et al.*, 2010).

Moreover, it has been observed that these disorders are more prevalent in female population (Romero *et al.*, 2010; Conley *et al.*, 2012; Moksnes *et al.*, 2012; Abbo *et al.*, 2013; Coughlan *et al.*, 2014). Similarly, one study found girls had twofold higher rates of mood disorders than boys, although there were no gender differences in the rates of anxiety disorders (Merikangas, He, Brody, et al. 2010a).

Also, several researches have estimated the prevalence of each subtype of anxiety disorder. In children and adolescents, the prevalence of specific phobia is between 0.7%-14.1%, of social phobia is 0.6%-20%, of panic disorder is 0.2%-3.9%, generalized anxiety disorder is 0.4% - 6.6%, and of separation anxiety is 0.6-8% (Abbo et al. 2013; Burstein et al. 2012; Canino et al. 2004; Coughlan et al. 2014; Gau et al. 2005;

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Kessler et al. 2012; Kim-Cohen et al. 2003; Merikangas, He, Burstein, et al. 2010b; Merikangas, He, Brody, et al. 2010a; Shear et al. 2006; Wells et al. 2006; Esbjorn et al. 2010).

Nevertheless, the different subtypes or disorders of anxiety often coexist between them, so the comorbidity, specifically homotypic comorbidity, is highly significant. For instance the comorbidity between anxiety symptoms was 87% in Spanish schoolchildren (Romero *et al.*, 2010). In another community sample of United States, from 6 to 28% of youngsters met criteria for two types of anxiety at the same time such as separation anxiety disorder with social phobia or generalized anxiety disorder or even more frequently the comorbidity among social phobia and generalized anxiety disorders (Kendall *et al.*, 2010). Indeed, 36% of children met criteria for three types of anxiety at the same time (i.e. separation anxiety, generalized anxiety and social phobia) (Kendall *et al.*, 2010). In a clinical sample, the homotypic comorbidity among subtypes of anxiety is higher, around 18-75% (Lewinsohn *et al.*, 1997; Masi *et al.*, 2004; Kendall *et al.*, 2010; Leyfer *et al.*, 2013). In time, this homotypic comorbidity could lead to more sever and chronic anxiety disorders and this effect could encompass a functional impairment and a worse prognosis (Ramsawh *et al.*, 2009; Costello *et al.*, 2011; Lamers *et al.*, 2011).

The comorbidity with other disorders (heterotypic comorbidity) is highly prevalent in both children and adults. The most frequent comorbidity is between anxiety and depression. Indeed, this comorbidity rate in children and adolescents is around 30% to 75% (Lewinsohn *et al.*, 1997; Essau, 2008; Esbjorn *et al.*, 2010; Lamers *et al.*, 2011). In Spanish scholars was found that 87% of children with depressive symptoms had comorbidity of anxiety symptoms and a 20% of children with anxiety showed depressive symptoms (Romero *et al.*, 2010).

2.3 HEALTH CONSEQUENCES AND EFFECT ON EATING BEHAVIOR

Adolescence has been recognised as a period of significant mental health vulnerability and risk for young people. The early onset of emotional disorder in children or during adolescence is prognostic of worse course of disorder and is a risk factor for future mental disorder (Kessler *et al.*, 2005; Ramsawh *et al.*, 2011). Emotional psychopathology is related to childhood complications such as physical dysfunction, disability, substance abuse, multiple risk behaviours, poor functioning, suicide attempts and hospitalization. Furthermore, anxiety and depression in childhood may predict adult depression and anxiety disorders (Kendall *et al.*, 2004; Bittner *et al.*, 2007; Katon *et al.*, 2010; Rockhill *et al.*, 2010). The presence of both disorders could lead to the development of worse mental disorders in children and adolescents, such as academic problems, social relations problems and impairment of interpersonal relationship (Sijtsema *et al.*, 2014). Children with primary diagnosis of anxiety and with comorbidity of mood disorders show higher levels of severity even after the treatment (Rapee *et al.*, 2013).

In addition, several studies have associated depression and anxiety with development of overweight, obesity or even cardiovascular and metabolic diseases (Rozanski *et al.*, 1999; Roy-Byrne *et al.*, 2008; Kivimaki *et al.*, 2009; Needham *et al.*, 2010).

Moreover, it is important to highlight that these disorders are difficult to detect and treat due to their interiorized condition, what could worsen the course and prognostic of the disorders. Therefore, due to mental disorders being considered one of the main causes of disability and that their consequences are usually maintained to adulthood (Kieling et al. 2011) more effort should be invested in their prevention and treatment.

Anxiety and depression are some of the most common co-occurring conditions with eating disorders or emotional eating. This comorbidity

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

could complicate the diagnosis, hinder recovery effort and be a marker of a great severity of disease, which might predict poorer outcomes (Hughes *et al.*, 2013; Zerwas *et al.*, 2013).

Eating disorders are characterized to focus on weight, shape and food concerns. Adolescents are more vulnerable to social influences related to thinness and beauty and begin to worry about their weight and body shape (Lawler and Nixon, 2011). Evidence suggests that the symptoms of eating disorders could occur at earlier ages, between 10 and 15 years and affect 8–17% of adolescents (Sancho *et al.*, 2007; Rome, 2012). Adolescents with symptoms of eating disorders are more vulnerable to suffer from depression and anxiety since there is an overlap among manifestations of eating disorders and anxiety and mood disorders (Touchette *et al.*, 2011). For instance, there are common signs in anorexia or bulimia and anxiety disorders such as intense anxiety around food and eating, fear of weight gain and anxiety about social evaluation. Depression also shares common features with eating disorders such as apathy, lethargy, poor concentration, emotional liability, low self-esteem.

Furthermore, generalized anxiety seems to occur more in girls with bulimic and binge eating and weight concern, and high risk of mood disorder has been observed in girls with non-specified eating disorders (Touchette *et al.*, 2011). Depressed children and adolescents with eating disorders tend to present high binge/purge disorders profile (Hughes *et al.*, 2013). Indeed, major depressive episode has also been associated with onset and persistent bulimic symptoms (Keski-Rahkonen *et al.*, 2013) and it was also found that dysthymia predict the presence of eating disorders or body dissatisfaction (Zaider *et al.*, 2000; Babio *et al.*, 2009). Also, it is suggested that anxiety could precede the onset of both eating and depressive disorders whereas depression coexist simultaneously with both disorders (Godart *et al.*, 2005, 2007). Nevertheless, Johnson et al. (2002) indicated that

depression during adolescence could be a predictor of eating disorder in girls at age of 18.

High body dissatisfaction is associated with low self-esteem that is a feature of depression as well as eating disorders. A longitudinal study of 186 children found that decreases of eating disorders symptoms were predicted by an increase of body satisfaction and self-steem (Berg *et al.*, 2009). Gual et al. (2002) in a sample of girls from 12 to 21 years found that high levels of neuroticism and low level of self-esteem could be cause and consequence of eating disorders.

It is important to bear in mind that eating disorders are also considered to be a cause of nutritional risk in adolescence since they generally are marked by restricted food, unusual patterns of food consumption and/or bingeing. Several cross-sectional studies have shown that adolescents even with symptoms of eating disorders often present higher weight and poorer dietary intake (i.e. lower intake of energy, macronutrients and certain micronutrients) than adolescents without symptoms of eating disorders (Dunker and Philippi, 2005; Babio *et al.*, 2009; Larson *et al.*, 2009; Chang *et al.*, 2011; Tsai *et al.*, 2011; Allen *et al.*, 2012). Also, individuals with eating disorder and comorbid depression have a high body dissatisfaction and food restriction. There is evidence that girls who want to be thinner or show body dissatisfaction tend to restrict energy intake and dense calorie food (Makinen *et al.*, 2012; Bibiloni *et al.*, 2013).

In addition several authors have described other eating disturbances like emotional eating which could be triggered by stress situations, depression or anxiety. Emotional eating has been defined as eating in response to emotional stimuli, often as a coping response to negative emotions (Bruch, 1973). In children, studies consistently report that emotional eating is often followed by negative emotions (Fernstrom *et al.*, 1994; Braet and van Strien, 1997; Elfhag *et al.*, 2008; Czaja *et al.*, 2009; Goossens *et al.*, 2009; Michels *et al.*, 2012b). Consequently,

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

emotional eating could be the result of self-inability to regulate emotions. This maladaptive strategy might underlay the relationship between emotional dysregulation and obesity by modifying dietary intake (Czaja *et al.*, 2009; Francis and Susman, 2009; Graziano *et al.*, 2010; Greene *et al.*, 2011; Harrist *et al.*, 2013) or lead to overeating or loss of eating control (Goossens *et al.*, 2009).

3. FOOD CONSUMPTION AND DIETARY PATTERNS

3.1 FOOD CONSUMPTION AND PHYSICAL ACTIVITY IN ADOLESCENCE

Dietary habits are acquired early in life in response to physiological requirements and psycho-social pressure, which could have a considerable impact on long-term health status. Adolescence is an important stage of development marked by an accelerated growth. For this reason, the adequate food consumption that provided the optimal and correct development is needed. However, the dietary patterns of the adolescents differ so much from recommendations.

Food consumption and eating habits are variable from early childhood to adolescence. While children usually show structured eating habits, determined mainly by parental influence and home availably, adolescents show irregular dietary and eating out-of-home patterns , which is influenced by peers, media and social environment and economical availability. In this sense, adolescents present rapid changing eating habits and have particular food choices compared to younger children and adults. According to Cutler et al. (2011), from mid childhood to adolescence, a new fast-food dietary pattern emerges, and it is characterized by high intakes of hamburgers, French fries and fried food. it is important to highlight that other unhealthy dietary

patterns acquired during childhood, like snacks, are also maintained during adolescence or even adulthood.

The main features of adolescents eating habits is that they are unstructured, with frequent snacks rich in fat and sugar, frequent meal-skipping, particularly breakfast, frequent consumption of fast food and eating outside home. Also, adolescents sometimes show high levels of restrained eating or follow diets (Mataix-Verdú and Sánchez-Campo, 2002; Muñoz *et al.*, 2014). Most adolescents do not have an appropriate breakfast habit, some have a breakfast of poor quality and around 7% of adolescents skip it (Hallström *et al.*, 2011). This eating behaviour usually emerges and increases from adolescent to adulthood (Niemeier *et al.*, 2006). Also, 57.2% of girls and 31.6% of boys have done some attempt to follow a diet (usually without the control of a dietician or nutritionist) or have used other unhealthy methods to control weight (Neumark-Sztainer *et al.*, 2004).

Food consumption of adolescents is characterized by an excess of meat, fat, sweets and refined products along with a poor consumption of fruits, vegetables, fish and seafood and whole grain (Aranceta *et al.*, 2003b; Muñoz *et al.*, 2014). Approximately three out of every four adolescents report two unhealthy dietary habits (Iannoti and Wang, 2014). Longitudinal findings in a large American adolescent cohort found that adolescents showed adverse changes in dietary intake. For instance, fruits and vegetables intake decreased significantly to less servings per day during transitional periods of 5 years (Larson *et al.*, 2009). Also, it is indicated that fast food consumption markedly increases and consumption of breakfast decreases from adolescent to adulthood (Niemeier *et al.*, 2006). In Europe, HELENA study results show that European adolescent food choice shifted to unhealthy choices. They found that adolescents eat half of the recommended amount of fruits and vegetables and less than two-thirds of the recommended amount of milk and milk products, but consume much more meat and meat products, fats and sweets than recommended

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

(Diethelm *et al.*, 2012; Moreno *et al.*, 2014). Also, a national Irish survey found that there was a noticeable transition from a less energy-dense diet in pre-school children to a more energy-dense diet in older children and adolescents, and this change was associated with an reduction of fibre, fat, fruits and vegetables, and higher intakes of confectionery and snacks and sugar- sweetened beverages (Walton *et al.*, 2014). According the data of The Catalonian Heath Survey (2013), population of 3-14 years old, 24.7% consume hyper caloric products (fast food, sugary drinks, or salty snacks and food) more than three times per week and only 7.7% of the recommended serving of fruits and vegetables. Also, 88.1% eat breakfast before leaving the house and at mid-morning at least four times per week. However, this percentage sharply decreases to 45.7% among population of 15-44 aged (Government of Catalonia Ministry of Health, 2014).

The beverages are also a particular issue in adolescence. The majority of studies that investigated trends in beverage consumption reported that between 7.6 and 53.1% of adolescents have a high intake of soft drinks more than once per day (Janssen *et al.*, 2005). It has been shown that after water consumption, the largest contributor to fluid consumption are beverages such as sugar-sweetened beverages, sweetened milk drinks, and fruit juice (Duffey *et al.*, 2012) and that these beverages provided a high amount of energy to daily intake (Moreno *et al.*, 2014). Results of Nelson et al. (2009) reflect longitudinal shifts in American adolescent beverage consumption during the critical transition period from early to mid-adolescence and mid- to late adolescence. They observed that the intake of soda and sugar-sweetened beverages and alcohol increased, whereas consumption of certain beverages decreased with age like fruit juice, milk or milk beverages.

Therefore, the adolescents shift their dietary pattern to choose more unhealthy food that leads to a poor diet quality of. Indeed, although the energy intake is close to the recommendation, the nutrient intake,

especially vitamin and mineral could be compromised. The intakes of vitamin D, folate and iodine were less than about 55% of the recommendations (Diethelm *et al.*, 2014) and they did not reach the recommendation of iron, calcium, vitamin A, C and B6 intakes (Serra Majem *et al.*, 2003; Neumark-Sztainer *et al.*, 2004; Aranceta Bartrina *et al.*, 2006; Serra-Majem *et al.*, 2006), mainly in girls that used unhealthy methods to control their weight (Larson *et al.*, 2009).

Regardless lifestyle, physical activity and sedentary behaviour are other topics to take into account in adolescence (Gubbels *et al.*, 2013). The lifestyle in adolescents is characterized by an increase of sedentary activities and a decrease of physical activity during leisure time. Several sedentary activities are part of children and adolescence daily routine (for instance classes at school, studying) as well as their leisure time (for instance watching television, playing video games or surfing the internet). Among adolescents, the most common leisure-time sedentary behaviour is watching television and playing video games and nowadays increasingly surfing the internet and using new media technologies (Iannoti and Wang, 2014; Santaliestra-Pasías *et al.*, 2014a). It is suggested that less than 4 out of 10 children and adolescents met both physical activity and screen-time recommendations of healthy style life (Fakhouri *et al.*, 2013). In the Irish national survey, 35% of school-aged children and adolescents reported to be watching television more than 2 hour on an average school-day and 65% watching television more than 2 hours of a weekend day (Walton *et al.*, 2014). A Spanish study showed that 24% of adolescents watch television, 9% of adolescents use computer, 7% play videogames, 17% surf on the internet more than two hour per day on weekdays (Gómez *et al.*, 2012). Likewise, the recent data of Catalonian population aged 3 -14 shows that 96.1% watch television daily during their free time while 79.7% play in the park or street. Also, a 20.7% of this population has sedentary leisure habits meaning that they dedicate two hours or more every day of the week to watching television or video games.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Several authors suggest the association between sedentary habits and unhealthy dietary pattern (Ottevaere *et al.*, 2011; Lissner *et al.*, 2012; Gubbels *et al.*, 2013; Santaliestra-Pasías *et al.*, 2014b). In this sense, Santaliestra-Pasías *et al.*, (2014a) showed that increased watching television and computer and Internet use during adolescence was associated with higher risk of consumption of sweetened beverages and lower risk of fruit consumption.

Therefore, the current dietary and lifestyle behaviour of adolescents is considered an important public health issue due to its potential harmful effects linked to obesity, cardio metabolic risks and other health outcome. Moreover, poor dietary habits in this critical period of adolescence might continue into adulthood and then become difficult to modify. Therefore the establishment and maintenance of a healthy diet early in life is of great public health importance.

3.2 METHODS OF FOOD CONSUMPTION ASSESSMENT

There are several methods to assess food consumption that allow us to examine the food consumption and energy and nutrient intake in collective or individual level.

These methods provide us with important information about the frequency of food consumption and/or the quantity of food, energy and nutrient intake. Therefore, it enables us to identify inadequate diets and nutritional status, to assess and monitor nutritional health, and also to examine trends and changes in dietary patterns of the population.

Dietary food information could be obtained in three levels (del Pozo de la Calle *et al.*, 2015):

- National level: using the Balance Food Sheet based on data provided by the Ministry of Agriculture. This method allows to know the food availability in a country.
- Familiar level: by household budget surveys or family record or diary.
- Individual level: using dietary survey from which cross-sectional information is obtained to assess dietary intake (Gibson, 2005). A wide variety of dietary survey methods exists, the most used:
    - Food frequency questionnaire
    - 24 hour recall method
    - Food record
    - Diet history
    - Screener and brief assessment methods

3.2.1 Food frequency questionnaire

Food frequency questionnaire is a dietary assessment tool which is highly used in epidemiological studies to examine the relation between dietary intake and disease or health outcomes. Food frequency questionnaire is a retrospective and direct method to estimate food consumption by which global dietary information is obtained from a certain period of time (i.e last 3 month or last one year). Briefly, this method consists in asking how often and how much food items are consumed over a reference period (Martin-Moreno and Gorgojo, 2007). This method enables us to classify the participants that show a high or low consumption of certain food (Gorgojo-Jiménez and Martín-Moreno, 2006; Martin-Moreno and Gorgojo, 2007; Arija, 2014).

This method can be self-administered, on paper or web-based, or interview administered (face-to-face or by telephone). More complete data may be collected if the food frequency questionnaire is administered by an interviewer; although self-administered

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

questionnaires may reduce respondent bias. Thus, many food frequency questionnaires are designed to be self-administered. The estimated average time to complete the questionnaire is 30 to 60 minutes, but it depends on the questionnaire and the respondent (Pérez-Rodrigo *et al.*, 2015a)

There are three types of food frequency questionnaire: qualitative, semi-quantitative and quantitative. Qualitative questionnaires are those which only ask about frequency of food consumption, not about the size of consumed portions. Otherwise, semi-quantitative questionnaires include standard portions or reference portion sizes for each item and respondents are asked how often they consume the specified portion of a particular food item. Quantitative questionnaires ask respondents to estimate either in grams or household measures the size of the portions consumed (Gorgojo-Jiménez and Martín-Moreno, 2006; Pérez-Rodrigo *et al.*, 2015a).

The food frequency questionnaires contain three main components: the list of foods, frequency of consumption and optionally the portion size consumed (Pérez-Rodrigo *et al.*, 2015a). The food list ought to show the food habits of the study population. The frequency of consumption might be open ended questions (where respondents write the number of times a day, week or month that they consume this food) or by presenting frequency categories (for instance if the food item is consumed always, often, hardly ever, never or once a week, three or four times a week, etc.). Additionally, semi-quantitative or quantitative food frequency questionnaires include the portion size which could be standard or self-referred size. Both can be used to obtain absolute quantity of energy and nutrient intakes, multiplying the frequency by nutritional content of standard or self-referred portion size.

Food frequency questionnaires may be designed to focus on whole diet or on particular group of foods or nutrients such as calcium (Huybrechts *et al.*, 2007). When the respondents are children, elder or disable people, parents or caregivers should be present or respond the

questionnaires even though some food frequency questionnaires are sometimes designed to administer specific population groups, such as children (Huybrechts *et al.*, 2007; Shai *et al.*, 2007).

The food frequency questionnaire shows several strengths. Food frequency questionnaires assess food consumption over a wide period of time and enable us to estimate the usual intake. They are highly cost-effective, easy and fast administration since they have a standard format; therefore they are widespread used in large epidemiological cohort studies. This method implies low respondent load compared to other methods, and this increases the cooperation and participation. Moreover, being a retrospective method, the habitual consumption is not influenced. They show a considerable validity and accuracy to estimate the dietary intake (Arija, 2014). Furthermore, food frequency questionnaires requires less nutrition knowledge in data entry compared to other food consumption assessment methods and therefore do not require trained professionals (Pérez-Rodrigo *et al.*, 2015a).

Despite these advantages, several drawbacks exist. Firstly, there is high complexity in designing these questionnaires or their validation, which involve systematic errors and biases in dietary intake estimates (Pérez-Rodrigo *et al.*, 2015a). It could produce inaccuracies in the result from an incomplete listing of all possible foods and from errors in frequency and usual portion size estimations. However, a comprehensive and complete list of all foods cannot be included since the length of questionnaire influence on accuracy of the dietary report for instance over-estimation increases. Therefore, a balance on disadvantages of longer list and of over-estimation of intake and additional respondent burden should be assessed (Block, 1982; Nelson and Bingham, 1997; Thompson and Subar, 2012). Other limitation is the inaccuracies in the dietary report due to respondent memory. Also, respondent should have a relatively high degree of literacy and numeracy skills are required if self-administered.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

3.2.2 24-hour dietary recall

The 24-hour dietary recall method is one of the most widely used methods in nutrition epidemiology. The 24-hour dietary recall is an open interview, retrospective and quantitative method that examines the food consumption of the 24 hours before. This method is a direct interview (face to face or by telephone) and currently can also be self-administered using computer programmes (Salvador *et al.*, 2015). The estimated average interview time can be between 20 to 30 minutes.

The method consists of describing and quantifying the consumption of foods and beverages consumed in the previous 24 hours (i.e during the day before the interview), from the first intake in the morning until the last intakes at night (Beaton *et al.*, 1979). The information should describe the type of food and its characteristics (i.e. fresh, precooked, frozen, canned, preserved), the quantity consumed, method of preparation (i.e. fried, boiled, steamed), commercial brands, sauces, dressings or condiments to add (i.e. sauces, type of fats or oils used, sugar), or accompanied food (i.e. bread) as well as the time and place of consumption (i.e. at home, away from home, restaurants).

The method requires several support tools (such as examples of dishes, volumes and household measures, drawings or photographic models or three dimensional models) as well as contribution of novel technologies could be helpful to obtain an accurate assessment of food consumption (Salvador *et al.*, 2015). This method involves professional trained interviewer who should have dietetic and nutrition knowledge (ingredients, food preparation, and dishes) and be familiarized with the eating habit of the study population to be able to estimate and record accurate information of daily food consumption. Also, the interviewer should attempt not to influence on the interviewee answers (Salvador et al., 2015). Once the food consumption information is recorded, this should be analysed with a data base to obtain grams of food and nutrients and energy intake per day using a table of food composition.

One single 24-hour dietary recall does not estimate usual intake. A minimum of two or five days of the 24-hour dietary recall is needed to examine usual dietary intake, it depends on the aim of study, the nutrients of interest and the sample size. In common practice two or three days of 24-hour recall are used and they must be carried out on non-consecutive days including a weekend day (Serra-Majem and Ribas-Barba, 2006; Martin-Moreno and Gorgojo, 2007). This period of time involved acute dietary information without diminish the participation. Otherwise, 24-hour recall during more days (i.e 7 days) could diminish the participation. Also, it is better to administer in different periods of time of the year so as to examine seasonal variation (Arija, 2014).

The 24-hour recall is easy and quick method as well as it requires low cost and shows high precision. Response rate is high since its administration does not require so much time and could be administered to low literacy population. Moreover, the habitual food consumption of the participant is not altered since it is a retrospective method. Serial recalls can estimate the usual intake at the individual as well as the community level (Serra-Majem and Ribas-Barba, 2006; Martin-Moreno and Gorgojo, 2007; Shim *et al.*, 2014).

Nevertheless, this method shows several limitations. One of the main drawbacks is that it depends on the recent memory of the interviewee. Therefore this method is not recommended for the elderly or children less than 12 years of age. Also, the accuracy of this method is influenced by the capacity of interviewee to refer food information. For instance, women and individuals who follow a diet tend to specify more exactly the dietary information than men or individuals who not follow a diet. Moreover, the "Flat slope syndrome" is described as the tendency to overestimate low intakes and underestimate high intakes. Underestimated intake is often in the elderly, children, and obese people or with unhealthy eating habits (for instance alcohol or fat excess intake) (Salvador *et al.*, 2015).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

3.2.3 Dietary record

The dietary record is a prospective and quantitative method in which the subject records all the foods and beverages consumed and quantities over a specific period of time, usually between 3-7 days (Thompson and Byers, 1994).

This method usually record detailed information about portion size, food preparation methods, ingredients of mixed dishes and recipes, and even the brand name of commercial products. Therefore, the participant should be specifically trained to be able to describe adequately the food items and the quantities used, including the name/brand of the consumed food, recipes of dishes, method of preparation or cooking, and also the portion sizes. At the end of the recorded period, a trained interviewer should review the dietary record with the participant, to clarify any doubts or ask by possible forgotten foods consumed (Thompson and Subar, 2012; Ortega *et al.*, 2015).

Mainly, there are two types: dietary record by household measure or by estimation and weighted dietary record.

In *dietary record by household measure or by estimation*, the participants have to record all food consumption and the estimation of portion sizes could be estimate by household measure (plates, spoons, bowls, cups, and glasses, in reference to standard household measures, using three-dimensional food models, or two-dimensional photographs (Thompson and Subar, 2012). This is easy, cost-effective and it represents a little load to the participant. Therefore, it obtains a high degree and participation.

In *weighed dietary record*, the amount of food consumed should be precisely measured by a kitchen weighing scale. The food consumed should be weighted before eating and after eating (the food rest) and the participant should estimate the food eat-out-home. By this way, it

is obtained the real quantity food consumed. This method requires a standardized kitchen weight scale of all participants of the study to diminish the bias. Moreover, this method shows two derivations:

- Weighed dietary record with interviewer in which the interviewer is who weight and record food consumption. This method is useful for institutionalized population or low literacy population. It could be combined with a 24 hour dietary recall to know the out-of-home dietary intake. Both methods, by weight and by weight with interviewer, show a high accuracy. However, they require a high level of cooperation from the participants' part, which could diminish the participation.

- Weighted dietary record with chemical analysis in which the methodology is similar to method of record by weight, but the food composition is obtained chemically. This method requires that respondent keep up a portion of food which will be chemically analysed. This last method presents the highest validity and accuracy, for this reason, it is considered the gold standard method in empirical researches. However, the limitations are the high complexity of the technic, high economic cost and high level of participation of the respondent (Aranceta-Bartrina and Pérez-Rodrigo, 2006; Arija, 2014).

Generally, the main advantage of all these dietary record methods is their potential to collect accurate quantitative information (Thompson and Subar, 2012). Because of the quality of the dietary data, it is considered to be the gold standard of the dietary methods. Also, this method not depends on the memory of the participant and is more accurate, since the amount of consumed food is recorded when eaten.
Regardless of the length of the dietary recall, it is important to establish the number of days and which days (consecutive/non-consecutive, working days/weekend days) to be monitored in the dietary record. The optimal number of days to collect more reliable data depends largely on the nutrient or the sample size. Traditionally the most common dietary record monitors the diet for 7 consecutive days. This time period allows

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

for collecting information about the diet minimizing bias related to the day of the week. Ideally, it is needed a long enough period of time to provide accuracy information of dietary (a minimum of 3 days is required) but without diminishing the participation and compliance (periods of no more than 4 consecutive days) (Aranceta-Bartrina and Pérez-Rodrigo, 2006; Moreno *et al.*, 2008; Arija, 2014; Ortega *et al.*, 2015).

In addition, other limitations are that these methods require that interviewers and participant are well trained and a high cooperation and literate of the participant. This could influence on the participation of some population groups (people with low literacy, immigrants with low language skills, children, elderly, and people with writing difficulties) (Ortega *et al.*, 2015). Another limitation is that these methods can alter food consumption of the participants since participants are more conscious about the food and amount they consume since their diet will be analysed (Aranceta-Bartrina and Pérez-Rodrigo, 2006; Kristjansdottir *et al.*, 2006; Thompson and Subar, 2012).

3.2.4 Diet history

The Diet History is a retrospective and quantitative method to describe food and usual nutrient intake during a relatively long period (Kohemeier, 1991). This method is used more often in the clinical practice than in research studies (Morán Fagúndez *et al.*, 2015).

It consists in a long interview that can take from 1 to 2 hours and require a highly qualified interviewer in nutrition. Participants are asked to try to remember the food consumption for a certain period of time (Jain, 1989; Nelson and Bingham, 1997; Martin-Moreno and Gorgojo, 2007).

The diet history method assesses quantitatively the global food intake of individual, habits in relation to food consumption, distribution and usual composition of meals throughout the day (Aranceta-Bartrina and Serra-Majem, 2006). Some authors considered that the complete method usually consists of:

– An interview recalls estimating the habitual food consumption in the different eating occasions in a day. Often a 24-hour recall is included.

- A food frequency questionnaire to verify information to assess the overall pattern of food consumed.

– A 3 day dietary record with estimated portion sizes of the foods and beverages consumed.

Therefore, this method provides detailed information on usual food intake during a specific periods of life at individual levels, thus we could obtain more representative patterns than other methods of diet assessment.

Nevertheless, the method has several limitations that should be considered. For instance, this method entails a great effort of memory, a high participation and cooperation of the participant and a large duration to implement it. Furthermore, diet history method tends to overestimate intake. Otherwise, as it is focused on evaluation of usual patterns, exceptional intakes are underestimated. The diet history is a complex, large and costly method that require highly well trained personnel, and there is not a standard protocol of complete diet history, for this reason, it is not applicable in large scale population studies. Therefore, currently the main application of the diet history method is in clinical practice (Morán-Fagúndez *et al.*, 2015).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

3.2.5 Screener and brief assessment methods

Brief assessment tools are easy, self-administered and qualitative questionnaires. From the brief questionnaire scores are obtained and are usually categorized according to levels. Usually, these tools ask about frequency of consumption or about dietary habits, thus they are useful to identify individuals with a very low or high intake and identify risk of malnutrition or inadequate consumption patterns for specific food groups (Pérez-Rodrigo *et al.*, 2015b). Brief questionnaires are therefore useful tools to identify individuals and groups who require more attention.

These methods do not require a trained professional nor depend on the memory of the participant (Green and Watson, 2005), thus there is a high participation rate and cooperation. Therefore, recently these new brief assessment and self-evaluation tools are used by health professionals in primary health care, in community intervention to early screening and health promotion (Aranceta-Bartrina *et al.,* 2006; Pérez-Rodrigo *et al.*, 2015b).

In childhood and adolescents it could be particularly interesting to examine their dietary habits and for health promotion and nutrition education interventions.

Several brief dietary assessment tools used in children and adolescent population are like: *Youth Healthy Eating Index (YHEI), Kreceplus and the KIDMED; Day in the Life Questionnaire (DILQ); Synchronised Nutrition and Activity Programme (SNAPTM); Child Nutrition Questionnaire (CNQ); Family Eating and Activity Habits Questionnaire (FEAHQ).*

Youth Healthy Eating Index is the adapted version of the Healthy Eating Index (HEI) (Kennedy *et al.*, 1995) which was developed by the United States Department of Agriculture with the purpose to monitor the

adherence to the Dietary Guidelines for Americans. The revised version to use in children and adolescents consists in 13 items (Feskanich *et al.*, 2004).

Kreceplus questionnaire is screener tool to identify and monitor energy balance behaviours and quality of diet. It consists in 16 items. It was developed by the Spanish Society of Community Nutrition and the Spanish Association of Paediatrics (Serra-Majem *et al.*, 2003). Based on this questionnaire, the KIDMED screener was designed to assess adherence to Mediterranean Diet in children and adolescents. The base of Mediterranean diet which includes a high consumption of fruits and vegetables, olive oil, fish, legumes, grains and nuts, and dairy, whereas snacks, and pastries, sweets or fast food are not characteristic (Serra-Majem *et al.*, 2004).

Child and Diet Evaluation Tool (CADET) consist of a list of 115 items foods focus on fruits and vegetables and with a section with questions about breakfast. The participant should mark which food is consumed during a 24-hour. It is targeted to 3–7 year old children but the instrument contains two questionnaires that should be fulfilled one by parent and the other by a lunchtime supervisor school or classroom assistant (Cade *et al.*, 2007).

Day in the Life Questionnaire (DILQ) is targeted to 7-9 years children and consists of 17 items. The modified version is targeted to 9–11 years and contains 23 items. It was developed as a supervised classroom activity to assess fruit and vegetable consumption in the previous 24 hours. This questionnaire includes items, pictures and words (Edmunds, 2002).

Synchronised Nutrition and Activity Programme (SNAPTM) is a web-based programme target 7–15 year old children. This program uses a typical 24-hour recall method to assess dietary intake using a 40

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

different food and 9 different drink items, and performance of physical activity through a typical school day (Moore *et al.*, 2008).

Child Nutrition Questionnaire (CNQ) was developed in Australia to children from 10–12 year olds to examine dietary patterns. It measures the consumption of sweetened beverages, sweet and fat foods as well as healthy eating behaviours. These questionnaires contain a 14-item and require to be fulfilled by an adult (Wilson *et al.*, 2008).

Family Eating and Activity Habits Questionnaire (FEAHQ) examines environmental factors and family behaviours associated with weight gain and weight loss in children. It is a self-administered questionnaire which contains 21-item and is designed to 6-11 year-old obese children for co-completion by parents or caregivers (National Obesity Observatory, 2011)

3.3. DIETARY ASSESSMENT IN CHILDREN AND ADOLESCENTS

The 24-hour recall, dietary records, dietary histories, food frequency questionnaires and brief questionnaires have all been used to assess dietary intake in children and adolescents. However, there is no consensus of which dietary assessment method is the most accurate for school-age children and adolescents (Pérez-Rodrigo *et al.*, 2015c). The accurate assessment of dietary intake in children and adolescents show certain limitation and difficulties related to their developmental, cognitive, social and behavioural characteristics (Livingstone *et al.*, 2004). Therefore, there are several factors that we ought to consider.

The accuracy of these methods may be affected by cognitive factors, such as children memory about food consumption or details about food (food preparation, ingredients, time of consumption, eating occasion), the ability of children to estimate the size of portions, the level of

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

attention along the interview is reduced, and certain literacy and writing skills are needed (Pérez-Rodrigo *et al.*, 2015c).

Children and adolescents dietary assessment can be reported by themselves, by their parents, by caregivers or a combination. This depends on factors such as child independence (mainly in adolescence), cognitive abilities and increased consumption of food and drinks outside the home (Burrows *et al.*, 2010). Mainly, parents or caregivers as reporters along with the child may provide better estimates than the child alone, but it could depend on the age. Children under 7 years old probably need to be helped by parents or caregivers to report the food consumption. By aged of 14, it is suggested that they may be accurate self-reporter of their dietary intake. However, there are transition periods between ages 8 -13 years in which it is not clear who is the best reporter. Some children from 10 years could feel their parent as an intrusion, and they likely prefer to complete the dietary assessment by themselves, although they cannot report food consumption with accuracy (Livingstone *et al.*, 2004). Thus, no recommendations are established on who is the most appropriate reporter of dietary intake for children in the age range (Burrows *et al.*, 2010; Pérez-Rodrigo *et al.*, 2015c). Also, parents or caregivers as reporters of children and adolescents dietary assessment have raised some bias since those parents are more likely to report desired health behaviours than children do (Livingstone *et al.*, 2004). In this vein, children and adolescents could also under or over-report some food consumption in line with perceived social and desirable norms. In this sense, the association between dieting and weight consciousness with misreporting is the most frequent. Overweight in children or their parents also could compromise the accuracy of dietary report (Pérez-Rodrigo *et al.*, 2015c).

Moreover, the motivation is need to increase the collaboration of children and adolescents and achieve accurate food information. This consideration is important during adolescence since although the

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

adolescents are more able to report, they may be less interested in giving accurate reports or not participating. Also, body image often influences on dietary intake especially in this period of age. For this reason , it is advisable to bear it in mind when assessing dietary intake in adolescents (Pérez-Rodrigo *et al.*, 2015c).

On the whole, all these facts imply that the accuracy of the dietary assessment is even more difficult than other age groups.

3.4 DIETARY PATTERNS

Dietary patterns allow to characterize dietary behaviour and to explain the relationship between diet and health (Moeller *et al.*, 2007).

The traditional studies of diet have examined associations between individual nutrients or food with health outcomes. The importance of dietary patterns has received more attention despite the discrepancies in the epidemiological literature on the role of individual nutrients in many health outcomes (Steffen, 2006; Siri-Tarino et al., 2010). In this sense, dietary patterns are biologically important because foods are consumed in complex combinations, not food and nutrient isolated. Thus, these approaches measure total diets and take into account the interactions and synergies occurring between nutrients. Therefore, nutritional epidemiologists have studied dietary patterns, or combinations of foods and nutrients, often intended to represent the whole diet or certain factors of the diet in relation to chronic disease or health outcomes (Moeller *et al.*, 2007; Mcnaughton, 2011).

From the dietary intake data obtained by food consumption assessment methods, many approaches exist that allow identifying and characterizing dietary patterns in a population. Dietary patterns can be created *a priori* using score based approaches or *a posteriori* using data-driven techniques such as factor analysis and cluster analysis (Hu,

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

2002; Jacobs and Steffen, 2003; Kant, 2004; Newby and Tucker, 2004).

3.4.1 Score-based methods *a priori*

Score-based approaches are based on dietary recommendations as well as other knowledge about nutrition and health. These approaches evaluate the quality of the diet in terms of intake of nutrients, variety and balance. They are easy to create and also they are reproducible and comparable with other studies. These methods generally include: *nutrient adequacy* or *density scores*, *variety or diversity scores*, *food-group patterning scores*, and *index-based summary scores* (Moeller *et al.*, 2007).

Nutrient adequacy score evaluate overall dietary adequacy (Guthrie and Scheer, 1981). It includes *the nutrient adequacy ratio* (i.e average daily intake of a nutrient divided by the age and sex-specific recommended intake of that nutrient) and *mean adequacy ratios* (the sum of the nutrient adequacy ratio divided by the number of nutrients having a nutrient adequacy ratio).

Nutrient density scores evaluate the dietary quality of individual foods in terms of nutrient content in relation to total energy, but do not evaluate a total dietary pattern (Drewnowski, 2005).

Dietary variety score examines variety among food groups (the mean number of different food groups consumed daily) and variety within food groups (the mean number of foods within food groups consumed daily (Krebs-Smith *et al.*, 1987).

Food-group patterning scores consist of creating scores of each food group and examine the variation and diversity of food groups in the diet. There are other food-group patterning scores based on the five

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

major food groups: fruits, vegetables, grain, dairy and meat (Kant *et al.*, 1991).

Index-based summary scores are based on interpretation of current dietary guidelines. They are algorithms that allow evaluating the overall diet and classifying individuals according to their "healthy" dietary intake. There are many different types of indexes, but the three main categories are: a) nutrient-based indicators; b) food/food group based indicators; and c) combination indexes (Moeller *et al.*, 2007). The most well-known index scores are: the Diet Quality Index (DQI and DQI-Revised) (Patterson *et al.*, 1994; Haines *et al.*, 1999), the Healthy Eating Index (HEI) (Kennedy *et al.*, 1995), Healthy Diet Indicator (HDI) (Huijbregts *et al.*, 1997), and the Mediterranean Diet Score (MDS) (Trichopoulou *et al.*, 2003; Bach *et al.*, 2007). Moreover, several indexes have been adapted and modified from those originals (Moeller *et al.*, 2007; Gil *et al.*, 2015): Recommended Foods Score (Kant *et al.*, 2000), the Not Recommended Foods Score (Michels, 2002); Alternate Healthy Eating Index (AHEI) (McCullough and Willett, 2006); Mediterranean Diet Adherence Screener (MEDAS) (Schröder *et al.*, 2011).

Nowadays, dietary quality indexes or healthy life indexes tend to include even more giving more information on behaviours, associated with specific dietary patterns, physical activity, rest and selected socio-cultural habits (Gil *et al.*, 2015). For instance, the Mediterranean Lifestyle (MEDLIFE) (Sotos-Prieto *et al.*, 2015).

3.4.2 Data-driven approaches *a posteriori*

Data-driven approaches use multivariate statistical approaches to derive dietary patterns. The aim is to reduce a larger group of dietary variables to a smaller group of variables (Moeller *et al.*, 2007; Mcnaughton, 2011). These methods, a part from being created to

associated dietary pattern with health outcomes, they also enable us to know how food is consumed together and describe eating behavioural in a certain population. Thus, they are useful for behavioural research. These methods do not require a prior definition of dietary patterns. However, they require a thorough knowledge of the literature on nutrition to identify and interpret the new set of variables as a dietary pattern (Moeller *et al.*, 2007). Therefore, a review of literature has shown that "Health/prudent," "Traditional/western," "Sweets/desserts," and "Alcohol/Drinker" patterns are common and easy reproducible across populations (Newby et al., 2004). However, one of the limitations is that some subjectivity is introduced at several points in the process: group of food, treatment of input variables (i.e grams or portions, standardized by energy, etc) selecting and interpreting a final pattern solution.

The most used *a-posteriori* methods to identify dietary patterns are factor analysis and cluster analysis of dietary intake.

*Principal components Analysi*s is the most commonly used method in nutritional epidemiology. It uses an algebra matrix to identify the principal components based on correlations or covariance matrix in a large set of variables (food groups). As a result, several "factors" are created being these linear combinations of the original variables that explain the variance in the data. To improve the interpretability of the factors, they can be rotated for instance using an orthogonal rotation as varimax rotation which is commonly used. Each factor is composed by factor loadings (or scoring coefficients) for each original variable. These factors are used to interpret the dietary pattern. Thus, dietary patterns are characterized with those factor loading are higher, and they describe food groups. Dietary patterns are labelled according to food items with higher load. At the end, individuals receive factor scores for each derived factor. Factors are continuous variables that are often categorized into quantiles (Moeller *et al.*, 2007).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Cluster analysis derives dietary patterns based on differences in intake among individuals. They describe variations in food consumption and separate individuals into exclusive and non-overlapping groups. Thus, individuals belong to one cluster only. For this method it is important to choose a similar variable and select which clustering procedure is best (Moeller *et al.*, 2007).

3.5 DIETARY REFERENCE INTAKE

Dietary Reference intake (DRI) is a collection of nutrient standards used to assess the nutrient intake in United States and Canada. They are carried out by the Institute of Medicine in different reports. The reports are focused on specific nutrients as well as their appropriate manners of use and interpret the DRIs for assessing nutrient intakes and for planning nutrient intakes (Murphy *et al.*, 2002; Institute of Medicine (IOM), 2006).

DRIs include a wide range of safety precautions to compensate variations in the requirements among individuals. Firstly, DRI were established to prevent deficiency disease, buy nowadays, this concept was amplified and does not only encompass deficiency disease but also chronic diseases like osteoporosis, cancer, cardiovascular disease.

Several international organizations have established DRI in order to use them to determine adequate food patterns. Among the international organizations we can highlight Food and Agriculture Organization (FAO) and WHO Expert Committee and Food and Nutrition Board of National Research Council which periodically publish their reports based on update knowledge about nutrition. The related concepts with reference intake have progressed significantly during the last 50 years pointing out the importance of individual variability and influence of cultural and individual factors.

The new DRI include the following concepts (Murphy *et al.*, 2002; IOM, 2006):

Recommended Dietary Allowance (RDA): is defined as the average daily nutrient intake level sufficient to meet the nutrient requirement of nearly all (97-98%) healthy individuals in a particular life stage and gender group. They are established by the assessment of the estimated average requirement and a statistical dispersion of ± 2 standard deviation.

Estimated Average Requirement (EAR): is the average daily nutrient intake estimated to meet the requirement of 50% the healthy individuals in a particular life stage and gender group.

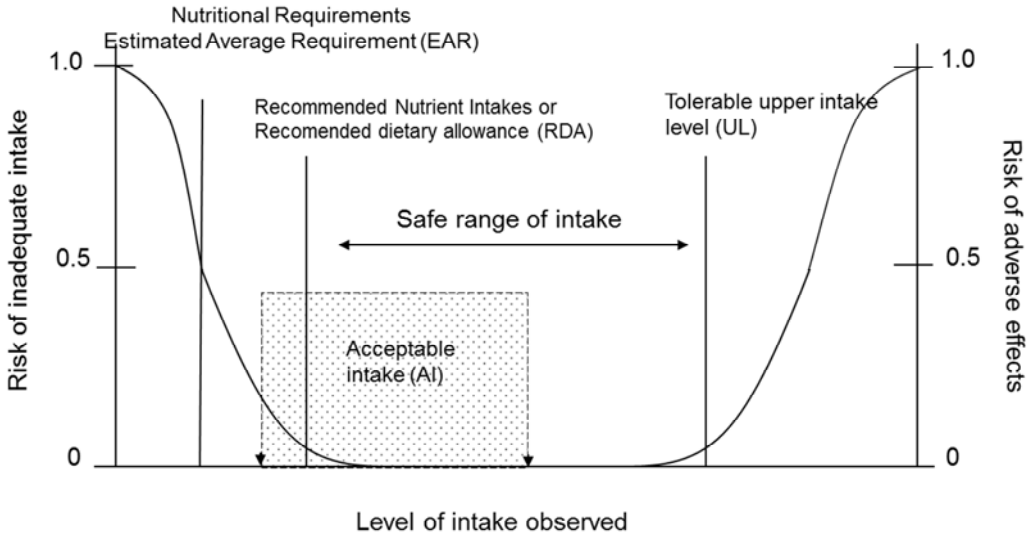
Adequate Intake (AI): A recommended average daily nutrient intake level based on observed or experimentally determined approximations or estimates of nutrient intake by a group (or groups) of apparently healthy people that are assumed to be adequate, whether it is used when an RDA cannot be determined.

Tolerable Upper Intake Level (UL): The highest average daily nutrient intake level likely to pose risk of adverse health effects to almost all individuals in the general population. As intake increases above the UL, the potential risk of adverse effects is increased.

When adequate information is available, each nutrient has a set of DRIs. A nutrient has either an EAR and an RDA or an AI. When an EAR for the nutrient cannot be determined (therefore, neither can the RDA), then an AI is set for the nutrient. In addition, many nutrients have a UL (Murphy *et al.*, 2002; IOM, 2006).

In case of energy, estimated Energy Requirements (EERs) can be calculated for individuals based on sex, age, body size, and physical activity. The mean energy intake for a group should approximate the mean EER. If the mean energy intake were to exceed the EER, then the individuals would be gaining weight as a group, and if they were to fall below the EER, they would be losing weight (Murphy et al., 2002; IOM, 2005).

In 1993, The Scientific Committee of Food and the European Union defined three values of reference: Estimated *Average Requirement (as EAR)*, *Population Reference Intake (as* PRI), and *Lowest Threshold Intake* (as LTI). These concepts are the same as American terminology*: Estimated Average Requirement, Reference Nutrient Intake, and Lower Reference Nutrient Intake (LRNI),* respectively. The terms of *Population Reference Intake* and R*eference Nutrient Intake* were changed by RDA (Serra-Majem and Aranceta-Bartrina, 2006). *Inferior limit of the intake* is the lowest value of intake below which it occurs a deficient intake. This value is included in European and British DRIs' but not in United States and Canadian DRIs'. Furthermore, the terminology in the Spanish and English literature is different (Serra-Majem and Aranceta-Bartrina, 2006) (Figure 1).



Dietary reference intakes include four distinct concepts: a) the average of the nutritional needs of the population group, b) nutritional recommendations located two standard deviations of the average needs, except the recommendations of energy, c) the acceptable nutrient intake when there is insufficient data to estimate the recommendations but adequate information to make this dietary advice is available, and d) the tolerable upper intake levels, above which there can exist a health risk.

Figure 1. Dietary reference intake (Adapted from Arija et al., 2015)

Food and Agriculture Organization (FAO) and World Health Organization (WHO) created a DRIs of vitamins and minerals and a prevention plan for chronic diseases (WHO / FAO, 2003). In Europe, tables of nutrient and energy intake were created in 1993 (Scientific Committe on Food, 1993). In Spain, the first standard reference of recommended intake appeared in 1981. From this year, it has been regularly reviewed and updated by the Nutrition Department of Pharmacy Faculty at Universidad Computense de Madrid. And the last review was updated in 2013 (Moreiras *et al.*, 2013).

There are a few studies on nutritional requirements in adolescent population. Hence, the DRIs for adolescents are obtained by extrapolated results of studies in children and adults. Moreover, another

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

limitation is that the DRI are established according to the chronologic age, whereas in adolescence it is considered better to take into account the biologic age (Serra-Majem and Aranceta-Bartrina, 2006).

Comparability of reference values: Index of nutritional adequacy enables us to assess deficient intakes (Serra-Majem and Aranceta-Bartrina, 2006). It is one of the most commonly used methods to assess the percentage of adequacy to recommendations, and compares the intake average of each nutrient with their respective DRI values (As it has been mentioned above in *a priori pattern* section).

The index of nutritional adequacy (INA) is used to compare the mean intake of each nutrient with their respective DRI values. It is calculated as follows:

INA of a nutrient = dairy mean intake of a nutrient/ DRI of nutrient

The result could be multiplied by 100 to obtain the percentage of nutritional adequacy.

Percentage of nutrient adequacy = (dairy mean intake of a nutrient/ DRI of nutrient)*100

When this process is applied to different nutrients, it enables us to calculate the mean of index of nutritional adequacy (MINA):

MINA: sum of INA of n number of nutrients/ n number of nutrient

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

4. OBESITY AND OVERWEIGHT

4.1 DEFINITION

Overweight and obesity are defined as the excess of body fat that may be unhealthy for people and that may contribute to the development of cardiovascular disease risk factors (WHO, 2003).

The distribution of excess body fat is associated with risk factors. There are two types of obesity *android or central obesity* and *gynoide or peripheral obesity* and they show different risks for our health.

4.2 EPIDEMIOLOGY OF OBESITY AND OVERWEIGHT IN CHILDHOOD

Childhood and adolescence are critical periods of the development to onset overweight and obesity. There is an increasing tendency worldwide towards excess weight in childhood (Branca *et al.*, 2007; Center for Diseases Control and Prevention, 2015). Currently, overweight and obesity affect 9–36% of the child and adolescent population in several developed and developing countries (Lobstein and Frelut, 2003; Gupta *et al.*, 2012; Ogden *et al.*, 2012, 2014; Organisation for Economic Co-operation and Development, 2012; Valdes Pizarro and Royo-Bordonada, 2012). In the United States, the prevalence of obesity in adolescents from 11 to 18 years is nearly 25% in boys and 18% in girls (Cunningham *et al.*, 2014). In Europe, the greatest obesity prevalence of childhood are found in the southern regions (Brug *et al.*, 2012). Particularly, In Spain, the prevalence of obesity ranges approximately from 11.0% to 20.9% in boys and from 11.2% to 15.5% in girls. Whereas the prevalence of overweight reaches 26.7% and 25.7% in boys and girls, respectively (Pérez-Farinós *et al.*, 2013). In our region, the current report of The Catalonian Health

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Survey point out that a 31.1% of children from 6 to 12 years-old show excess of weight, concretely 21.0% of children are overweight and 10.1% are obese. Children aged 6-9 show more obesity and overweight than children aged 10-17. The rates of prevalence of overweigh is higher in girls (22.0%) than in boys (20.1%), whereas obesity affect more boys (10.4%) than girls (9.8%). The prevalence of obesity is higher among children under 15 years who belong to low SES (15.3%) (Government of Catalonia Ministry of Health, 2014).

4.3 METHODS OF OBESITY AND OVERWEIGHT ASSESSMENT

The assessment of overweight and obesity in children and adolescents is difficult because a standard definition does not exist. Body fat is the most variable component which depends on the balance on energy intake and expenditure and modifies the body weight.

There are different methods to assess the body fat mass:

Anthropometry: The main anthropometric measures are weight, height, skinfold and body circumferences (Mataix-Verdú and López-Jurado, 2002):

Body mass Index (BMI) or also called Quetelet index: It is calculated by weight in kilograms divided by height squared. This parameter is one of the most widely used to determine excess of weight in epidemiologic studies and clinical practice since it has a good correlation with body fat and it is also easy, fast and cost-effective to determine (Mataix-Verdú and López-Jurado, 2002). In adult population, WHO defined overweight and obesity as a cut-off of 25 and 30 respectively. However, in children and adolescent population we find the reference values of BMI standardized by age and gender to assess overweight or obesity. In Spain, Faustino Orbegozo Foundation has published several reference tables which are frequently used in routine clinical practice to define BMI categories. Hernández et al. (1988)

created a table with reference values in which the 85th percentile was used to define overweight and the 97th percentile to define obesity. Sobradillo et al. (2004) developed a reference table from data obtained in cross-sectional and longitudinal studies. The cut-off point was the 85th percentile to define overweight and the 95th percentile to define obesity.

The international reference value has been developed and they are widely used and allow comparability with the population. On the one hand, the WHO Multicentre Growth Reference Study developed growth reference curves from birth to 18 years old in a sample represented by widely different cultural settings and ethnicities (WHO, 2006; WHO Multicentre Growth Reference Study Group., 2006). International Obesity Task Force developed the BMI reference tables from information from six countries. According the age and gender, they proposed to define overweight and obesity from a value according to BMI in children equal or above 25 (overweight) and 30 (obesity) in adults (Cole *et al.*, 2000; Cole and Lobstein, 2012). However, this BMI cut-off seems not to show enough accuracy to detect percentage of body fat (%BF) and other supplementary measures are recommended (Moreno et al. 2006). It has been suggested to use international references, along with country-specific standards (Walton *et al.*, 2014).

In addition, the BMI z-score is the deviation of an individual's value from the mean value of a reference population, divided by the standard deviation of the reference population. In children and adolescents, overweight is defined with a BMI value for each age above + 1 point z-score above and obesity is defined with a BMI value for each age +2 point z-scores above (de Onis *et al.*, 2007).

Skinfolds: they estimate the subcutaneous fat tissues using a caliper. They are non-invasive and low cost technics that require a trained professional. The skinfold can be obtained from biceps, triceps, subscapular, iliac crest, supraspinale, abdominal, front thigh and median calf subscapular (Mataix-Verdú and López-Jurado, 2002).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Several reference values have been developed (Moreno et al. 2006; Addo and Himes 2010) as well as exists different equations to calculate the body fat based on the sum of different skinfold.

Waist circumference (WC): It is considered as a good measure of abdominal fat in adult as well as in children and adolescents (Mataix-Verdú and López-Jurado, 2002). Moreno et al. (1999) developed a reference values of WC and determine that the 75th percentile and 95th percentile to screening of high moderate values and high severe values of WC. The WC is higher in boys than in girls especially after the 11.5 years old, and it increase with the age. McCarthy *et al.*, (2001), also develop a WC percentile form British schoolchildren aged from 5 to 17 years. As it is defined as an indirect indicator of abdominal fat, it is suggested that it is an adequate measure for screening metabolic syndromes in children (Moreno *et al.*, 2007). It is observed that the WC and BMI show a strong and positive correlation with % BF assessed by densitometry (Neovius *et al.*, 2004). Indeed, the use of WC and waist/height ratio along with the BMI is recommended for definitive diagnosis of metabolic syndrome in children. In addition, WC and subscapular fold thickness may estimate better the metabolic risk than BMI (Marković-Jovanović *et al.*, 2015).

Waist-Hip index: this index is obtained dividing WC by hip circumference. It allows describing the distribution of the fat tissues. This is an easy and frequently used method in epidemiologic studies in adults (Mataix-Verdú and López-Jurado, 2002). However, the drawbacks are that it does not allow differentiating with accuracy between subcutaneous and abdominal fat. Also it is poorly sensible when there is a loss of weight since if it entails a decrease of both waist and hip circumferences, hence the value of the index do not decrease (Mataix-Verdú and López-Jurado, 2002). Other measures as WC and waist-height index seem to be more adequate. Moreno et al. (1997) describe the percentiles of waist and hip index in Spanish population aged 3-15 years. The waist-hip index decrease with the age in girls due

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

to the increase of the pelvis and the physiological accumulation of fat in a gluteal level.

Waist-height index: This measure has been used in adults to estimate the risk of disease related to abdominal obesity (Ashwell *et al.*, 1996; Gruson *et al.*, 2010; Sabah *et al.*, 2014). These parameters are obtained dividing the WC by height. It is a simple and non-invasive tool that has an adequate correlated with abdominal fat. This index decrease with age and is significantly lower in girls. An acceptable value of low risk of health problem is 0.5 (Ashwell *et al.*, 1996) in adults as well as in children and adolescents. This index is used to predict metabolic syndrome or metabolic risk in children (Elizondo-Montemayor *et al.*, 2011; Nambiar *et al.*, 2013).

Bioelectrical impedance: This method allows estimating the percentage of body fat (%BF). This method is based on electric conductivity of the water which is higher in the lean mass than fat mass. The lean mass has a higher content of water and electrolytes which work like electric conductors, while fat mass has a low content of water and electrolytes, so it does not contribute to conduct electric signals. To improve the accuracy of this tools, there are some factors to consider since they could modify the results, such as: the position of the individual, the content of metal objects in the pockets, cleaning the electrodes, beverages intake, the presence of oedema, dehydration status, menstrual period and febrile episode (Mataix-Verdú and López-Jurado, 2002). There are no unified nor international standard values to define overweight and obesity in children and adolescents. However, several tables with reference values based on percentiles have been developed in some countries (Moreno *et al.*, 2005; McCarthy *et al.*, 2006). Moreno et al. (2005) in a sample of Spanish adolescents showed that the BF percentage was 29% and 32% in boys and girls respectively in terms of overweight. In contrast to girls, the adiposity in boys decreases with the age and pubertal status (Taylor *et al.*, 2003).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

In addition, other more complex methods are used to assess the body fat such as densitometry, air-displacement plethysmography, water doubled marked or dual energy X-ray (DEXA). They are the most accurate methods however they are complex, sophisticated and have a high economical cost. For this reason they are not often used in large epidemiological studies (Mataix-Verdú and López-Jurado, 2002; Michels *et al.*, 2012a).

4.4 CONSEQUENCES OF OBESITY

Childhood obesity is associated with a higher risk of obesity in adulthood and also with premature deaths and disabilities in adulthood (World Health Organization, 2015). Indeed, overweight and obesity before or during puberty is a risk factor for weight excess in adulthood (Singh *et al.*, 2008; Pelone *et al.*, 2012; Martos-Moreno *et al.*, 2014).

Overweight and obese children and adolescents are at greater risk of atherogenic disorders and are more likely to develop a cardiovascular disease in adulthood. Obese children and adolescents tend to have high triglycerides and cholesterol, including total cholesterol and low-density protein (LDL), and low-high density protein (HDL) (Martos-Moreno *et al.*, 2014). So, early complications are common and include high blood pressure, dyslipemia and hyperinsulinism and insulin resistance. In addition, these factors could increase the risk of premature death and cancers (Franks *et al.*, 2010). Other comorbidities are related with breathing difficulties, increased risk of fractures and even psychological effects. In adulthood some types of cancer are also related with obesity comorbidities.

In addition, android obesity involves higher risk of health complications than gynoid obesity. Peripheral obesity is associated with vein disease, biliary lithiasis and decrease of insulin. Android obesity is associated with higher risk of arthrosclerosis, hypertension, hyperuricemia,

diabetes mellitus, hypercortisolism, hypertriglyceridemia, decrease of HDL cholesterol, heart diseases, decrease of life expectancy, hyperinsulinism, hypercholesterolaemia (Figure 2) (Bueno *et al.*, 2007). As a result, the abdominal obesity assessment is needed and has a high clinic and epidemiologic interest. Therefore, obese and overweight children show an increased risk for their health and well-being. The progressive increased of obesity is associated with physical and psychological health consequences in a short or long term in adulthood (WHO, 2013; WHO, 2015).

ANDROID OBESITY

- Arthrosclerosis
- Hypertension
- Hyperuricemia
- Diabetes mellitus
- Hypercortisolism
- Hypertriglyceridemia
- Low HDL
- Heart diseases
- Low life expectancy
- Hyperinsulinism
- hypercholesterolaemia

GYNOID OBESITY

- Vein disease
- Biliary lithiasis
- Low insulin

Figure 2. Types of Obesity according to fat distribution

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

4.5 RISK FACTORS

The epidemic of overweight and obesity in children observed in recent years has underscored the need for a better understanding of the risk (Lobstein and Millstone, 2007; Ogden *et al.*, 2012). Numerous genetic and environmental factors have been found to contribute to the recent epidemic of obesity and complex interactions between them have been described (Martos-Moreno *et al.*, 2014).

It is generally accepted that the main lifestyle factors related to the rising prevalence of obesity are a misbalance on energetic homeostasis due to an increase in total energy intake and an increase of sedentary behaviours (Janssen *et al.*, 2005; Rey-López *et al.*, 2013; Iannoti and Wang, 2014). We live in an obesogenic environment which enhances sedentary lifestyle, stress and the consumption of high-density/low-nutritional value food. Indirect factors that could be associated with obesity or obesogenic environment factors are societal changes such as lack of supportive policies in sectors such as health, agriculture, transport, urban planning, environment, food processing, distribution, marketing and education (WHO, 2013; World Health Organization, 2015).

In addition, stress and psychological factors are also associated with a higher risk of obesity (Incledon *et al.*, 2011). However the potential role of psychological and emotional distress in obesity development has been less extensively studied (Hemmingsson, 2014).

During the last decade much effort was invested in nutrition education interventions in children, adolescents and their parents. Despite public health campaigns, the prevalence of obesity does not decrease (Doak *et al.*, 2006; Stice *et al.*, 2007; van Grieken *et al.*, 2012; Martin *et al.*, 2013). It is suggested that new approaches are needed for prevention, education and treatment.

5. RELATION BETWEEN EMOTIONAL SYMPTOMS AND NUTRITIONAL STATUS

5.1 EMOTIONAL SYMPTOMS AND FOOD CONSUMPTION

It is suggested that emotional distress may lead to unhealthy eating behaviour and imbalanced dietary patterns or a shift of food choices, as it has been observed in epidemiologic studies (Kandiah *et al.*, 2006; Liu *et al.*, 2007; Yannakoulia *et al.*, 2008b; Konttinen *et al.*, 2009, 2014; Mikolajczyk *et al.*, 2009; Groesz *et al.*, 2012) as well as laboratory studies (Roemmich *et al.*, 2002, 2011; van Strien and Bazelier, 2007; Mooreville *et al.*, 2014). In fact, participants of experimental studies were found to be consuming more high-fat snack food and more sweet and fatty foods in response to stress (Roemmich *et al.*, 2011). Also, Kandiah et al. (2006) in an epidemiologic study showed that 81% of women had a change in appetite under stress and a 63% had an increase in appetite and opted for significantly more types of sweet foods and mixed dishes. Other authors observed differences between gender and showed that women, compared to men, may be more likely to increase food consumption, in particular sweet food or fat consumption under negative emotions (Wansink *et al.*, 2003; Dubé *et al.*, 2005; Yannakoulia *et al.*, 2008a).

In children and adolescent population, cross-sectional studies also showed that stress was mainly associated with high sweet food (Jenkins *et al.*, 2005; Oddy *et al.*, 2009; Michels *et al.*, 2012d) and fat (Cartwright *et al.*, 2003) as well as less healthy food (De Vriendt *et al.*, 2012; Jääskeläinen *et al.*, 2014) like fruit and vegetables (Cartwright *et al.*, 2003). Emotional symptoms have been considered as a chronic stressor. Researches carried out on adult population showed that suffering anxiety or depression has influence on food consumption (Liu et al., 2007; Mikolajczyk, El Ansari and Maxwell, 2009; Yannakoulia, Yiannakouris, et al., 2008). However, the epidemiological evidence in children and adolescents is less than in adulthood and it shows inconsistent results. Some authors only observed this association

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

between sweet food and stress and behavioural disorders but not with emotional disorders (van Kooten *et al.*, 2007; Oddy *et al.*, 2009).

On the other hand, the relationship between emotional symptoms and dietary patterns could be complex and were influenced by other psychological factors, especially during adolescence. In this sense, emotional symptoms are often accompanied by eating disorders, in particular during adolescence since it is a vulnerable period in which eating disorders arise (Sancho *et al.*, 2007; Stephen *et al.*, 2014). Eating disorders are characterized by unhealthy eating behaviours but on a contrary direction since they lead to decreased food consumption. Therefore, these disorders could modulate and mask the relation between emotional symptoms and dietary intake, especially during adolescence (Maxwell and Cole, 2009).

Also, stress and emotional symptom are linked to other aspects of obesity-related lifestyle like sedentary behaviour and low physical activity. Children and adolescents with stress or negative affect may perform less physical activity due to decreased motivation and are likely to spend more time on sedentary activities like video games, internet and television (Anton et al. 2006; Reeves et al. 2008; Ouwens et al. 2012).

5.2 EMOTIONAL SYMPTOMS AND WEIGHT GAIN

The potential role of psychological and emotional distress in relation to obesity has been less researched (Hemmingsson, 2014). The most important psychological and emotional distress factors presented in child populations include low self-esteem and self-worth, powerlessness and apathy, negative emotions, depression, anxiety, negative self-belief, insecurity and stress, and evidence for their role is increasing (Anderson *et al.*, 2011; Michels *et al.*, 2012c; Aparicio *et al.*, 2013). According to a systematic review, specific psychosocial factors in childhood may act as determinants for developing obesity in adulthood

(Vamosi *et al.*, 2010). Specifically, Anderson et al. (2006) studied a community-based US cohort from childhood to adulthood and reported that anxiety and depression disorders were associated with higher weight status. Goodman & Whitaker (2002) found that North American adolescents with symptoms of depression showed risk of obesity at 1-year follow-up in both genders. Although there are some studies that did not observed any association, generally a positive relationship was found between negative affect or stress in children and weight gain (see Table 1). These results were also replicated with the stress concept (Torres and Nowson 2007; Pervanidou and Chrousos 2012; van Jaarsveld, *et al.* 2009; Wardle *et al.* 2011; Roemmich et al. 2007; Roemmich *et al.* 2002).

On the other hand, most of the studies examine the relation between emotion psychopathology and the excess of weight using BMI, however a very few of them were methods measuring fat excess and its abdominal distribution (Tanofsky-Kraff *et al.*, 2006; Hillman *et al.*, 2010; Midei *et al.*, 2010). Nevertheless they showed mixed and inconsistent findings.

Therefore, some research on the relationship between emotions and weight gain and overweight during critical stages of the development could be extremely useful for designing preventive and treatment-based obesity programs.

Table 1. Child and adolescent stress and negative affect as predictors of weight gain/overweight/obesity: Prospective studies

First author, year and country	Follow-up duration	Sample (n) /gender (% girls)	Age or grades at baseline	Psychological predictor: Measurement tool	Adiposity measures	Results
Goodman, 2002 (USA)	1 year	N=9374 (48.6%)	Grades 7-12 (12-18 years)	Depressive symptoms: CES-D	Measured and self-reported: BMI	Depressive symptoms increased risk of obesity in obese and non-obese subjects
Anderson, 2010 (USA)	2 years	N=918 (100%)	Grade 6 (11 years)	Depressive symptoms: CES-D	Measured: BMI	Depressive symptoms associated with greater likelihood of obesity in white females
Bradley, 2008 (USA)	11 years	N=1254 (48%)	2 years to grade 6	Externalizing and internalizing problems: CBCL	Measured: BMI	Positive association between externalizing or internalizing problems and BMI
van Jaarsveld, 2009 (UK)	5 years	N=4065 (42%)	11-12 years	Perceived stress at a single time point and mean perceived stress over 5 years: PSS short form	Measured: waist, BMI	Perceived stress associated with Δ BMI z-scores, Δ waist High Mean of stress associated with 5-year BMI z-score trajectory and 5-year waist z-score trajectory
Midei, 2009 (USA)	3 years	N=160 (48.7%)	14 years	Trait anxiety symptoms: STAIC. Anger and anxiety: Cook-Medley Hostility Scale	Measured: waist hip ratio	Higher anger associated with increased waist-hip ratio
Tanofsky-Kraff, 2006 (USA)	4.2 years	N=146 (52%)	6-12 years	Depressive symptoms: CDI Child Symptom Inventory	Measured: body fat mass	No association
Rofey, 2009 (USA)	3 years	N=285 (49%)	8-18 years	Depression and anxiety: K-SADS, K-SAFD-P (DSM-III and DMS-IV criteria)	Measured: BMI	Females: depression and anxiety associated with high BMI Males: anxiety associated with high BMI No association with depression

First author, year and country	Follow-up duration	Sample (n) /gender (% girls)	Age or grades at baseline	Psychological predictor: Measurement tool	Adiposity measures	Results
Aparicio, 2013 (Spain)	3 years	N=229 (62%)	10 years	Depression and anxiety: SCARED, CDI MINI-Kid (DSM-IV criteria)	Measured: BMI, waist, body fat mass	Females: anxiety symptoms associated with Δ BMI and body fat mass; depression disorder associated with Δ waist Males: anxiety and depression associated with Δ BMI and waist Inverse relationship observed between major depression disorder and BMI
Larsen, 2014 (Netherlands)	3 years	N=1465 (49.4%)	11.4-16.9 years	Depressive symptoms: CES-D	Measured: BMI	Females: depressive symptoms associated with higher zBMI Males: no association
Rhew, 2008 (USA)	1 year	N=466 (46.2%) Sub-sample BMI measures: N=165	12 years mean	Depressive symptoms: MFQ	Measured and self-reported: BMI	BMI self-reported: Males: depressive symptoms associated with lower BMI than non-depressive symptoms Females: depressive symptoms associated with higher BMI than non-depressive symptoms BMI measures: no association
Michels, 2014 (Belgium)	2 years	N=316	5-12 years	**N**egative events: Coddington Life Events Scale for Children Negative emotions: anger, anxiety, sadness Behavioural Problems: SDQ	Measured: BMI, waist-to-height ratio, fat percentage	Stress positively or negatively associated with adiposity depending on cortisol and life-style, which had a moderating effect
Stice, 2005 (USA)	4 years	N=496 (100%)	11-15 years	Depressive symptoms: SADS for School-Age Children	Measured: BMI	No association

First author, year and country	Follow-up duration	Sample (n) /gender (% girls)	Age or grades at baseline	Psychological predictor: Measurement tool	Adiposity measures	Results
Jansen, 2008 (Netherlands)	3 years	N=787 (49%)	9-10 years	Depressive symptoms and social anxiety : Rotterdam Youth Health Monitor RYM questionnaire, Short Depression Inventory for Children, Dutch social anxiety scale for children	Measured: BMI	No association
Chen, 2010 (USA)	4 years	N=543 (100%)	10 years	Depressive symptoms: CSI (reported by parents) (DSM-IV criteria)	Measured: BMI	No association
Hammerton, 2014 (UK)	1-2 years	N=289	9-17 years	Depressive disorder: CAPA (DSM-IV criteria)	Measured: BMI	No association
Child or adolescent emotional problems and adult overweight/obesity						
Pine, 1997 (USA)	8-10 years	N=644	9-18 years	Depressive symptoms: CES-D (DSM-III criteria)	Self-reported: BMI	Depressive symptoms associated with higher BMI
Pine, 2001 (USA)	10 years	N=177	6-17 years	Depressive symptoms: SADS Lifetime Disorders version	Self-reported: BMI	Depressive symptoms associated with high BMI in adulthood
Franko, 2005 (USA)	2-5 years	N=1554 (100%)	16 years	Depressive symptoms: CES-D	Measured and self-reported: BMI	Depressive symptoms associated with high BMI and with obesity
Anderson, 2006 (USA)	22 years	N=661	15 years mean (9-18 years)	Depression and anxiety: DISC (DSM-IV criteria)	Self-reported: BMI	Females: positive relation Males: no association

First author, year and country	Follow-up duration	Sample (n) /gender (% girls)	Age or grades at baseline	Psychological predictor: Measurement tool	Adiposity measures	Results
Richardson, 2003 (USA)	10 years	N=881	11-15 years	Depression disorder: DISC (DSM-III criteria)	Measured: BMI	Females: depression in later adolescence increases risk of obesity in adulthood; no association in early adolescence Males: no association
Ternouth, 2009 (UK)	20 years	N=3359	10 years	Emotional problems: Rutter B scale	Self-reported: BMI	Females: childhood emotional problems predicted weight gain in women Males: no association
Hasler, 2005 (Germany)	21 year	N=591 (50.5%)	19 years	Child depressive symptoms: SPIKE	Self-reported: BMI	Females and males: depressive symptoms before age 17 associated with increased weight gain
Duarte, 2010 (Finland)	18-23 years	N=2209 (0%)	8 years	Depressive symptoms: Depression, CDI Emotion problems (Rutter questionnaire)	Measured: BMI	No association
Wickrama, 2009 (USA)	6 years	N=11,404	12-19 years	Depressive symptoms: CES-D	Self-reported: BMI	No association

CDC: Centers for Disease Control and Prevention; CES-D: Center for Epidemiologic Studies Depression Scale; CBCL: Child Behaviour Checklist; PSS: Perceived Stress Scales; STAIC: State-Trait Anxiety Inventory for Children; CDI: Children's Depression Inventory; SCARED: Screen for Child Anxiety Related Emotional Disorders; MINI-KID: MINI-International Neuropsychiatric Interview for Children and Adolescents; MFQ: Mood and Feeling Questionnaire; SDQ: Strengths and Difficulties Questionnaire; RYM: Rotterdam Youth Monitor; SADS: Schedule for Affective Disorders and Schizophrenia; CSI: Child Symptom Inventory; CAPA: Child and Adolescent Psychiatric Assessment; DISC: Diagnostic Interview Schedule for Children; SPIKE: Structured Psychopathological Interview and Rating of the Social Consequences for Epidemiology.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

5.3 PHYSIOLOGICAL MECHANISMS LINKING EMOTIONAL SYMPTOMS TO OBESITY

In a physiologic level, it has been observed that some brain regions are mainly related to anxiety and correspond to prefrontal cortex circuits. Among them we can find the orbitofrontal cortex and anterior cingulate as well as amygdale, which is responsible for processing emotions and recognizing potential dangerous stimuli. When the amygdale detects a dangerous stimuli, the neuro-endocrinal axis, the parabraquial nucleous and the locus ceruleus are also activated (Strawn *et al.*, 2014). In all this process, neurotransmitters assist to communicate among neurons and act like chemical messengers, for instance: gamma amino butyric acid (GABA), serotonin, noradrenalin, glutamate and dopamine.

Some studies have shown that stress and emotional symptoms can increase overweight and the related adverse metabolic consequences through neuroendocrine changes (Pervanidou and Chrousos, 2011). Repeated activation of the hypothalamic-pituitary-adrenal (HPA) axis mediates stress responses and increases cortisol secretion. This chronic cortisol hypersecretion may cause fat to accumulate in the visceral adipose tissues (Charmandari *et al.*, 2005) through interaction with lipid homeostasis at several levels: lipolysis, adipogenesis, lipogenesis, and the regulation of circulating fatty acids (Peckett *et al.*, 2011). However, intense acute or chronic stress might also lead to hypo-activation of the HPA axis, or hypocortisolism (Pervanidou and Chrousos, 2011). Cortisol response to stress is complex, but both hypo- and hypercortisolism, may be harmful for the body (Chrousos, 2009). According to a meta-analysis, HPA activity is shaped by the person's response to stress, since cortisol output increases with subjective distress and is generally lower in people with post-traumatic stress (Miller *et al.*, 2007). Recent research in schoolchildren indicates that stress is associated with hypercortisolism (Pervanidou and Chrousos, 2011; Michels *et al.*, 2013). Therefore, stress in combination with maladaptive stress-coping strategies could lead to dysregulated cortisol secretion and obesity.

Since cortisol is appetite-stimulating, it also increases appetite and the attraction to calorie-dense foods, mainly by stimulating reward pathways or by influencing other hormones (Dallman *et al.*, 2003; Michels *et al.*, 2013).

6. GENETIC FACTORS, EMOTIONAL SYMPTOMS AND NUTRITIONAL STATUS

Current literature shows that the relationship between emotional symptoms and obesity or food consumption could vary as a result of genetic polymorphisms. Indeed, it is showed that a 12% of the genetic component of depression is shared with obesity, especially in women (Afari et al. 2010). Individuals who are depressed or anxious may show different degree of seeking out palatable food based on underlying genetic differences. Several candidate genes have been studied related with serotoninergic system (serotonin transporter (5-HTT), serotonin receptor (HTR2C), brain derived neurotrophic factor (BDNF), as well as dopaminergic system (i.e monoamine oxidase-A (MAOA), dopamine transporter (SLC6A3), dopamine receptor D2 (DRD2), dopamine receptor (DRD4) and Val158Met COMT gene), which are the most studied (Cecil et al. 2012; Vimaleswaran et al. 2010; Loos et al. 2008; Kring et al. 2009; Fuemmeler et al. 2009; Walter et al. 2015).

This thesis focuses on studying two of these key genes in the serotoninergic neurotransmission (5-HTT and MAOA) and also involved in dopaminergic system (MAOA).

On the one hand, serotonin plays a role in key process such as sleep, appetite, nutrient intake, addiction and some psychiatric disorders (Bellivier *et al.*, 2002; Lowry *et al.*, 2008; Voigt and Fink, 2015). Serotonin is synthetized by tryptophan amino acid, in two enzymatic processes. Firstly, tryptophan is converted in 5-Hydroxytryptophan (5-HTP) by tryptophan hydroxylase enzyme. Secondly, the enzyme 5-HTP

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

decarboxylase acts on 5-HTP and the serotonin is obtained. Once this process is finished, the serotonin is released into the intersynaptic space to be neurotransmitted. But also, serotonin molecules could be inactive by a re-uptake mechanism inside of presynaptic neuron using the serotonin transport (5-HTT). Inside of the neuron, some serotonin is stored in vesicles and the rest is metabolized in 5-hydroxy indole acetic acid by the enzyme MAOA (Delgado and Moreno, 2006).

On the other hand, dopamine is a neurotransmitter that regulates our capacity to feel pleasure, including the motivation to obtain the rewarding proprieties of palatable food and food intake, in part through the mesolimbic dopaminergic system. The dopamine is also metabolized by the enzyme MAOA which influences on the availability of dopamine. Hence, MAOA are involved in dopaminergic system which is essential to reward-induced feeding behaviour. The role of this system in motivation and hedonic response has been implied in the affective disorders.

Several researches have confirmed the crucial role of the serotonergic and dopaminergic neurotransmission systems in the pathophysiology of emotional and behavioural disorders (Gutiérrez *et al.*, 2004; Lowry *et al.*, 2008; Maron *et al.*, 2012; van Strien *et al.*, 2013). However, the dopaminergic function in depression and anxiety has been less studied than the serotoninergic, but some evidence shows a role of dopamine in the pathophysiology of depressive and anxiety state.

6.1 MONOAMINE OXIDASE-A POLYMORPHISM: MAOA

The MAOA is mitochondrial enzyme that metabolizes neurotransmitters such as dopamine, serotonin, and norepinephrine. This enzyme plays an important role in the metabolism of amines, in the regulation of the levels of neurotransmissions and in the intracellular storage of amines (Berry *et al.*, 1994; Jacob *et al.*, 2005).

The genes that code MAOA are localized in the short arm of chromosome X between Xp11.23 and Xp11.4 brands, this gene reaches 60kb,and is constituted by 15 exons (Ozelius *et al.*, 1988; Lan *et al.*, 1989). As Sabol et al. (1998) described, the MAOA gene possesses a variable number of tandem repeats polymorphism (MAOA-uVNTR or MAOA polymorphism) in its promoter region. This polymorphism influence gene transcription and is considered to be a precursor of dopamine and serotonin activity from the affect levels of neurotransmission availability. Five different alleles rise from this polymorphism depending on whether there are 3, 3.5, 4, or 5 copies of a sequence of 30 base pairs. Later, other authors have observed two new variants of this repeated sequence, alleles with 2 copies and alleles with 6 copies. However, their frequency is very low in general population. The described frequencies of different alleles in Spanish population are of 31% of 3 repetitions and 67% of 4 repetitions, followed by 0.8% of 3.5 repetitions and 0.4% of 5 repetitions (Gutiérrez *et al*., 2004).

The most commons forms, 3, 3.5 and 4 repetitions, have been found to affect in different manner on how the efficiency with which the MAOA gene is transcribed (Sabol *et al.*, 1998; Deckert *et al.*, 1999; Denney *et al.*, 1999). The 3.5 and 4 repetition alleles are associated with increased transcriptional efficiency (and more MAOA enzyme, which would lead to increased serotonin degradation) compared to the 3 repetition alleles. In most of the studies, the alleles have been divided into two groups according to their transcriptional activity, resulting in genotypes with low-activity and high-activity alleles (Figure 3) (Sabol *et al.*, 1998; Rivera *et al.*, 2009; Reif *et al.*, 2014). It has also been found some controversial data about the efficiency of transcriptional activity in this 5-copies allele. Some authors considered this as high efficiency transcriptional activity whereas others believe that this variant shows a low efficiency transcriptional activity (Deckert *et al.*, 1999).

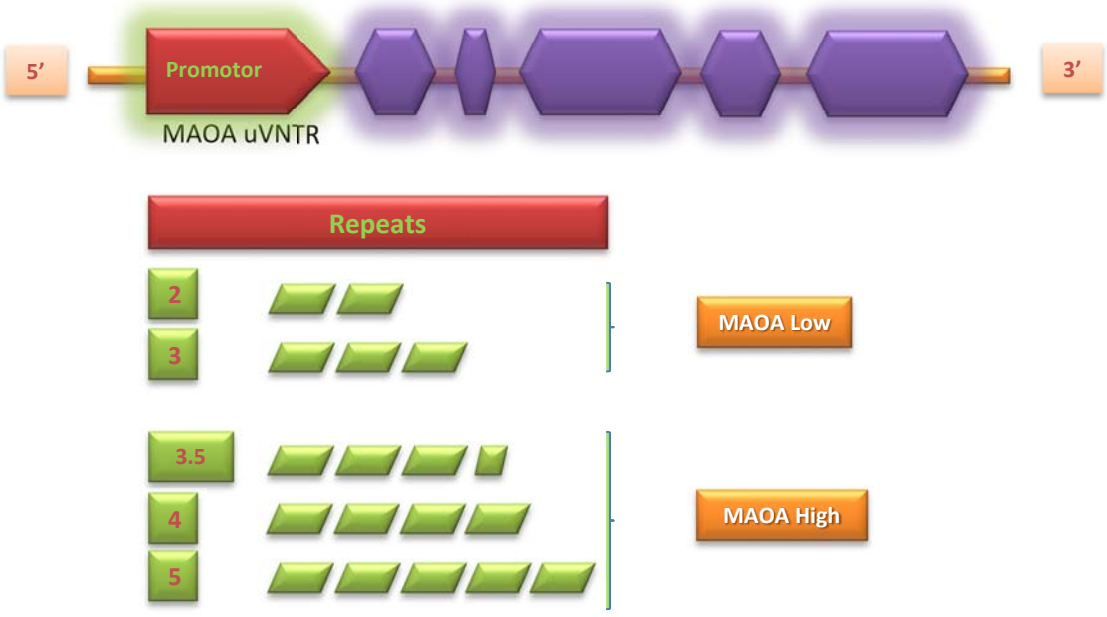


Figure 3. Genotype of MAOA-uVNTR

At the biological level, high MAOA activity degrades the serotonin and dopamine, rendering it inactive in the synapses of the brain. It is also known that a dysfunction of the serotonergic system and dopaminergic system is involved in the development and pathophysiology of affective or behaviour disorders (Bellivier *et al.*, 2002; Lowry *et al.*, 2008; Voigt and Fink, 2015). Indeed, MAOA is also considered a likely depression and anxiety candidate gene because it is also known that MAOA inhibitors have been found to be effective in treating these disorders (Libert *et al.*, 2011). Due to MAOA polymorphism affects the MAOA gene at the transcriptional level and it has been suggested that the polymorphism is involved with diverse mental health conditions in children and adults, for instance major depressive disorder (Rivera *et al.*, 2009) or panic disorder (Reif *et al.*, 2014). However there are

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

mixed results in the studies. The genotype that entails high levels of expression of MAOA is associated with a low tendency to develop antisocial behaviour in subjects who suffer childhood maltreatment (Caspi *et al.*, 2002) with violent and antisocial behaviours (Sjöberg *et al.*, 2008). In contrast, other studies associated a low activity of MAOA with impulsive and violent behaviours (Manuck *et al.*, 2000; Pavlov *et al.*, 2012).

In addition, studies examining the main effects of MAOA variants for the psychopathological disorders of children and adolescents are relatively few (Lavigne *et al.*, 2013) and, in addition, results are mixed or have found many differences between genders. Also, although there is evidence that MAOA genotype interacts with early adversity to predict problem behaviour in human, the interaction of MAOA effects on adolescent remain practically unstudied.

Some researchers have suggested that MAOA polymorphism could affect nutritional status since significant associations were found between MAOA and BMI or obesity. A family-based study found that preferential transmission of the low activity allele spread among subjects with morbid obesity (Camarena *et al.*, 2004). This finding was supported by a large cohort of females from the United Kingdom and this study showed that low-activity MAOA genotype was more frequent among obese females (Need *et al.*, 2006). Similarly, Fuemmerler et al. (2008) observed in a US cohort of young adolescents the association between low activity and obesity among white and Hispanic men but not in women and African-American men. Nevertheless, there are mixed findings of which of the two polymorphisms (low and high) is involved in obesity or excessive weight gain. In this vein, findings in pregnant women show that those with high-activity MAOA genotype gained more weight during pregnancy (Goldfield *et al.*, 2013). In children, high-activity MAOA genotype predicts a higher intake of lipid dense food in boys but not in girls, and no association was observed with weight or other adiposity parameters (Galvão *et al.*, 2012).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

However, due to these mixed results on the polymorphs and obesity and unhealthy obesity-related behaviours, there is a need for more research to confirm the results of which polymorphism being responsible for more vulnerability of weight gain or palatable food consumption.

6.2 SEROTONIN TRANSPORTER POLYMORPHISM: 5-HTTLPR

There is a wide variety of genes directly related to serotonin synthesis and neurotransmission, although the serotonin transporter gene *5-HTTLPR-Linked Polymorphic Region (5-HTTLPR)* has captured the most attention. 5-HTT is a protein responsible for reuptaking, and thus may play an important role in determining the amount of serotonin available in the synaptic cleft (Ramamoorthy *et al.*, 1993) and is involved in ending serotoninergic transmission. 5-HTT produces a rapid elimination and recycling of serotonin released after neuronal stimulation, by this process the action of serotonin is limited in a short period of time. Therefore, it has a critical role in homeostatic regulation of the magnitude, duration and space distribution of signals that reach the serotoninergic receptors (Murphy *et al.*, 2004).

The 5-HTT is encoded by the SLC6A4 gene which is localized in the chromosome 17, in region 17q11.1-q12 (Ramamoorthy *et al.*, 1993). It is constituted by 14 exons that are extended along 31kb and codify protein for 630 key amino acids in serotoninergic neurotransmission (Lesch *et al.*, 1994; Gelernter *et al.*, 1995)**.** There are different kinds of polymorphism described in this gene, including several single nucleotide polymorphism that change the structure or function of the protein transport (Kilic *et al.*, 2003; Prasad *et al.*, 2005). Among all polymorphism described, there is one which has a particular interest in relation with vulnerability to affective disorders: a polymorphism with short (S) and long (L) repeats in the 5-HTTLPR and they differ by the presence or absence of 44base pair: an insertion/deletion in the

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

promoter region (Heils *et al.*, 1996) (Figure 4). The transcriptional activity of transporter gene of serotonin is modulated by the variation in the length of LPR polymorphism, that shows two allelic variants, a short (484 or S) and along (528 or L). The described frequencies of the two polymorphisms in Spanish population are of 57% of L allele and of 43% of S allele. The genotypes are LL (32%), L/S (51%) SS (17%) (Gutiérrez *et al.*, 1998).

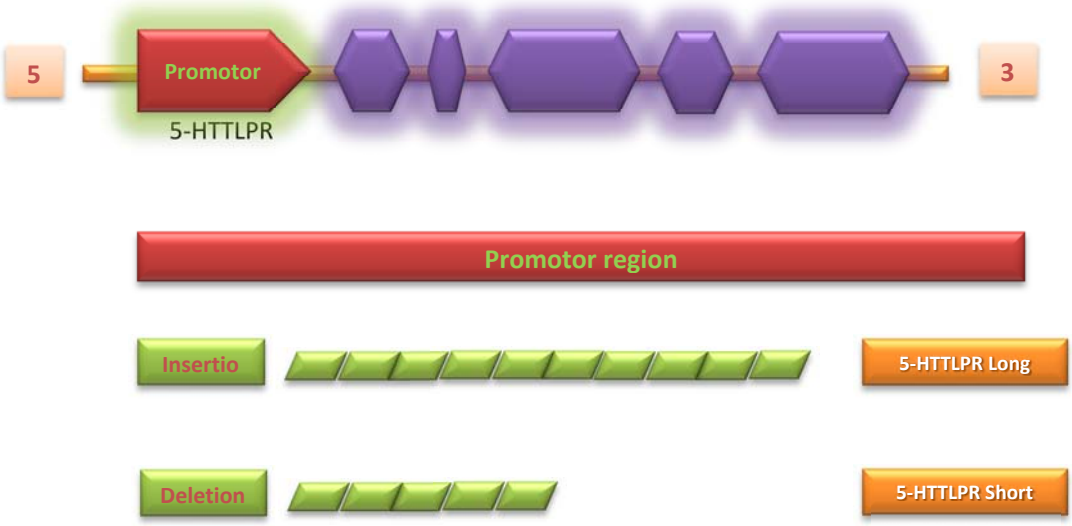


Figure 4. Genotype of Serotonin Transporter Link Promoter Region (5-HTTLPR)

The L polymorphism determines a transcriptional activity three times larger than S polymorphism (Heils *et al.*, 1996; Lesch *et al.*, 1996b). Thus, S allele reduces the efficiency in which the 5-HTTLPR gene is transcribed (i.e reduced 5-HTT-mRNA expression) and S polymorphisms results in a decrease in the serotonin transporter expression and in the reuptake of serotonin in comparison to L polymorphism (Heils *et al.*, 1996, 2002; Murphy *et al.*, 2008). It is suggested that SS and SL genotypes did not differ significantly from each other. This data

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

indicated that in vivo neuroimaging studies higher serotonin transporter availability were associated with the LL genotype compared to SS and SL genotypes (Murphy *et al.*, 2008).

Because 5-HTTLPR SS polymorphism diminishes availability of serotonin, it has been associated with mental disorders like depression. Several authors observed that this polymorphism is associated with a tendency to anxiety and depression or subclinical manifestation of symptoms of depression and anxiety in healthy subjects (Cervilla *et al.*, 2006; Polito *et al.*, 2014). However, these findings could not be considerate as conclusive and require more research (Anguelova *et al.*, 2003; Lasky-Su *et al.*, 2005). With regards to 5-HTTLPR polymorphism and obesity, researches in Argentinean adolescents (Sookoian *et al.*, 2007) and young adult males (Sookoian *et al.*, 2008) found significant associations between polymorphisms of 5-HTTLPR S and overweight. In addition, in a US sample of young adults, this gene was found to be associated with obesity, primarily in men (Fuemmeler *et al.*, 2008). In this vein, it is suggested that S allele is associated with an increase of appetite mainly from sweets. Sweet food consumption is associated with a fast increase of glucose and, as a consequence, higher levels of insulin are produced in blood. This release of insulin in blood, caused by sweet food, increases the concentration of neutral amino acids (similar to tryptophan) and also increases the concentration of the transporter that facilitates that tryptophan synthesise serotonin (Markus et al., 2008). Also, it has been showed that the administration of a serotonin antagonist increased caloric intake and feeling of hunger.

Under this evidence, it is proposed that emotional disorders and obesity could share common pathophysiological elements of the serotoninergic and dopaminergic neurotransmitter system.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

6.3 INTERACTION OF GENETIC FACTORS AND EMOTIONAL SYMPTOMS AND NUTRITIONAL STATUS

The study of genetic factors is complex since interactions gene-x-gene and gen-x-environment could also occur. Specially, candidate gene-x-environmental interaction studies tested the hypothesis that the effect of some environmental variable on some outcome measure depends on a particular genetic polymorphism (Keller, 2014). The interaction of specific alleles with depressive or anxiety symptoms could be important to understand gene-x-environment interactions, since depressive symptoms have been linked with obesity and dysregulation in eating (for instance hyperphasia and loss appetite or different food consumptions (Faith *et al.*, 2002)). Given that emotional symptoms and genetic factors have been associated with obesity and food intake (especially high calories of fat and sweet food) and that food intake patterns and emotional symptoms appear to share common underlying genetic correlates, there may be potential synergies that occur with these two factors that influence obesity and food intake pattern.

The results are less clear about MAOA genotype since there are inconsistences of which type of polymorphisms is associated with obesity and if there are influences on the emotional status. Although it has been observed that low-activity MAOA genotype was more frequent in obese subjects (Camarena *et al.*, 2004; Need *et al.*, 2006), other studies found that adult males with MAOA-H polymorphism showed more depressive symptoms and, on the contrary, reduced risk of obesity. It could be supposed that MAOA-H genotype protects against obesity in case of depression among adolescents males (Fuemmeler et al. 2009). However, MAOA-L polymorphism along with depressive symptoms was associated with a greater intake of calorie but non-sweet food (Agurs-Collins and Fuemmeler, 2011).

Fuemmeler et al. (2008) showed that adolescents with depressive symptoms and S allele of 5-HTTLPR were at risk of obesity and S allele

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

of 5-HTTLPR increased more BMI from adolescence to adulthood. In addition, S allele of 5-HTTLPR polymorphism in adolescents with neuroticism traits has been associated with higher energy intake preferentially from sweet food (Capello and Markus, 2014). Moreover, adolescents with SS or SL genotype showed higher scores of emotional eating (van Strien *et al.*, 2010).

Therefore, genetic factors may play a fundamental role in the development of emotional symptoms and obesity. According to the Gene-x-Environment hypothesis, genetic factor could have a moderate or synergic effect on the relation to emotional symptoms and obesity-related behaviour. Thus, this could help to understand mixed findings and explain gender differences observed.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Hypothesis and Objectives

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

HYPOTHESIS AND OBJECTIVES

HYPOTHESIS

The main hypothesis is that the presence of emotional psychopathology is related to unhealthy dietary patterns and weight gain during adolescence, and this relation could be moderated by genetic factors such as MAOA and 5-HTTLPR polymorphism.

In addition, we hypothesised that the intervention aimed at enabling children to learn to cope with emotions (i.e emotional regulation) could be effective in prevention and treatment programs against obesity.

MAIN OBJECTIVE

To assess the effect of emotional psychopathology on dietary intake and adiposity in a school-based population from preadolescence to adolescence, according to the gender. We examine the influence of genetic factors on this relationship.

SPECIFIC OBJECTIVES

1. To describe diet, anthropometric and body composition characteristics, and physical activity according to emotional symptoms.

2. To assess the effect of emotional symptoms on food consumption, dietary pattern, diet quality and physical activity.

3. To assess the effect of emotional psychopathology on anthropometry and body composition.

4. To analyse the effect of emotional symptoms on anthropometric and body composition parameters and dietary patterns in relation to genetic polymorphism of MAOA and 5-HTTLPR.

5. To develop a conceptual framework model for the role of emotion regulation in the prevention and treatment of childhood obesity.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Material and methods

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

MATERIAL AND METHODS

1. STUDY DESIGN

This study was an epidemiological longitudinal study from preadolescence to adolescence The baseline phase (preadolescence) was a screening phase of emotional symptoms of depression and anxiety disorders, a risk sample of emotional symptoms and a control sample were selected and diagnosis of disorders were confirmed. The follow-up phase (adolescence) was conducted 3 years later (Figure 5).

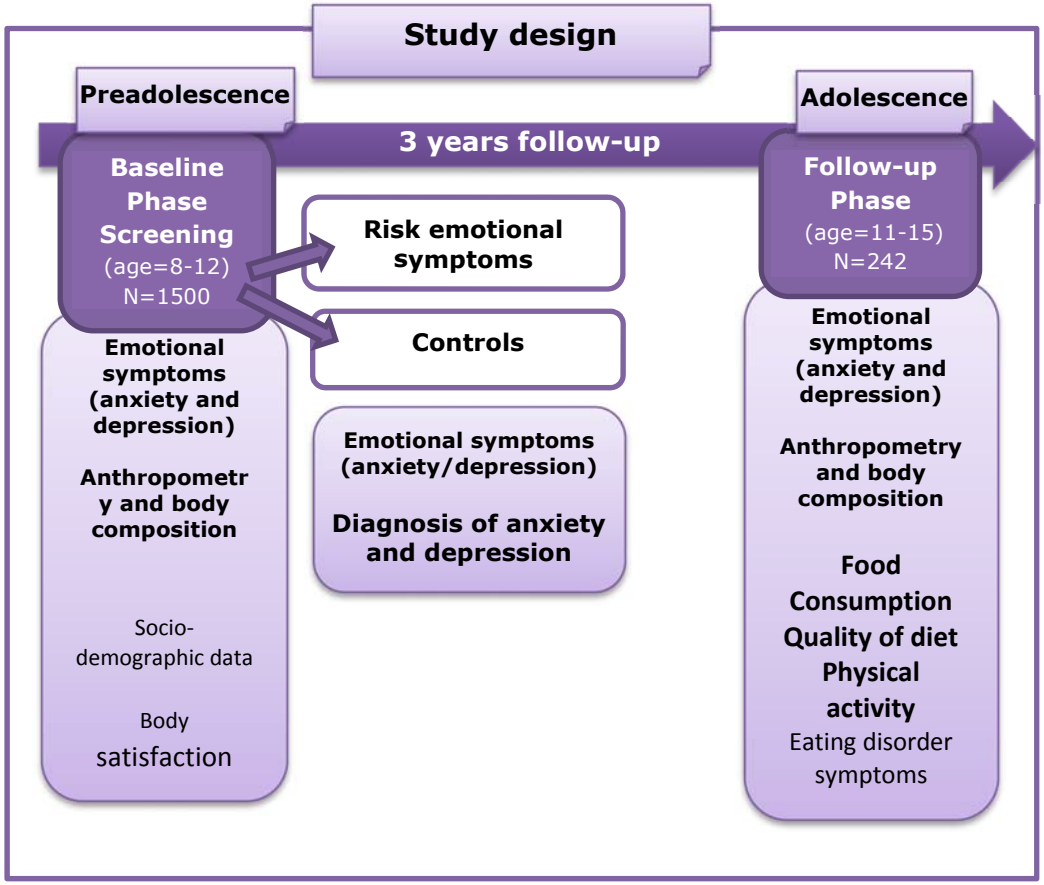


Figure 5. Study design

2. PARTICIPANTS

The study began in 2007 and 2,023 children of 4th, 5th and 6th grade were invited to participate in an epidemiological follow-up study of depression and anxiety disorders that began in the academic year 2006/2007 in the town of Reus (a medium-sized Spanish town of 100,000 habitants). The children came from thirteen primary schools randomly chosen from the towns' state schools and state-subsidised private schools (7 state schools and 6 state-subsidised private schools). In baseline phase, 1,514 children between 8 and 12 years old (10.23±1.23 years old) agreed to participate (720 boys and 794 girls). Seven children were excluded by incomplete psychological data. The final sample at the baseline phase was 1507. Screening questionnaires for anxiety and depression were used to select a sample at risk of emotional problems and a risk-free control sample. The control group was selected randomly, it was chosen from those without risk of emotional psychopathology, matching for age, gender and type of school. One year later, the selected sample was invited to continue participating to confirm diagnosis of depression and anxiety disorder. There were 562 subjects (254 boys and 308 girls) between 9 and 13 years of age (11.25±1.04 years old) who participated, from which 405 (72.1%) were at risk of an emotional disorder and 157 (27.9%) were controls. The attrition of the risk subjects was 16%. If a control subject was invited to participate and declined, we selected another participant with similar characteristics from the baseline phase.

At the follow-up phase, three years after the baseline phase, the selected subjects were invited to participate in the follow-up phase and 242 subjects (95 boys and 147 girls, mean age was 13.52±0.94 years old) participated. There were no emotional, anthropometric and socio-demographic differences between subjects who participated in the follow-up phase and subjects who dropped out in this last step of the study. However, there were differences related to socioeconomic status

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

(SES): low SES participants were associated with higher dropout rates than medium or high SES participants ($\chi^2_{2.561}$=13.557; p =0.001).

We obtained complete data of depressive and anxiety symptoms in 238 participants. This final sample was classified into two groups according to the presence of emotional symptoms: 1) Control group: those scoring below the cut-off for anxiety and depression questionnaires in the baseline and follow-up phases (n=84); 2) emotional symptoms: those with a score equal to or above the cut-off of anxiety and/or depression questionnaires in the baseline and/or follow-up phases (n=154). Due to lack or uncompleted data, the size of the sample was lesser in some analysis according to the main variables. Completed data of anthropometric and body composition were obtained in 229 participants, of food consumption in 165 participants, of MAOA polymorphism in 228 participants and of 5-HTTLPR in 205 participants.

3. PROCEDURE

The project was approved by the Rovira i Virgili University ethics committee for research on individuals and received permission from the Ministry of Education of the Government of Catalonia. A representative sample of subjects came from 13 state and state-subsidized private schools from Reus and they were chosen randomly among 26 schools that belonged to 5 representative town areas. Each school's board of governors was subsequently asked to participate in the baseline and follow-up phases. The parents provided written informed consent in the baseline phase and another one when they were again invited to participate in the follow-up phase.

We conducted the study in two phases:
Baseline phase: During preadolescence, in the baseline phase which took place throughout the academic year 2006/2007, we did a screening process of psychological symptoms (depression and anxiety).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

A child was considered to be at risk of emotional psychopathology if he/she had a score equal to or greater than cut-off on the Screen for Children's Anxiety Related Emotional Disorders (SCARED (Birmaher, 1997) and/or Children's Depression Inventory (CDI) (Kovacs, 1985). For the control group, one child without risk of emotional psychopathology was selected for every three children at risk of emotional psychopathology, matching for age, gender and type of school. We also recorded anthropometric, body composition, body satisfaction (*Body Areas Satisfaction Scale* (BASS)) and socio-demographic data. One year later, during the academic year 2007/2008, subjects at risk of emotional symptoms and a control group without risk were reassessed by the same screening tests (SCARED and CDI) and we evaluated individually the presence or absence of a diagnosis of anxiety or depression disorder using a M.I.N.I-kid structured interview.

Follow-up phase: In the follow-up phase, which took place during the academic year 2009/2010, subjects who agreed to participate completed self-reported questionnaires on psychopathological symptoms (based on SCARED, *Yoth Inventory*-4 and *Eating disorder inventory-2)*, diet quality by Mediterranean diet adherence (*Krece plus food questionnaire*) and physical activity (*Krece plus short physical activity test)* as well as anthropometric and body composition parameter were measured. A saliva sample was also collected from participants and DNA was extracted for the subsequent analyses of the MAOA and 5-HTTLPR polymorphisms. Parents also completed questionnaires about the children's food consumption using a validated food frequency questionnaire. After assessing the children, we gave them an envelope containing these questionnaires for their parents. Once the questionnaires had been completed, they were returned to the school in a sealed envelope, and collected by a researcher.

The participants completed the questionnaires in groups of three or four in the students' classroom during regular school hours. Researchers

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

gave the children instructions on how to answer the test and helped them during the session. On the other hand, anthropometric and body composition measures and structured diagnostic interview were taken individually to ensure privacy of the participants.

4. INSTRUMENTS

4.1 PSYCHOPATHOLOGY ASSESSMENT

Screen for Childhood Anxiety and Related Emotional Disorders (SCARED) (Birmaher *et al.*, 1997). This is a 41-item questionnaire used in the pediatric population to screen for anxiety symptoms. The questionnaire was designed based on clinical studies of the anxiety disorders in the DSM-IV-TR. We used the validated Spanish version (Vigil-Colet et al., 2009) which considers four factors in the factorial analysis: somatic/panic, social phobia, generalized anxiety and separation anxiety. It had good levels of reliability (overall Cronbach's alpha of 0.86). A score of 25 was considered the cut-off to first selection sample (sensibility (75.9%); specificity (68.5%) (Canals *et al.*, 2012). However, to classify the subjects according to presence or absence of emotional symptoms during the study, a 32 score was considered the cut-off point for risk of anxiety in the follow-up since it had a greater specificity (88.8%) that ensured greater severity of anxiety. The SCARED was administered in the baseline and follow-up phases.

Children's Depression Inventory (CDI) (Kovacs, 1985). This is a 27-item questionnaire for people aged 7–17 years old. It assesses depressive symptoms in the cognitive, affective and behavioral spheres. The Spanish version has good internal consistency and good test–retest reliability (Cronbach's alpha between 0.70 and 0.94). We used a score

of 17 as the cut-off point for depressive symptoms (Kovacs *et al.*, 2004). The CDI was administered in the baseline phase.

Youth's Inventory-4 (YI-4) (Gadow and Sprafkin, 1999). YI-4 is a 120-item self-report rating scale that evaluates DSM-IV symptoms of emotional and behaviour disorders in youths aged 12 to 18. In this study, the Youth's Inventory-4 demonstrated satisfactory internal consistency (α=0.95). To obtain our results, we considered the following categories: depression, anxiety, eating disorder symptoms, attention deficit hyperactivity disorder and conduct disorder. Also, depression category was used to create the variable *emotional symptoms* (in the baseline and follow-up phases). The presence of depressive symptoms was considered if they exhibited symptoms of major depression and/or dysthymia. The YI-4 was administered in the follow-up phase.

Body Areas Satisfaction Scale (BASS) (Cash and Szymanski, 1995). This scale assesses an individual's degree of satisfaction or dissatisfaction with 10 body areas. The scale rates satisfaction with each different body part with a score of 1–5. BASS were administered in the baseline phase.

Eating Disorder Inventory-2 (EDI-2) (Garner, 1991). EDI-2 is a 91-item self-report measure of the cognitive and behavioural characteristics commonly associated with anorexia nervosa and bulimia nervosa. We used the validated Spanish version (Garner, 1998). Responses were made on a 6-point Likert-type scale ranging from *never* to *always.* We used 29 of the 91 items that correspond to the four subscales (Drive for thinness, Bulimia, Body dissatisfaction and perfectionism subscales). In this study the EDI-2 internal consistency was α=0.80. The EDI was administered in the follow-up phase.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

MINI-International Neuropsychiatric Interview for Kids (MINI-Kid) (Sheehan *et al.*, 1998). This is a structured diagnostic interview for children aged 6–17 years old, based on DSM-IV and ICD-10 criteria. The MINI-Kid was organized into diagnosis sections. All questions had a binary response format (yes/no). The administration time was approximately 30 min. The reliability and validity of this interview has been demonstrated in a recent study (Sheehan *et al.*, 2010). Mood disorders and anxiety disorders present good psychometric properties (AUC= 0.81, k= 0.56, sensitivity= 0.85, specificity= 0.76; and AUC= 0.84, k= 0.59, sensitivity= 0.90, specificity= 0.77, respectively). This study assessed the diagnosis of major depressive episode and dysthymia, as well as anxiety disorders: panic disorder with or without agoraphobia, separation anxiety disorder, generalized anxiety disorder and social phobia.

4.2. FOOD CONSUMPTION, DIET QUALITY AND PHYSICAL ACTIVITY

Food Frequency Questionnaire (Trinidad-Rodríguez *et al.*, 2008). This is a semi-quantitative food frequency questionnaire validated previously in the adult and adolescent population of Reus. This questionnaire contains 45 items that ask about the usual frequency of consumption per week or per month of food and beverages over the previous year. The frequency categories were converted to a consumption frequency per day or per week. The size and weight of serving portions were standardised, and we calculated grams per day for each item and estimated daily energy intake using the French Regal food composition table (Favier, Ireland-Ripert, Toque and Feinberg, 1997). We also calculated the percentage of nutrient intake adequacy to recommendation using the Recommended Dietary Intake of nutrients created by the Spanish population (Moreiras *et al.*, 2013). We used the adequacy intake value of Institute of Medicine tables (2006) for those nutrients which recommended dietary intake were not available.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Krece plus food questionnaire (Serra-Majem, Aranceta-Bartrina, Ribas-Barba, Sangil-Monroy and Pérez-Rodrigo, 2003). This test determines dietary quality. It consists of 16 items, with a score of 1 or -1 for each item. The maximum possible score is 11, and the minimum is -5. The subjects were classified into three categories according to their total score on the questionnaire; the participants' adherence to Mediterranean diet was thus classified as high (total score ≥ 9), medium (total score 6-8) or low (total score ≤ 5).

Krece Plus short physical activity test (Román-Viñas, Serra-Majem, Ribas-Barba, Pérez Rodrigo and Aranceta-Bartrina, 2003). This test consists of two questions for quick screening of physical activity (hours spent doing physical activity) / inactivity level (hours spent watching television and playing videogames). Each question has six possible responses, with a score of 0–5. The maximum score for the test is 10 and the minimum is 0. According to the total score on the questionnaire, individuals are classified into three categories that correspond to the physical activity level: good (total score between 9 to10 for boys and between 8 to 10 for girls), regular (total score between 6 to 8 for boys and between 5 to 7 for girls), and bad (total score ≤ 5 for boys and total score ≤ 4 for girls).

4.3 OBESITY ASSESSMENT

The *anthropometric parameters* evaluated in the baseline and final phase were weight, height and waist circumference (WC). The measurements were taken with the subject in a standing position. Weight was measured using the Tanita® TBF-300 scale, which has an accuracy of 100 g and a maximum weight of 200 kg. WC was measured using a flexible and inelastic tape and height was measured to the nearest ± 1 mm using an inextensible tape measure. WC was measured at the midpoint between the iliac crests and the lower costal margin at the end of gentle expiration, without clothes. Weight and height were

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

measured with light clothing, barefoot and without heavy objects in pockets. The BMI (kg/m2) was then calculated and standardized (BMI z-score or zBMI), adjusting for age and gender using data obtained from Sobradillo et al. (2004) for the Spanish population. Moreover, we used the International Obesity Task Force cut-off points to classify subjects according to their BMI as: underweight, normal-weight, overweight and obesity (Cole *et al.*, 2000, 2007). In addition, in the follow-up phase other anthropometric measurements were taken as hip circumference and skinfold. Anthropometric measurements were measured by highly trained nutritionists following standard guidelines from the International Society for the Advancement of Kinanthropometry (ISAK) (Stewart *et al.*, 2011). Hip circumference was measured using a flexible and inelastic tape. It was measured at the point yielding the maximum circumference over the buttocks, with the tape held in a horizontal plane, without clothes. Skinfold thickness were measured to the nearest 0.1mm using a Holtain skinfold calliper. The skinfolds were taken at the following sites: triceps, biceps and subscapular. We calculated the ratio of subscapular to triceps skinfold as index of truncal obesity (other measure of abdominal fat). The triceps skinfold was taken at halfway between the acromion and the olecranon in the posterior surface of the arm. It was taken perpendicularly to the long axis of the arm, around the back of the arm, and intersecting the projected line with a vertically in the middle of the arm. The biceps skinfold was measured at the same level as the triceps skinfold, directly above the centre of the cubital fossa. It was taken perpendicularly to the long axis of the arm, around it to the front of the arm, and intersecting the projected vertical line in the middle of the arm. The subscapular skinfold was measured about 20 mm below the tip of the scapula at an angle of 45º in a line running obliquely downward to the lateral side of the body. Briefly, the skinfold was taken firmly between thumb and forefinger and pulled away slightly from the underlying tissue. At the moment that the calliper jaws were applied to the skinfold, the thumb and forefinger were removed and a reading after 2 or 3 seconds. The complete set of anthropometric

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

measurements was performed three times not consecutively. Mean values were obtained from the three measurements.

The *body composition* was measured by bioelectrical impedance using TANITA® TBF-300 body composition analyser. Data of body fat, fat free body mass, and water content were obtained. Our results were expressed as %BF. The %BF was calculated by TANITA using a standard formula that combines impedance measure and weight with height, gender and age. Furthermore, we used the McCarthy et al. (2006) cut-off points to classify subjects according to their body fat as: normal-weight and overweight/obesity.

4.4 SOCIO-ECONOMIC STATUS

A socio-demographic questionnaire designed by the authors for this study was used to assess the socio-demographic characteristics of the sample. The socio-economic level was calculated according to the parents' professions and education, using the Hollingshead index (Hollingshead, 2011). This index allows the social status of each individual to be determined by categorizing his or her occupation into one of nine categories (from unskilled work to highly skilled work) and his or her level of education into one of seven categories (from non-completed primary education to completed higher education). The status score is estimated by multiplying the occupation scale value by a weight of five and the education scale value by a weight of three and then combining the two scores. We thus determined family SES on a scale from 0 to 66. This gave us three categories (low, medium and high). We considered scores under 22 to be low, scores of between 23 and 44 to be medium, and scores over 44 to be high.

4.5 DNA EXTRACTION AND GENOTYPING

Genomic DNA was extracted from buccal cells derived from Oragene•DNA self-collection kits (DNA, Genotek) (Figure 6). We collected saliva samples after the children had not eaten or drunk anything for at least 30 minutes.

The DNA was extracted, purified and amplified, and quantified at the BioBank of the Institute for Health Sciences Research (IRCIS) at Sant Joan University Hospital in Reus (Spain).

Figure 6. Method to collect saliva sample: Oragene•DNA self-collection kits

The genotyping was undertaken by Scientific and Technical Services of Rovira i Virgili University. The polymorphism Monoamino Oxidasa A-*uVNTR* (MAOA) polymorphism and Serotonin Transporter-linked polymorphism region (5-HTTLPR) gen were analysed.

Monoamino Oxidasa A-uVNTR (MAOA-uVNTR or MAOA polymorphism): The polymorphism 30 base pairs variable number of tandem repeats (VNTR) in the promoter of the MAOA gene was genotyped using a previous published protocol (Haberstick *et al.*, 2005). Briefly, polymerase chain reaction (PCR) was performed in a total volume of 20

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

µl containing 20ng of DNA, using the primers forward, 5-D2-ACAGCCTGACCGTGGAGAAG-3_and reverse, 5_-GAACGGACGCTCCATTCGGA. PCR products included five possible fragment sizes—291, 321, 336, 351, and 381 bp (2, 3, 3.5, 4 and 5 repeats, respectively) —and were classified into two groups. The first group combined those with the 2-repeat and 3-repeat alleles and was subsequently referred to as the low-activity group of the MAOA (MAOA-L). The second group combined those with the 3.5-repeat, the 4-repeat, and the 5-repeat and is subsequently referred to as the high-activity group of the MAOA (MAOA-H). The MAOA gene is located on the X chromosome; therefore, a heterogeneous genotype does not exist in men. We classified the heterogeneous genotype (i.e. 2/3.5, 2/4, 2/5, 3/4, 3/5, 3.5/4, 3.5/5, 4/5) of girls in to the high-activity group, as in other studies (Reif *et al.*, 2014).

Serotonin Transporter-linked polymorphism region (5-HTTLPR): The serotonin transporter gene (*SLC6A4)*, contains a 44 base pairs insertion/deletion in the 59 regulatory region of the gene (Heils *et al.*, 1996). The insertion/deletion in the promoter appears to be associated with variations in transcriptional activity: the long variant (528 bp, L) has approximately three times the basal activity of the shorter promoter (484 bp) with the deletion (Lesch *et al.*, 1996). This polymorphism was genotyped using a previously published protocol (Anchordoquy *et al.*, 2003) which is a modification of the method of Lesch et al. (1996). Briefly, polymerase chain reaction (PCR) was performed in a total volume of 20 microliters containing 20 ng of DNA, using the primers forward, 5-D2-ATGCCAGCACCTAACCCCAATGT-3' and reverse 5'–GGACCGCAAGGTGGGAGGGA-3'. These primer sequences yield products of 528 and 484bp. The amplification yield distinguished bands at 484bp (short "S" allele) and 528bp (long "L" allele) (Figure 7). In compliance with previous work (Lesch *et al.*, 1996), tri-allelic variants (SS, SL or LL) were reclassified into bi-allelic model as follows: SS and SL were classified as SS/SL and LL was classified as LL.

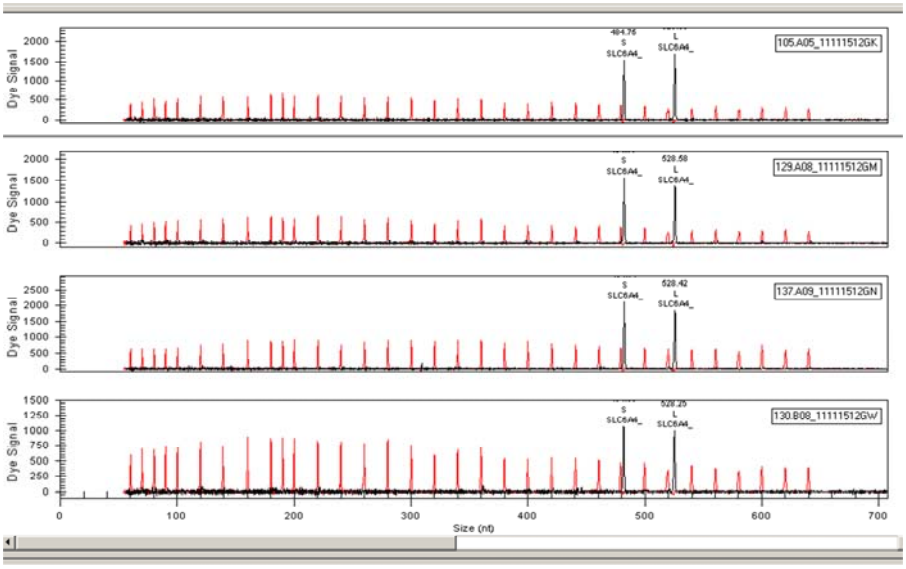


Figure 7. Representation of the band for each genotype

Amplification of target sequences by polymerase chain reaction: Polymerase chain reactions contained 1 ml of genomic DNA (20 ng), 10% DMSO (Hybra-Max® grade; Sigma, St. Louis, MO), 1.8 mM MgCl2, 180 microM mixed deoxynucleotides (with 79-deaza-29-deoxyGTP), substituted for one half of the dGTP, forward and reverse primers (vrnt-MAOA, 200nM; 5HTTLPR, 600 nM;) and 1 U of AmpliTaq Gold® polymerase (AmpliTaq® gold DNA polymerase, Applied Bio systems), in a total volume of 20 ml. Amplification was performed using touchdown PCR cycling: A 95°C incubation for 10 min was followed by two cycles of 95°C for 30 s, 65°C for 30 s, and 72°C for 60 s. The annealing temperature was decreased every two cycles from 65°C to 57°C in increments of 2°C (10 cycles total), and a final 30 cycles of 90°C for 30 s, 65°C for 30 s, and 72°C for 60 s and a final 30-min incubation at 72°C. Analyses were run using CEQ8000 Beckman Coulter sequencer. The PCR products were analyzed on a 2% agarose gel with Ethidium bromide. At the end, results were observed by UV transilluminator for the examination of the product bands.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

All trials were repeated twice. If the results were negative or discordant, the trials were repeated 3, 4, or 5 times. Nine subjects were unsuccessfully genotyped for the MAOA gene and fourteen subjects were unsuccessfully genotyped for the 5-HTTLPR gene. They were dropped from all genetic analyses.

4.6 SUMMARIZE OF VARIABLES IN THE STUDY

Independent variables

- Emotional variables:
  - *Depressive disorders:*
    - Depressive symptoms
    - Depression diagnostic: Major depression Episode and dysthymia
  - *Anxiety disorders: Anxiety symptoms and diagnostic*
    - Subtype of anxiety symptoms and disorders: panic disorder with or without agoraphobia, separation anxiety disorder, generalized anxiety disorder and social phobia

Based on the presence of anxiety and depression along the study we created a new variable:

- *Emotional symptoms:* The sample was classified into two groups according to the presence of emotional symptoms in any of the phases of the study:
  - *Control group:* those scoring below the cut-off for anxiety and depression questionnaires in any of phases
  - *Emotional symptoms:* those with a score equal to or above the cut-off of anxiety and/or depression questionnaires in any of in any of phases.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

- Genetic variables: The genetic polymorphisms were classified as:

  - *MAOA*:
    - Low-activity MAOA (MAOA-L): Homozygote MAOA polymorphism with 2 and 3 repetitions.
    - High-activity MAOA (MAOA-H): Homozygote MAOA polymorphism with 3.5, 4 and 5 repetitions and heterozygotes.

  - *5-HTTLPR*:
    - LL: Homozygote 5-HTTLPR with long alleles
    - SS/SL: homozygote 5-HTTLPR with short alleles and heterozygote 5-HTRR with short and long allele.

Main outcome variables:

- *Weight, body fat and abdominal fat gain*: change in WC, change in BMI and change in %BF. The change in anthropometric and body composition measurements from preadolescence to adolescence was calculated as the difference between the final values in adolescence (follow-up phase) and the baseline values in preadolescence (baseline phase).

- *Dietary patterns:* the dietary pattern variables were used as a quantitative variable (measured on the z-score scale) and as a qualitative variable (categorized into tertiles). Tertile 1 was low adherence (the lowest score), tertile 2 was medium adherence and tertile 3 was high adherence (the highest score) to each dietary pattern.

- *Mediterranean diet adherence:* it was used as a quantitative variable (measured on a scale) and as a qualitative variable (low, mid and high adherence).

5. NARRATIVE REVIEW

The narrative review focused on an update analysis of the most relevant psychological issues in childhood obesity, especially emotion regulation. In order to identify the relevant articles on this topic, we conducted a comprehensive and non-systematic electronic database search in MEDLINE, Web of Knowledge and Scopus of observational and interventional/experimental literature concerning the emotion regulation-obesity link, its underlying concepts and emotion regulation intervention techniques in prevention and treatment of obesity in children. The first searching strategy was to identify the papers that focus concretely on emotion regulation and obesity or unhealthy-obesity behaviour. Later, due to the limited literature and insight of the potential and novel relation of emotion regulation on obesity, we carried out a second search strategy to develop and construct this novel conceptual framework for the role of emotion regulation and obesity in children. Also, a manual review of references from key studies and review articles were checked to ensure a comprehensive search.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

6. STATISTICAL ANALYSIS

Statistical analysis was performed by using SPSS 22.0 software. The results were expressed as means and standard deviations for the quantitative variables, and as percentages for the qualitative variables. We verified compliance with the statistical tests' conditions of use. We therefore used the chi-square test, the Student *t*-test and analysis of variance adjusted for the Bonferroni and Pearson correlation depending on the types of variables compared.

In analysis of anthropometric and body composition variables, the degree of non-independence of observations from children nested within the same school can be estimated using intraclass correlation coefficients (ICC) (Kenny *et al.*, 2002; Pardo *et al.*, 2007). We found no evidence to suggest that observations were non-independent for the outcome variable: "change in WC" (ICC=0.0827), "change in BMI" (ICC=0.0001), and "change in %BF" (ICC=0.0192, ps>0.05). Therefore, we applied traditional statistical analysis.

The genotypes were tested for deviations from Hardy–Weinberg Equilibrium (HWE). Deviations from HWE could indicate either a problem with the genotype assay or a true association with outcome. The Hardy–Weinberg Equilibrium of the genotype distributions of the girls was approximated for all samples using chi-squared tests.

Principal Components Analysis (Martínez-González *et al.*, 2006) was used to identify the dietary pattern. Dietary patterns based on factor analysis have been used in several settings, and have shown to be suitable for describing usual dietary intake (Newby and Tucker, 2004). First, the 45 items in the food frequency questionnaire were collapsed into 19 food groups (Table 2). A factor analysis (major components) based on 19 food groups was conducted to assess the main dietary patterns. We used parameters similar to those in other studies of dietary patterns (Hu, 2002). The patterns were rotated by orthogonal

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

transformation (varimax rotation) to maintain uncorrelated factors and improved factor interpretation. Only factors with an eigenvalue greater than 1 were retained in the factor solution, and the slope Cattell test or the screen plot was used to confirm the number of factors to retrain.

Table 2. Food groups included in the factor analysis.

Food Groups	**Food included**
Dairy products	Milk, yogurt, cheese
Sweet dairy products	Crème caramel, custard, pudding, chocolate dairy desserts, ice-cream
Breakfast cereals and biscuits	Breakfast cereals and standard biscuits
Baked goods and chocolates	croissants, donut, sweet bun, cream and chocolate cake, biscuits with chocolate-flavoured filling, chocolate bars
Sweets	candies, sugar, honey
Starchy	wheat, rice, pasta, bread
Beans	lentils chickpea, various types of beans
Potatoes	baked, boiled or fried potatoes
Vegetables	leafy green vegetables (lettuce, chard, spinach) cruciferous vegetables (cabbage, Brussels sprouts, broccoli, cauliflower, coleslaw), yellow and red vegetables (carrots, pumpkins, capsicum), other vegetables (cucumber, tomato, beetroot, mushroom, celery, turnip, swede, onion, mixed vegetables, green beans)
Fruits	Citrus fruit (oranges, mandarin, kiwis) other fruits (apple, banana, berries, strawberries, melon, water melon, peach, plum nectarine, apricot, grapes, pineapple) canned fruit, juices.
Nuts	Almond, nut, raisins, currants, hazelnuts, peanuts, pistachios
Meat and cold meat	Lamb, beef, pork, chicken, turkey, offal, minced meat, boiled ham, Parma ham
Fish and seafood	blue fish (salmon, tuna, sardines) white fish (hake, sole, grouper) seafood
Eggs	eggs
Pre-cooked meals	pizza, croquette, hamburgers,
Savoury snacks	chips, salad biscuits and snacks
Soft-drinks	Carbonated and/or sweet drinks (Coca-Cola, Fanta,..)

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Three independent factors or dietary patterns were identified, which explained 37.89% of the total variance. We used a factor loading matrix to extract the weights (factor loading) for each food group. The food groups with a factor load of 0.30 or more were regarded as important contributors to the dietary patterns. The food loading in each dietary pattern is shown in Table 2. Observing these weights, we named the three major factors as dietary patterns. We labelled these patterns as "Sweet and fatty food", "Western" and "Healthy" due to the food that contributed to each pattern. These variables were calculated as linear combinations of the standardised values of 19 food groups, using the factor scores found in the factor analysis as a coefficient. All adolescents received a score for the three dietary patterns measured on the z-score scale. The dietary patterns were categorized into tertils: low adherence (tertile 1, the lowest score), medium adherence (tertile 2) and high adherence (tertile 3, the highest score).

Moreover, several multiple linear and logistical regression models were applied:

- *To test the relation between emotional symptoms and dietary patterns:* multiple logistic regression analysis was applied adjusted to potential confounders (age, SES, BMI, eating disorder symptom score, physical activity score and energy intake).

- *To test which psychological variables predict low Mediterranean diet:* logistic regression adjusted models were applied. Before performing the regression models, collinearity between the variables was assessed by computing Pearson correlations between the candidate variables. We used two models. *Model 1* used baseline phase psychological variables as predictors, anxiety and depression symptoms, and it was adjusted for age, gender, SES, birthplace, family type, school type, BMI risk/control variable. In *model 2*, the predictors were the following psychological variables of the follow-up phase:

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

anxiety and depressive symptoms, eating disorders symptoms, conduct disorder symptoms and attention deficit hyperactivity disorder, and it was adjusted by age, gender, SES, and risk/control variable, %BF, BMI and physical activity factor.

- *Mediational analysis:* A series of regression analyses was conducted to determine if Mediterranean diet adherence mediates the relationship, found in some studies, between emotional symptoms and overweight and obesity. Gender, age and SES were included as covariables. Also, two more mediational models were performed to determine if depressive symptoms mediate the relation between SES and Mediterranean diet, or if Mediterranean diet mediate the relation between SES and overweight/obesity. Gender and age were included as covariables. The analysis was consistent with recommendations regarding mediational analyses in a population-based research (Hafeman and Schwartz, 2009).

- *To assess the effect of psychopathology on changes in anthropometry and body composition:* The multiple linear regression models used the ENTER method for psychopathological variables and the STEPWISE method for the other adjustment variables. The psychopathological variables were as follows: depressive symptoms in model 1; anxiety symptoms in model 2; symptoms of depression, separation anxiety, generalized anxiety, somatic/panic and social phobia in model 3; and diagnosis of panic disorder, separation anxiety disorder, generalized anxiety disorder, social phobia, diagnosis of major depressive episode and dysthymia in model 4. The other adjustment variables were age (years), baseline WC (cm), baseline BMI (kg/m2) and baseline %BF (%), according to the dependent variable in the multiple linear regression model, the Krece Plus diet test and Krece Plus physical activity test scores, and the body areas satisfaction score.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

- *To test effect of genetic factors and emotional symptoms on nutritional status*: multiple linear regression analysis was applied adjusted to potential confounders (age, SES, BMI (kg/m^2), and energy intake).

- *Interaction analysis:* we test various regression models to examine whether there was a moderator effect between 5-HTTLPR or MAOA genotype and emotional symptoms on dietary pattern. We run interaction models of two-way interaction 5-HTTLPR-x-emotional symptoms and MAOA-x-emotional symptoms and three-way interaction 5-HTTLPR-x-MAOA-x-emotional symptoms. A significant moderator effect would be demonstrated by a significant interaction term, whether there were or not a main effect for the moderator. When the interaction was significant, multiple linear regression adjusted analyses were split by genotype.

Analyses were run separately by gender. For all the analyses, the level of statistical significance was a p value <0.05.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Results

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

RESULTS

1. DESCRIPTIVE CHARACTERISTICS OF PARTICIPANTS

1.1 DESCRIPTIVE CHARACTERISTICS OF THE BASELINE SAMPLE

1.1.1 Socio-demographic characteristics

Table 3 shows socio-demographic characteristics of the baseline sample. The baseline sample was composed by 1507 schoolchildren 10.2 (0.9) years old. A 39.4% and 42.5 % of schoolchildren belonged to families with low SES, whereas 18.0% belonged to families with high SES. The majority of schoolchildren were native (87.5%) and lived in nuclear families (85.9%). Also, almost half of the sample attended state-school (43.5%) and slightly over half of them went to state-subsidized private school (56.5%). No significant results were found among genders.

Table 3. Socio-demographic characteristics of the baseline sample according to gender

		Total (n=1507)	**Boys** (n = 715)	**Girls** (n = 792)	*p* value between boys and girls
Age baseline (years) ‡		10.2 (0.9)	10.3 (0.9)	10.2 (0.9)	ns
Gender (% females)		52.6	-	-	
Socioeconomic status	Low (%)	39.4	38.8	40.0	
	Medium (%)	42.5	42.4	42.6	ns
	High (%)	18.0	18.8	17.4	
Family type	Nuclear (%)	85.9	86.7	85.1	ns
	Single parent (%)	14.1	13.3	14.9	
Birthplace	Native (%)	87.5	89.3	85.9	ns
	Foreign (%)	12.5	10.7	14.1	
School type	State school (%)	43.5	46.2	41.1	ns
	State-subsidized private school (%)	56.5	53.8	58.9	

‡Expressed as mean (standard deviation). ns: non-significant . Level of statistical significance: p value<0.05

1.1.2 Psychological characteristics

Psychological characteristics of the baseline sample are shown in table 4. The prevalence of depressive symptoms was 11.4 % (95%CI: 9.8-13.0) and anxious symptoms was 46.7% (95%CI: 44.1-49.2).There were no differences between genders in the prevalence of depressive symptoms (boys: 11.6% (95%CI:9.9-13.22); girls: 11.1% (95%CI: 9.5-12.6), X^2=0.081, p=0.775)) whereas, anxiety symptoms were greater in girls (51.4% (95%CI: 48.8-53.9) than boys (41.3% (95%CI: 38.8-43.7, X^2=15.3, p<0.001). Likewise, girls scored significantly above boys of total anxiety symptoms (girls: 25.3±9.9 scores; *vs* boys: 23.2±10.6 scores), social phobia (girls: 6.3±2.8 scores; *vs* boys: 5.5 ±2.9 scores), generalized anxiety (girls: 6.3±2.8 scores; *vs* boys: 5.9±3.0 scores) and separation anxiety (girls: 8.5± 3.9 scores; *vs* boys: 7.9±4.2 scores).

Table 4. Emotional symptoms of the baseline sample according to gender

	Total (n=1507)		**Boys** (n = 715)		**Girls** (n = 792)		*p* value between boys and girls
	Mean‡	(SD)	Mean	(SD)	Mean	(SD)	
Depressive symptoms (%)	11.4		11.6		11.1		ns
CDI(score)	9.1	(6.1)	8.9	(6.1)	9.1	(6.2)	ns
Anxiety symptoms (%)	46.7		41.3		51.4		**<0.001**
SCARED(score)	24.3	(10.3)	23.2	(10.6)	25.3	(9.9)	**<0.001**
Somatic panic (score)	3.9	(3.5)	3.8	(3.5)	4.1	(3.5)	ns
Social phobia (score)	5.9	(2.9)	5.5	(2.9)	6.3	(2.8)	**<0.001**
Generalized anxiety (score)	6.1	(3.0)	5.9	(3.0)	6.3	(2.8)	**0.004**
Separation anxiety (Score)	8.2	(4.1)	7.9	(4.2)	8.5	(3.9)	**0.002**

‡ Expressed as mean (standard deviation), or percentage (where % shown)
CDI: Children's Depression Inventory; SCARED: Screen for Childhood Anxiety and Related Emotional Disorders; ns: non-significant .
Level of statistical significance: p value<0.05

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

1.1.3 Anthropometric and body composition characteristics

Table 5 shows the anthropometric and body composition parameters of the baseline sample. It can be observed gender significant differences in BMI z-score, body fat and WC. While boys showed higher BMI z-score (boys: 0.2±1.0; vs girls: 0.1±0.9) and WC (boys: 67.9±8.4 cm; vs girls: 66.0±8.0 cm); girls had higher values of percentage body fat (girls: 22.7±8.6%; vs boys: 8.9 ±77.7%).

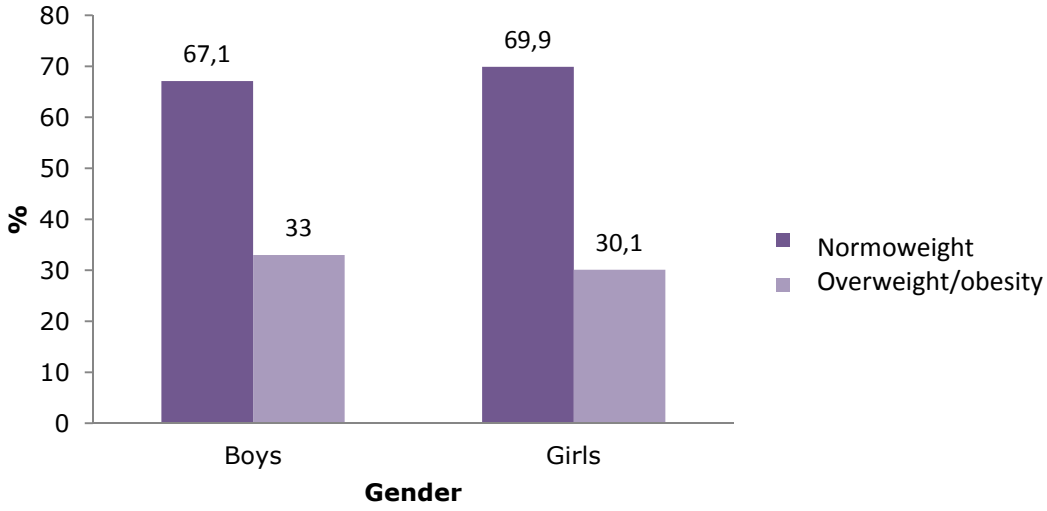
Table 5. Anthropometric and body composition parameters of the baseline sample in preadolescence according to gender

	Total (n=1507)		**Boys** (n=715)		**Girls** (n=792)		*p* value between boys and girls
	Mean	(SD)	Mean	(SD)	Mean	(SD)	
Height (cm)	144.3	(8.3)	144.0	(7.7)	144.6	(8.8)	ns
Weight (kg)	40.2	(9.6)	40.1	(9.5)	10.3	(9.8)	ns
BMI(kg/m^2)	19.1	(3.3)	20.0	(3.3)	19.1	(3.3)	ns
zBMI (score)	0.2	(0.1)	0.2	(1.0)	0.1	(0.9)	**0.031**
Body Fat (%)	20.9	(8.4)	18.9	(7.7)	22.7	(8.6)	**<0.001**
Waist circumference (cm)	66.9	(8.2)	67.9	(8.4)	66.0	(8.0)	**<0.001**

BMI: Body Mass Index; zBMI: z-score of BMI; SD: Standard deviation; ns: non-significant
Level of statistical significance: p value <0.05

Moreover, the frequency of overweight and obesity was 31.4% (95%CI: 27.7- 32.4) in our schoolchildren sample. There were no differences among genders: a 33.0% (95%CI: 30.63-35.37) of boys and a 30.1% (95%CI: 27.7-32.4) of girls were overweight or obese (X^2=1.374, p=0.241) (figure 8).

Figure 8. Prevalence of normoweight and overweight/obesity in the baseline sample



UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

1.2 DESCRIPTIVE CHARACTERISTICS OF THE FOLLOW-UP SAMPLE

1.2.1 Socio-demographic characteristics

The follow-up sample was composed by 242 schoolchildren, 95 boys and 147 girls. Table 6 shows descriptive data for socio-demographic characteristics. We did not find significant differences in age, SES, family type, birthplace and school type by gender.

Table 6. Socio-demographic characteristics of the follow-up sample

		Total (n= 242)	**Boys** (n = 95)	**Girls** (n = 147)	*p* value between boys and girls
Age at follow-up (years) ‡		13.5 (0.9)	13.4 (1.0)	13.6 (0.9)	ns
Gender (% females)		61.9	-	-	
Socioeconomic status	Low (%)	34.8	33.3	35.8	ns
	Medium (%)	44.3	44.1	44.4	
	High (%)	20.9	22.6	19.9	
Family type	Nuclear (%)	84.4	88.2	82.1	ns
	Single parent (%)	15.6	11.8	17.9	
Birthplace	Native (%)	90.6	90.3	90.7	ns
	Foreign (%)	9.4	9.7	9.3	
School type					
	State school (%)	34.8	36.6	33.8	ns
	State-subsidized private school (%)	65.2	63.4	66.2	

‡Expressed as mean (standard deviation)
ns: non-significant Level of statistical significance: p-value <0.05

1.2.2 Psychological characteristics

Table 7 shows the psychological characteristics of follow-up sample in preadolescence and adolescence by gender. In preadolescence, boys showed higher scores of anxiety symptoms, especially somatic panic (p=0.024), while in adolescence girls showed higher scores than boys (p=0.019). Also, body dissatisfaction (p=0.043) and eating disorders symptoms (p=0.028) were higher in girls than boys.

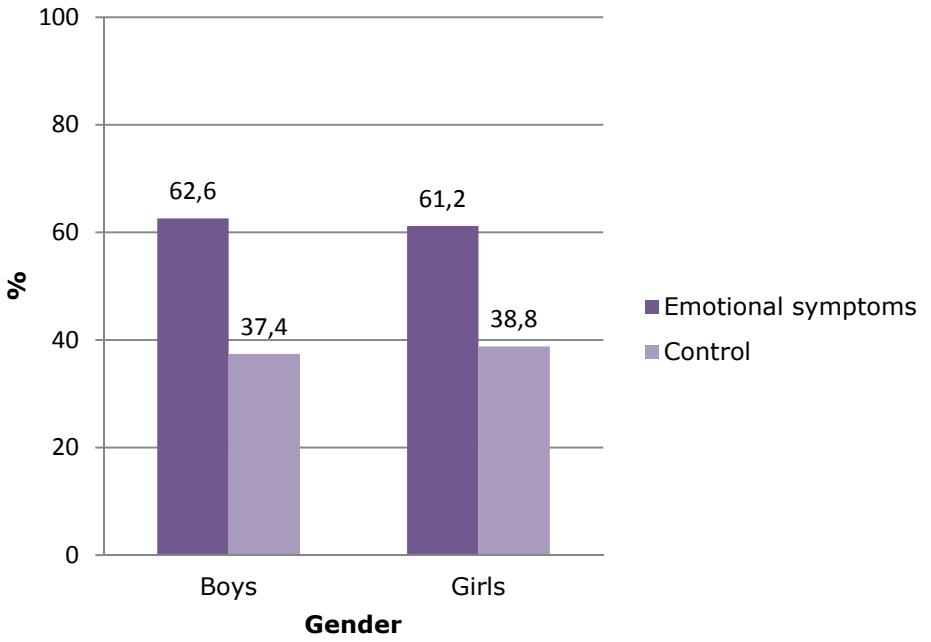
Table 7. Psychopathological symptoms of the follow-up sample in preadolescence and adolescence according to gender

	Total (n=238)		**Boys** (n=91)		**Girls** (n=147)		*p* value between boys and girls
	Mean	(SD)	Mean	(SD)	Mean	(SD)	
Preadolescence[a]							
Depressive symptoms CDI (score)	11.0	(7.1)	11.8	(7.3)	10.5	(6.9)	ns
Anxiety symptoms SCARED(score)	29.6	(10.9)	30.0	(29.3)	29.3	(9.8)	ns
Somatic panic (score)	5.5	(4.0)	6.3	(4.7)	5.0	(3.5)	**0.024**
Social phobia (score)	6.6	(3.0)	6.2	(3.1)	6.9	(2.8)	ns
Generalized anxiety (score)	7.4	(3.3)	7.3	(3.5)	7.4	(3.2)	ns
Separation anxiety (score)	9.9	(4.2)	10.1	(4.7)	9.7	(3.9)	ns
Body satisfaction BASS(score)	30.2	(5.3)	31.2	(5.3)	29.6	(5.3)	**0.043**
Adolescence[b]							
Depressive symptoms YI-4 (score)	14.6	(8.5)	14.0	(8.2)	15.0	(8.8)	ns
Anxiety symptoms SCARED (score)	20.4	(10.3)	18.8	(10.3)	21.4	(10.3)	ns
Somatic/panic (score)	3.3	(3.3)	2.6	(3.1)	3.7	(3.3)	**0.019**
Social phobia (score)	5.4	(3.2)	5.3	(3.3)	3.7	(3.3)	ns
Generalized anxiety (score)	6.3	(3.0)	5.9	(3.5)	6.6	(3.4)	ns
Separation anxiety (score)	5.3	(3.6)	4.8	(3.3)	5.6	(3.8)	ns
Eating disorders symptoms EDI-2(score)	14.7	(11.4)	12.7	(9.2)	16.0	(12.4)	**0.028**

[a]Preadolescence: baseline phase; [b]Adolescence; follow-up phase.
SD: Standard deviation; ns: non-significant; CDI: Children's Depression Inventory; SCARED: Screen for Children Anxiety and Related Emotional Disorders; BASS: Body Areas Satisfaction Scale;YI-4:Youth's Inventory-4;EDI-2: Eating Disorder Inventory-2. Level of statistical significance: p value<0.05

Of 242 schoolchildren, 238 completed psychological test in follow-up phases. Of them, 154 adolescents showed emotional symptoms and 84 did not showed symptoms in any phase and were considered control. Figure 9 depicts the percentage of adolescents with or without emotional symptoms in any phase of the study from preadolescence to adolescence. We observed that a 61.2% of adolescents showed emotional symptoms during adolescence. We did not found significant differences in emotional symptoms by gender.

Figure 9 . Percentage of adolescents with emotional symptoms during the follow-up of the study



The frequency of diagnosis is shown in table 8. In general, 2.1% and 4.6% of subjects showed diagnosis of major depressive disorder and dysthymia, while 21.4% showed diagnosis of any anxiety disorder. We did not observe differences on gender.

Table 8. Percentage of population with diagnosis[a] of emotional disorder in preadolescence according to gender

	Total (n=238)		**Boys** (n=91)		**Girls** (n=147)		p value between boys and girls
	n	(%)	n	(%)	n	(%)	
Diagnosis of major depressive episode	5	(2.1)	3	(3.3)	2	(1.4)	ns
Diagnosis of dysthymia	11	(4.6)	5	(5.5)	6	(4.1)	ns
Diagnosis of any anxiety disorder	51	(21.4)	20	(22.0)	31	(21.1)	ns
Diagnosis of separation anxiety disorder	13	(5.5)	5	(5.5)	8	(5.4)	ns
Diagnosis of generalized anxiety disorder	33	(13.9)	13	(14.3)	20	(13.6)	ns
Diagnosis of panic disorder	6	(12.5)	3	(3.3)	3	(2.0)	ns
Diagnosis of social phobia	13	(5.5)	3	(3.3)	10	(6.8)	ns

[a]MINI-Kid: MINI-International Neuropsychiatric Interview for Kids
ns: non-significant. Level of statistical significance: p value<0.05

1.2.3 Anthropometric and body composition characteristics

Anthropometric and body composition parameters from preadolescence to adolescence are shown in table 9. Girls showed greater values of %BF than boys ($p<0.001$) in preadolescence and adolescence. In adolescence WC ($p=0.038$) and biceps ($p=0.03$), triceps ($p<0.001$) and subscapular ($p=0.002$) skinfold were higher in girls than boys. The change of %BF ($p=0.007$) and WC ($p<0.001$) from preadolescence to adolescence were higher in girls than boys.

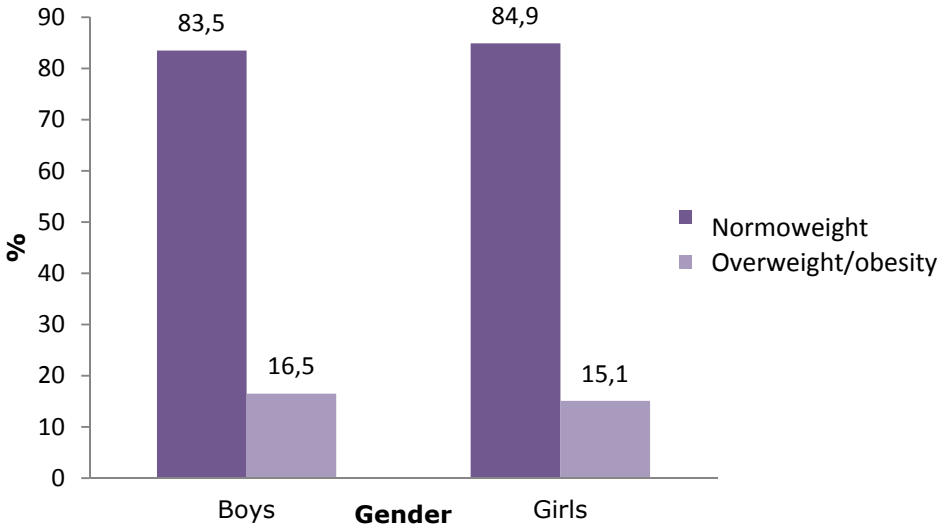
Table 9. Anthropometric and body composition characteristics of the follow-up sample according to gender

	TOTAL (n=229)	**Boys** (n=87)	**Girls** (n=142)	p value between boys and girls
	Mean (SD)	Mean (SD)	Mean (SD)	
Preadolescence[a]				
Height (cm)	143.9 (8.2)	141.7 (7.1)	145.3 (8.6)	**0.010**
Weight (kg)	39.2 (9.4)	37.2 (8.0)	40.6 (9.9)	ns
BMI[e](kg/m^2)	18.8 (3.2)	18.5 (2.9)	19.1 (3.3)	ns
zBMI (score)	0.1 (0.9)	0.1 (0.9)	0.1 (0.9)	ns
Body Fat (%)	20.7 (7.8)	18.0 (6.5)	22.2 (8.1)	**<0.001**
Waist circumference (cm)	66.0 (7.6)	66.1 (7.0)	66.0 (7.9)	ns
Adolescence[b]				
Height (m)	161.3 (7.7)	163.3 (9.0)	160.1 (6.5)	**0.007**
Weight (kg)	52.5 (10.1)	52.8 (10.4)	52.2 (9.9)	ns
BMI(kg/m^2)	20.1 (3.3)	19.8 (3.5)	20.3 (3.2)	ns
Body Fat (%)	20.9 (8.8)	14.1 (6.8)	24.7 (7.4)	**<0.001**
zBMI (score)	-0.07 (0.1)	-0.1 (0.8)	-0.01 (0.9)	ns
Waist circumference (cm)	72.4 (8.1)	73.7 (8.8)	71.5 (7.5)	**0.038**
Biceps skinfold (mm)	9.9 (5.2)	9.0 (5.1)	10.5 (5.2)	**0.030**
Tricep skinfold (mm)	16.3 (6.7)	13.6 (7.1)	17.9 (6.0)	**<0.001**
Subscapular skinfold (mm)	13.1 (6.2)	11.5 (6.2)	14.1 (6.0)	**0.002**
Change[c] from preadolescence to adolescence				
Change in BMI(Kg/m^2)	1.3 (1.7)	1.4 (1.7)	1.3 (1.8)	ns
Change in Body Fat(%)	0.3 (5.9)	-3.6 (4.5)	2.5 (5.5)	**0.007**
Change in waist circumference(cm)	6.4 (5.7)	7.8 (5.6)	5.6 (5.7)	**<0.001**

[a]Preadolescence: baseline phase; [b]Adolescence; follow-up phase. [c]Change: difference between the baseline point (preadolescence) and the endpoint (adolescence). SD: Standard Deviation. ns: non-significant BMI: Body Mass Index; zBMI: z-score of BMI. Level of statistical significance: p value< 0.05

Figure 10 depicts the percentage of overweight/obesity by gender in adolescence (follow-up phase). In our follow-up sample, 15.6 % showed overweight or were obese (a 16.5% were boys and a 15.1% were girls). There were no significant differences among gender (X^2=0.085, p=770). Moreover, the percentage of overweight/obesity in preadolescence of the follow-up sample was 27.7%, which did not differ from the baseline sample (p=0.519) (data not shown).

Figure 10. Percentage of normoweight and overweight/obesity at the follow-up phase



UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
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2. EFFECT OF EMOTIONAL SYMPTOMS AND DIETARY INTAKE AND PHYSICAL ACTIVITY

2.1 FOOD CONSUMPTION AND ENERGY AND NUTRIENT INTAKE

2.1.1 Description of food consumption and energy and nutrient intake

We obtained complete food consumption data of 165 participants. General characteristics of food frequency consumption in adolescents are shown in table 10. We can observe that boys consumed dairy products (p=0.025), breakfast cereal and biscuits (p=0.018) and soft-drink (p=0.003) more often than girls.

Table 11 shows energy and macronutrient intake by gender. There were no significant differences among gender in energy, carbohydrate and protein intake, except for saturated fatty acid intake (p=0.035), which were slightly higher in boys than girls.

The vitamin and mineral intake and their adequacy to recommendations are shown in table 12. We found that boys ingested higher amount of micronutrients such as calcium (p=0.003), phosphorous (p=0.016), thiamine (p=0.035) and riboflavin (p=0.003) than girls. In the total sample, the percentage of adequacy was below two thirds of the recommendation of calcium (59.9±17.4%), iron (57.7±18.1%), magnesium (60.9±16.1%), vitamin D (13.3±5.7%), and folic acid (61.1±21.0%) in both boys and girls. In addition, the percentage of adequacy of calcium (p=0.003), iron (p=0.001) and pantothenic acid (p=0.008) were lesser in girls than boys. In contrast percentage of adequacy of magnesium (p=0.006) and vitamin A (p=0.016) were higher in girls than boys.

Table 10. Food consumption according to gender

	Total (n= 165)		**Boys** (n= 59)		**Girls** (n= 106)		P value between boys and girls
	Mean	(SD)	Mean	(SD)	Mean	(SD)	
Dairy products (s/d)	2.3	(0.8)	2.5	(0.8)	2.2	(0.8)	**0.025**
Sweet dairy desserts (s/w)	3.1	(3.0)	3.3	(3.0)	3.0	(2.9)	ns
Breakfast cereals and biscuits (s/d)	0.7	(0.5)	0.8	(0.6)	0.6	(0.4)	**0.018**
Baked goods and chocolates (s/w)	5.8	(4.7)	6.5	(5.9)	5.4	(3.9)	ns
Sweets (s/w)	1.3	(1.6)	1.2	(1.8)	1.3	(1.5)	ns
Starchy food (s/d)	2.1	(0.8)	2.1	(0.7)	2.2	(0.9)	ns
Beans (s/w)	1.6	(0.9)	1.7	(1.1)	1.5	(0.8)	ns
Potatoes (s/w)	2.9	(2.0)	3.0	(2.2)	2.8	(1.9)	ns
Vegetables (s/d)	1.1	(0.6)	0.9	(0.5)	1.1	(0.7)	ns
Fruits (s/d)	1.7	(0.9)	1.7	(0.9)	1.7	(0.9)	ns
Nuts (s/w)	0.1	(0.2)	0.1	(0.2)	0.1	(0.2)	ns
Meat and cool meat (s/w)	11.4	(3.4)	11.5	(2.7)	11.4	(3.8)	ns
Fish and shellfish (s/w)	3.3	(1.7)	3.1	(1.8)	3.4	(1.6)	ns
Eggs (s/w)	2.2	(1.1)	2.2	(1.0)	2.2	(1.2)	ns
Pre-cooked meals (s/w)	1.4	(1.1)	1.5	(1.3)	1.3	(0.9)	ns
Savoury snacks (s/w)	1.3	(1.4)	1.5	(1.7)	1.1	(1.2)	ns
Soft-drink (s/w)	2.0	(2.7)	2.7	(3.2)	1.6	(2.3)	**0.033**

SD: Standard Deviation; ns: non-significant ; s/d: servings per day; s/w: servings per week
Level of statistical significance: p value <0.05

Table 11. Daily energy and macronutrient intakes according to gender

	Total (n=165)		**Boys** (n=59)		**Girls** (n=106)		P value between boys and girls
	Mean	(SD)	Mean	(SD)	Mean	(SD)	
Energy (kcal)	2073.6	(439.0)	2133.5	(479.2)	2040.6	(413.6)	ns
Carbohydrate (g)	223.3	(77.2)	232.0	(84.7)	218.5	(75.7)	ns
Protein (g)	67.5	(14.6)	69.2	(15.0)	66.5	(14.3)	ns
Fat (g)	101.2	(11.9)	103.2	(12.9)	100.0	(11.2)	ns
Saturated fatty acids (g)	29.0	(5.3)	30.2	(5.9)	28.4	(4.8)	**0.035**
Monounsaturated fatty acids (g)	54.3	(4.3)	55.0	(4.6)	53.9	(4.2)	ns
Polyunsaturated fatty acids (g)	10.4	(1.3)	10.5	(1.3)	10.4	(1.3)	ns
Cholesterol (mg)	279.9	(62.8)	289.5	(71.5)	274.5	(56.9)	ns
Percentage of total energy intake							
Carbohydrate (%)	42.0	(6.1)	42.43	(6.1)	41.8	(6.2	ns
Protein (%)	13.0	(1.4)	13.0	(1.4)	13.0	(1.5)	ns
Fat (%)	44.8	(5.1)	44.5	(5.1)	45.0	(5.2)	ns
Saturated fatty acids (%)	12.7	(1.3)	12.8	(1.4)	12.6	(1.3)	ns
Monounsaturated fatty acids (%)	24.2	(3.3)	23.8	(3.2)	24.4	(3.4)	ns
Polyunsaturated fatty acids (%)	4.6	(0.5)	4.5	(0.5)	4.7	(0.5)	ns

SD: Standard Deviation; ns: non-significant
Level of statistical significance: p-value <0.05

Results

Table 12. Daily vitamin and mineral intake and percentage of adequacy to Spanish Recommended Dietary Intake♦ according to gender

	Total (n=165)				**Boys** (n=59)				**Girls** (n=106)				p[a] Intake between boys and girls	p[b] Adequacy between boys and girls
	Intake		% Adequacy♦		Intake[a]		% Adequacy[b]♦		Intake[a]		% Adequacy[b]♦			
	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)		
Calcium (mg)	778.8	(226.7)	59.9	(17.4)	849.0	(249.7)	65.3	(19.2)	739.7	(203.7)	56.9	(15.6)	**0.003**	**0.003**
Iron (mg)	9.4	(2.5)	57.5	(18.1	9.8	(2.8)	68.3	(20.5)	9.2	(2.3)	51.5	(13.2)	ns	**<0.001**
Magnesium (mg)	211.6	(54.9)	60.9	(16.1)	219.5	(60.8)	56.3	(15.7)	207.3	(51.6)	63.5	(15.8)	ns	**0.006**
Potassium (mg)	2474.5	(675.8)	79.8	(21.8)	2581.9	(747.2)	83.2	(24.1)	2414.3	(628.2)	77.8	(20.2)	ns	ns
Phosphorus (mg)	997.3	(233.6)	83.1	(19.4)	1055.5	(266.3)	87.9	(22.1)	964.8	(207.5)	80.4	(17.2)	**0.016**	**0.016**
Sodium (mg)	1799.4	(462.8)	119.7	(30.8)	1844.8	(430.9)	122.9	(28.73)	174.2	(479.7)	117.8	(32.0)	ns	ns
Vitamin D (µg)	2.0	(0.8)	133	(5.7)	2.0	(0.9)	13.3	(6.0)	2.0	(0.8)	13.3	(5.5)	ns	ns
Vitamin E (mg)	9.6	(1.0)	89.2	(9.8)	9.7	(1.0)	90.0	(10.0)	9.6	(1.0)	88.8	(9.7)	ns	ns
Vitamin C (mg)	73.1	(33.0)	121.9	(55.0)	74.6	(32.7)	124.3	(54.6)	72.3	(33.2)	120.6	(55.4)	ns	ns
Vitamin A (µg)	575.1	(146.6)	66.4	(17.2)	604.1	(162.5)	60.4	(16.2)	558.9	(135.0)	69.8	(16.8)	**0.057**	**0.001**

Table 12 (continue). Daily vitamin and mineral intake and percentage of adequacy to Spanish Recommended Dietary Intake♦ according to gender.

	Total (n=165)				**Boys** (n=59)				**Girls** (n=106)				p[a] intake between boys and girls	p[b] adequacy between boys and girls
	Intake		% Adequacy♦		Intake[a]		% Adequacy[b]♦		Intake[a]		% Adequacy[b]♦			
	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)		
Thiamine (mg)	1.1	(0.3)	116.9	(30.0)	1.2	(0.3)	116.7	(31.9)	1.1	(0.2)	117.0	(29.0)	**0.035**	ns
Riboflavin (mg)	1.6	(0.4)	105.1	(28.1)	1.7	(0.5)	107.2	(31.4)	1.5	(0.3)	103.9	(26.2)	**0.003**	ns
Pantothenic acid (mg)	4.0	(0.9)	84.2	(20.5)	4.2	(1.0)	89.9	(22.7)	3.9	(0.8)	81.1	(18.5)	**0.017**	**0.008**
Niacin (mg)	15.6	(4.0)	91.8	(24.1)	16.0	(4.5)	90.9	(25.5)	15.4	(3.8)	92.3	(23.5)	ns	ns
Vitamin B_6 (mg)	1.5	(0.4)	77.3	(23.7)	1.6	(0.5)	80.9	(26.6)	1.5	(0.4)	75.3	(21.8)	ns	ns
Vitamin B_{12} (µg)	5.2	(1.2)	260.8	(64.8)	5.4	(1.4)	273.4	(70.6)	5.0	(1.2)	253.8	(60.6)	ns	ns
Folic Acid (µg)	233.4	(75.4)	61.1	(21.0)	243.4	(81.6)	64.5	(23.0)	227.8	(71.5)	59.2	(19.7)	ns	ns

♦Percentage of intake adequacy to Reference Dietary Intake of nutrients created for the Spanish population (Moreiras et al. 2013) and Dietary Reference Intakes of Institute of Medicine (2006). SD: Standard Deviation; ns: non-significantLevel of statistical significance: p value <0.05

2.1.2 Association between emotional symptoms and food consumption, energy and nutrient intake.

Table 13 shows food consumption according to the presence of emotional symptoms. We can observe that food consumption was not significantly different between boys with or without emotional symptoms. In contrast, girls with emotional symptoms consumed significantly more sweet dairy desserts (3.5±3.2 s/w) and sweets (1.6±1.8 s/w) and lower intakes of dairy products (2.0±0.7 s/d) than girls without emotional symptoms (sweet dairy desserts, 2.1±2.4 s/w; sweets 0.9±0.9 s/w; dairy products 2.4±0.9 s/d ($p<0.05$)). Although it is not significant, we observed a tendency towards a higher consumption of baked goods and chocolates, pre-cooked meals, savoury snacks and soft drinks and a lower consumption of vegetables, fruits, beans, fish and seafood among girls with emotional symptoms in comparison to control group.

Regarding energy and nutrient intake, there were no significant differences between the group with emotional symptoms and the control group neither in boys nor girls (table 14, 15, 16). We only found that in boys the intakes of calcium (except the control group), magnesium, vitamin D and vitamin A and folic acid were below two thirds of the recommendation (table 14). Girls with and without emotional symptoms reported a percentage of adequacy lesser than two thirds of recommendation in calcium, iron, magnesium, vitamin D and folic acid (table 15).

Table 13. Food consumption according to the presence of any emotional symptoms

	Boys					Girls				
	Control (n=22)		Emotional symptoms (n=37)			Control (n=43)		Emotional symptoms (n=63)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Dairy products (s/d)	2.6	(0.8)	2.4	(0.8)	ns	2.4	(0.9)	2.0	(0.7)	**0.010**
Sweet dairy desserts (s/w)	3.7	(2.6)	3.0	(3.3)	ns	2.1	(2.4)	3.5	(3.2)	**0.012**
Breakfast cereals and biscuits (s/d)	0.9	(0.6)	0.8	(0.6)	ns	0.7	(0.5)	0.5	(0.4)	ns
Baked goods and chocolates (s/w)	6.5	(6.4)	6.4	(5.7)	ns	5.3	(4.0)	5.5	(3.8)	ns
Sweets (s/w)	1.0	(0.5)	1.3	(2.0)	ns	0.9	(0.9)	1.6	(1.8)	**0.021**
Starchy food (s/d)	2.0	(0.5)	2.1	(0.7)	ns	2.1	(0.7)	2.2	(1.0)	ns
Beans (s/w)	1.9	(1.6)	1.5	(0.7)	ns	1.6	(0.9)	1.4	(0.8)	ns
Potatoes (s/w)	3.2	(2.2)	2.8	(2.1)	ns	2.7	(2.0)	3.0	(1.8)	ns
Vegetables (s/d)	1.1	(0.5)	0.9	(0.6)	ns	1.2	(0.6)	1.1	(0.7)	ns
Fruits (s/d)	1.7	(0.8)	1.7	(1.0)	ns	1.7	(0.9)	1.6	(0.9)	ns
Nuts (s/w)	0.2	(0.2)	0.1	(0.1)	ns	0.1	(0.2)	0.1	(0.1)	ns
Meat and cool meat (s/w)	11.2	(3.2)	11.6	(2.3)	ns	11.2	(4.0)	11.5	(3.7)	ns
Fish and shellfish (s/w)	3.1	(1.5)	3.2	(1.9)	ns	3.5	(1.4)	3.3	(1.8)	ns
Eggs (s/w)	2.1	(0.7)	2.3	(1.1)	ns	2.4	(1.4)	2.1	(0.9)	ns
Pre-cooked meals (s/w)	1.2	(0.7)	1.7	(1.5)	ns	1.2	(0.6)	1.3	(1.0)	ns
Savoury snacks (s/w)	1.1	(0.8)	1.7	(2.0)	ns	1.0	(1.0)	1.2	(1.3)	ns
Soft-drink (s/w)	3.1	(4.0)	2.4	(2.6)	ns	1.3	(1.6)	1.9	(2.6)	ns

SD: Standard Deviation; ns: non-significant; s/d: servings per day; s/w: servings per week
Level of statistical significance: p value<0.05

Table 14. Daily energy and macronutrient intakes according to the presence of any emotional symptoms

	Boys					Girls				
	Control (n=22)		Emotional symptoms (n=37)			Control (n=43)		Emotional symptoms (n=63)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Energy (kcal)	2106.1	(400.4)	2149.8	(525.1)	ns	2043.9	(380.0)	2037.8	(438.0)	ns
Carbohydrate (g)	222.8	(67.1)	237.5	(94.0)	ns	216.9	(67.6)	219.6	(76.5)	ns
Protein (g)	69.9	(13.4)	68.9	(16.0)	ns	67.1	(13.1)	66.1	(15.1)	ns
Fat (g)	103.8	(11.3)	102.9	(13.9)	ns	100.7	(10.7)	99.5	(11.6)	ns
Saturated fatty acids (g)	30.7	(5.4)	29.9	(6.3)	ns	28.6	(4.7)	28.2	(4.9)	ns
Monounsaturated fatty acids (g)	55.1	(4.1)	54.9	(4.9)	ns	54.2	(4.1)	53.7	(4.2)	ns
Polyunsaturated fatty acids (g)	10.5	(1.0)	10.5	(1.4)	ns	10.5	(1.2)	10.4	(1.3)	ns
Cholesterol (mg)	291.2	(66.1)	288.4	(75.4)	ns	280.7	(54.2)	270.3	(58.8)	ns
Percentage of total energy intake										
Carbohydrate (%)	41.5	(5.4)	42.9	(6.4)	ns	41.5	(6.3)	42.0	(6.2)	ns
Protein (%)	13.3	(1.3)	12.9	(1.4)	ns	13.1	(1.5)	13.0	(1.4)	ns
Fat (%)	45.1	(4.7)	44.1	(5.2)	ns	45.1	(5.3)	44.9	(5.1)	ns
Saturated fatty acids (%)	13.0	(1.2)	12.7	(1.5)	ns	12.6	(1.3)	12.7	(1.3)	ns
Monounsaturated fatty acids (%)	23.8	(3.1)	23.8	(3.4)	ns	24.2	(3.7)	24.5	(3.2)	ns
Polyunsaturated fatty acids (%)	4.5	(0.5)	4.5	(0.4)	ns	4.6	(0.6)	4.7	(0.5)	ns

SD: Standard Deviation; ns: non-significant.Level of statistical significance: p-value <0.05.

Table 15. Daily vitamin and mineral intakes and the percentage of adequacy to Spanish Reference Dietary Intake♦ according to the presence of any emotional symptoms in boys

	Control (n=22)				Emotional symptoms (n=37)					
	Intake[a]		% Adequacy[c♦]		Intake[b]		% Adequac[d♦]		p[ab]	P[cd]
	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)		
Calcium (mg)	902.7	(243.5)	69.4	(18.7)	817.0	(251.1)	62.8	(19.3)	ns	ns
Iron (mg)	9.6	(2.5)	66.1	(16.0)	9.9	(3.1)	69.7	(22.9)	ns	ns
Magnesium (mg)	219.9	(52.3)	55.8	(12.6)	219.2	(66.1)	56.6	(17.5)	ns	ns
Potassium (mg)	2580.0	(626.3)	83.2	(20.2)	2582.6	(819.0)	83.3	(26.4)	ns	ns
Phosphorus (mg)	1082.2	(255.4)	90.1	(21.2)	1039.7	(274.8)	86.6	(22.9)	ns	ns
Sodium (mg)	1797.7	(345.5)	119.8	(23.0)	1872.8	(476.9)	124.8	(31.7)	ns	ns
Vitamin D (µg)	1.9	(0.6)	13.1	(4.5)	2.0	(1.0)	13.4	(6.8)	ns	ns
Vitamin E (mg)	9.7	(0.8)	89.7	(7.1)	9.6	(1.2)	90.2	(11.5)	ns	ns
Vitamin C (mg)	76.1	(29.5)	126.9	(49.3)	73.7	(34.8)	122.8	(58.1)	ns	ns
Vitamin A (µg)	614.7	(152.0)	61.4	(15.2)	597.9	(17.0)	59.6	(17.5)	ns	ns
Thiamine (mg)	1.2	(0.3)	116.8	(27.4)	1.2	(0.3)	116.7	(34.6)	ns	ns
Riboflavin (mg)	1.8	(0.5)	109.9	(29.4)	1.7	(0.5)	105.6	(32.8)	ns	ns
Pantothenic acid (mg)	4.3	(0.9)	90.6	(19.0)	4.2	(1.1)	89.5	(24.9)	ns	ns
Niacin (mg)	15.8	(4.1)	89.4	(22.5)	16.0	(4.7)	91.8	(27.4)	ns	ns
Vitamin B_6 (mg)	1.6	(0.4)	79.0	(20.9)	1.6	(0.5)	82.0	(29.7)	ns	ns
Vitamin B_{12} (µg)	5.4	(1.4)	273.2	(70.8)	5.4	(1.4)	273.5	(71.4)	ns	ns
Folic acid (µg)	248.0	(69.9)	64.4	(17.5)	240.7	(88.7)	64.5	(26.0)	ns	ns

♦Percentage of intake adequacy to Reference Dietary Intake of nutrients created for the Spanish population (Moreiras et al. 2013) and Dietary Reference Intakes of Institute of Medicine (2006)
SD: Standard Deviation; ns: non-significant . Level of statistical significance: p-value <0.05

Table 16. Vitamin and mineral intake and the percentage of adequacy to Spanish Reference Dietary Intake♦ according to the presence of any emotional symptoms in girls

	Control (n=43)				Emotional symptoms (n=63)					
	Intake[a]		% Adequacy[b♦]		Intake[a]		% Adequacy[d♦]		p^{ab}	P^{cd}
	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)		
Calcium (mg)	765.1	(206.6)	58.8	(15.8)	956.7	(209.3)	55.5	(15.5)	ns	ns
Iron (mg)	9.3	(2.1)	52.1	(11.7)	3.1	(2.5)	51.1	(14.2)	ns	ns
Magnesium (mg)	207.4	(46.2)	63.5	(14.3)	207.2	(55.3)	63.5	(16.8)	ns	ns
Potassium (mg)	2409.1	(602.9)	77.7	(19.4)	2417.8	(649.8)	77.9	(20.9)	ns	ns
Phosphorus (mg)	976.7	(206.8)	81.3	(17.2)	956.7	(209.3)	79.7	(17.4)	ns	ns
Sodium (mg)	1745.9	(374.6)	116.3	(24.9)	1793.5	(542.0)	118.9	(36.7)	ns	ns
Vitamin D (µg)	2.04	(0.7)	13.6	(5.0)	1.9	(0.8)	13.1	(5.8)	ns	ns
Vitamin E (mg)	9.7	(0.8)	89.4	(9.0)	9.6	(1.0)	88.4	(10.1)	ns	ns
Vitamin C (mg)	69.5	(25.1)	115.8	(41.9)	74.3	(37.9)	123.8	(63.1)	ns	ns
Vitamin A (µg)	562.0	(103.6)	70.2	(12.9)	556.7	(153.7)	69.5	(19.2)	ns	ns
Thiamine (mg)	1.1	(0.2)	115.2	(25.4)	1.1	(0.3)	118.3	(31.4)	ns	ns
Riboflavin (mg)	1.5	(0.3)	104.5	(24.5)	1.5	(0.4)	103.5	(27.3)	ns	ns
Pantothenic acid (mg)	3.9	(0.8)	81.1	(17.5)	3.9	(0.8)	81.1	(19.4)	ns	ns
Niacin (mg)	15.4	(3.5)	92.3	(21.6)	15.4	(4.0)	92.3	(24.8)	ns	ns
Vitamin B_6 (mg)	1.5	(0.3)	74.6	(19.4)	1.5	(0.4)	75.8	(23.5)	ns	ns
Vitamin B_{12} (µg)	5.0	(1.0)	254.5	(53.6)	5.0	(1.3)	253.3	(65.3)	ns	ns
Folic acid (µg)	225.9	(53.7)	58.8	(16.5)	229.2	(81.8)	59.5	(21.8)	ns	ns

♦Percentage of intake adequacy to Reference Dietary Intake of nutrients created for the Spanish population (Moreiras et al. 2013) and Dietary Reference Intakes of Institute of Medicine (2006) SD: Standard Deviation; ns: non-significant. Level of statistical significance: p-value <0.05

2.2 DIETARY PATTERNS

2.2.1 Description of dietary patterns

Dietary patterns were determined *a posteriori* using the principal component analysis. First, the 45 items in the food frequency questionnaire were classified into 19 food groups (Table 2). We obtained a good measure of sampling adequacy of Kaiser-Meyer-Olkin (0.64) and an adequate Bartlett test of sphericity (X^2=455.8; p<0.001). According to the Kaiser criteria, we obtained seven factors with an eigenvalue greater than 1, whereas using the slope Cattell test or screenplot we reduced the number of factor to three (figure 11).

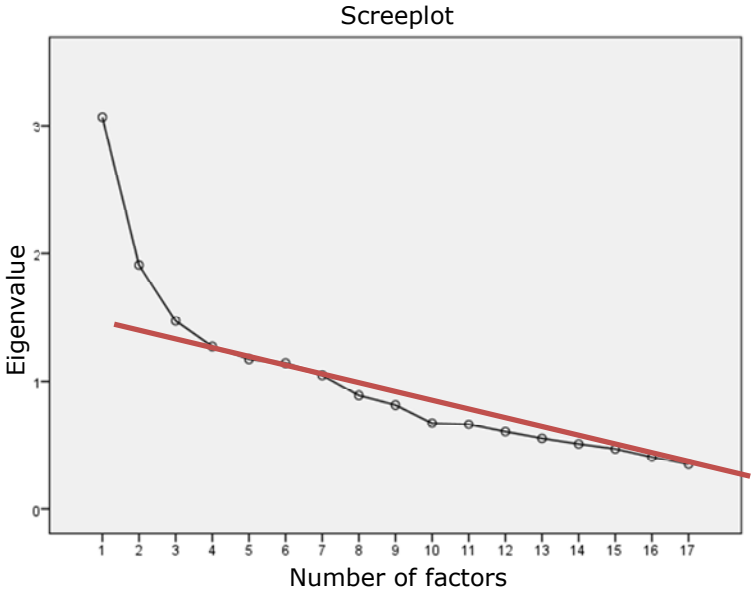

Figure 11. Factors extracted from Cattell test or screeplot

As a result, three independent factors or dietary patterns were identified, which explained 37.89% of the total variance. We used a factor loading matrix to extract the weights for each food group. The food groups with a factor loading of 0.30 or more were considered as important contributors to the dietary patterns. The food loading in each dietary pattern is shown in table 17. Observing these food loadings, we named the three major factors as dietary patterns. The first dietary pattern could be labelled "sweet and fatty food pattern". This pattern was characterised by a high consumption of sweets (0.751), soft drinks (0.675), sweet dairy products (0.627), baked goods and chocolates (0.623) and savoury snacks (0.577). The second dietary pattern was a typical "western pattern" because it was characterised by high consumption of meat and cold meat (0.332), starchy foods (0.790) and potatoes (0.724). The third dietary pattern was identified as a "healthy pattern" since it included fruits (0.543), beans (0.773), vegetables (0.507), fish and seafood (0.381).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Table 17. Factor loading matrix for the follow-up sample

	Factor loading		
	Factor 1: Sweet and Fatty Food Pattern	Factor 2: Western Pattern	Factor 3: Healthy Pattern
Sweets	**0.751**	0.161	-0.082
Soft drinks	**0.675**	-0.148	0.192
Sweet dairy products	**0.627**	-0.232	-0.108
Baked goods and chocolates	**0.623**	0.155	0.250
Savoury Snacks	**0.577**	**0.332**	0.086
Meat and cold meat	0.094	**0.790**	-0.196
Starchy	0.044	**0.724**	0.240
Potatoes	0.037	**0.344**	0.291
Fruits	0.152	0.399	**0.543**
Beans	0.253	-0.046	**0.773**
Vegetables	-0.185	0.069	**0.507**
Fish and seafood	-0.086	0.066	**0.381**
Dairy products	0.029	0.154	0.197
Eggs	0.064	-0.025	0.036
Breakfast cereals and biscuits	0.089	0.061	-0.109
Nuts	0.139	-0.010	-0.053
Pre-cooked meals	0.129	-0.039	-0.075
% variance	18.03	11. 23	8.63

Food items with a factor loading of ≥ 0.30 are highlighted in bold.

2.2.2 Association between emotional symptoms and dietary patterns

The association between the presence of emotional symptoms and the dietary patterns (scores and tertiles) are shown in table 18. Among girls, the score for the sweet and fatty food pattern was higher in those with emotional symptoms than in the control group (0.05±0.95 score vs -0.33±0.65 score, p=0.013). Indeed, 39.7% of girls with emotional symptoms had significantly a high adherence to the sweet and fatty pattern, in contrast to 18.6% of girls without symptoms (p=0.048). However, there were no significant differences in the western and healthy pattern. No significant differences were found among boys.

Multivariate analysis to predict the effect of emotional symptoms on sweet and fatty food pattern were conducted using the adjusted logistic regression (table 19). The data confirmed that the presence of emotional symptoms in girls increases the probability of greater adherence to the sweet and fatty food pattern fourfold (OR: 4.79, 95%CI (1.55-15.10, p=0.007)) than the control group. In boys, high physical activity was inversely related to adherence to the sweet and fatty dietary pattern (OR: 0.65, 95%CI (0.45-0.94), p=0.022).

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Dipòsit Legal: T 1593-2015

Table 18. Frequency of adherence to dietary patterns according to presence of emotional symptoms.

		Boys				Girls			
		Control (n=22)		**Emotional symptoms** (n=37)	p	**Control** (n=43)		**Emotional symptoms** (n=63)	p
Sweet and Fatty Food Pattern	score[#]	0.17	(1.19)	0.20 (1.23)	ns	-0.33	(0.65)	0.05 (0.95)	**0.013**
	Low adherence (%)	31.8		21.6		48.8		30.2	
	Medium adherence (%)	31.8		40.5	ns	32.6		30.2	**0.048**
	High adherence (%)	36.4		37.8		18.6		39.7	
Western Pattern	score[#]	-0.22	(0.94)	0.09 (0.86)	ns	-0.01	(1.09)	0.03 (1.03)	ns
	Low adherence (%)	31.8		32.4		34.9		34.9	
	Medium adherence (%)	31.8		35.1	ns	33.3		33.3	ns
	High adherence (%)	36.4		32.4		31.7		31.7	
Healthy Pattern	score[#]	0.20	(1.52)	-0.14 (0.87)	ns	0.14	(0.93)	-0.08 (0.87)	ns
	Low adherence (%)	31.8		35.1		27.9		36.5	
	Medium adherence (%)	27.3		37.8		34.9		31.7	ns
	High adherence (%)	40.9		27.0		37.2		31.7	

[#]Mean (Standard deviation). ns: non-significant Level of statistical significance: p value< 0.05

Table 19. Logistic regression models to predict the risk of a high sweet and fatty food pattern adherence according to presence of any emotional disorder

		OR	(95% CI)	p	
Boys					
Presence of emotional symptoms (0: no; 1: yes)		1.34	(0.32-5.60)	0.392	
Eating disorder symptoms (score)		0.95	(0.85-1.06)	0.683	
Age (years)		0.55	(0.23-1.34)	0.195	
Socioeconomic status					R^2*Nagelkerke* * 100 = 45.4
	Low	1	1		$\chi^2_{8.59}$= 23.56
	Medium	0.64	(0.09-4.63)	0.662	***p*=0.003**
	High	0.44	(0.49-4.02)	0.470	
BMIz (score)		0.77	(0.25-2.37)	0.661	
Energy intake (kcal)		1.00	(1.00-1.00)	**0.005**	
Physical activity (score)		0.65	(0.45-0.94)	**0.022**	
Girls					
Presence of emotional symptoms (0: no; 1: yes)		4.79	(1.55-15.10)	**0.007**	
Eating disorder symptoms (score)		0.99	(0.95-1.038)	0.767	
Age (years)		2.31	(1.26-4.242)	**0.007**	
Socioeconomic status					R^2*Nagelkerke**100 = 31.1
	Low	1	1		$\chi^2_{8.106}$ = 25.887
	Medium	0.31	(0.09-1.04)	0.059	***p*=0.001**
	High	0.16	(0.03-0.78)	**0.024**	
BMIz (score)		0.89	(0.49-1.63)	0.727	
Energy intake (kcal)		1.00	(1.00-1.00)	**0.011**	
Physical activity (score)		1.14	(0.88-1.48)	0.300	

zBMI: z-score of BMI;OR: Odds Ratio; 95%CI: 95% Confidence Interval.
Logistic regression models adjusted for age, socioeconomic status, zBMI, total energy intake and physical activity. Level of statistical significance: p value<0.05.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
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2.3 MEDITERRANEAN DIET ADHERENCE AND PHYSICAL ACTIVITY

2.3.1 General characteristics of Mediterranean diet adherence and physical activity

In the follow-up sample, 48.1% of subjects presented low Mediterranean Diet adherence, a 41.9% mid Mediterranean Diet adherence and only 10.0% presented high Mediterranean diet adherence. However, there were no differences between genders (p=0.524).

In relation to the physical activity test, a 36.4% of subjects showed low level of physical activity, a 49.4% mid-level of physical activity and a 14.2% high level of physical activity. The level of physical activity did not differ among genders (p=0.328). However, our results showed slight differences among genders in the test's score. Boys obtained more scores than girls in the total physical activity test (boys: 6.3±2.1; girls: 5.2±2.1; p<0.001) and also in television and games factor (boys: 3.1±1.0; girls: 2.9±1.1; p<0.001).

2.3.2 Association between emotional symptoms and Mediterranean diet adherence and physical activity

Mediterranean diet adherence and physical activity according to the presence of emotional symptoms are shown in table 20. We found that scores of Mediterranean diet adherence (p=0.019) and physical activity test (p=0.001) were lower in girls with emotional symptoms than control group. Indeed, 59.6% of girls with emotional symptoms showed a low adherence to Mediterranean diet in comparison to 34.6% of control groups. Furthermore, 45.1% of girls with emotional symptoms performed low physical activity in respect to 22.6% girls in the control group. By contrast, we did not observe significant differences in boys.

Table 20. Mediterranean diet adherence and physical activity according to the presence of any emotional symptoms

	Boys					Girls				
	Control (n=30)		**Emotional symptoms** (n=60)			**Control** (n=52)		**Emotional symptoms** (n=94)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Mediterranean diet Adherence[‡] (score)	5.6	(2.2)	5.7	(2.2)	ns	6.0	(1.8)	5.2	(2.3)	**0.019**
Low (%)	45.2		43.3			34.6		59.6		
Mid (%)	45.2		46.7		ns	55.8		31.9		**0.012**
High (%)	9.7		10			9.6		8.5		
Physical activity (score)	6.4	(2.0)	6.2	(2.2)	ns	5.9	(2.1)	4.8	(2.0)	**0.001**
Physical activity factor (score)	3.4	(1.8)	3	(1.9)	ns	2.7	(1.8)	1.9	(1.7)	**0.011**
Television and games factor (score)	2.9	(1.0)	3.3	(1.0)	ns	3.1	(0.9)	2.8	(1.2)	ns
Physical activity (level)										
Low (%)	30		36.7			22.6		45.1		
Mid (%)	60		53.3		ns	49.1		45.1		**0.003**
High (%)	10		10			28.3		9.9		

[‡]Expressed as mean (standard deviation), except where % shown. ns: non-significant
Level of statistical significance: p value<0.05

2.3.3 Psychological factors influence on Mediterranean diet adherence

In relation to psychopathological factors in adolescence (follow-up phase), table 21 shows that girls with low Mediterranean diet adherence presented significantly higher scores for depressive and eating disorders symptoms than girls with mid or high Mediterranean Diet adherence. However, although anxiety symptoms scores (SCARED score, p= 0.017; YI-4, p=0.036) showed significant differences between level of Mediterranean diet adherence, the post-hoc analyses did not show statistically significant results for anxiety symptoms. No significant differences were observed in boys.

Table 21. Psychopathological factors in adolescence according to Mediterranean diet adherence (low, mid and high)

	Boys				**Girls**			
	Low[a] (n=41)	**Mid**[b] (n=43)	**High**[c] (n=10)		**Low**[a] (n=75)	**Mid**[b] (n=58)	**High**[c] (n=14)	
	Mean (SD)	Mean (SD)	Mean (SD)	*p*	Mean (SD)	Mean (SD)	Mean (SD)	*p*
Anxiety symptoms (SCARED score)	17.4 (11.2)	11.2 (9.5)	20.4 (10.6)	ns	23.6 (11.7)	19.5 (8.2)	16.91 (6.2)	ns
Anxiety symptoms (YI-4 score)	9.2 (6.2)	10.9 (5.6)	7.9 (5.0)	ns	11.8 (6.4)	9.6 (4.8)	8.7 (3.7)	ns
Depressive symptoms (YI-4 score)	14.5 (9.3)	14.0 (7.2)	12.6 (7.6)	ns	17.7 (9.6)	12.3 (7.2)	13.0 (5.8)	**0.001**[ab]
Eating disorders symptoms (EDI score)	12.6 (9.8)	13.1 (9.4)	11.0 (6.4)	ns	18.9 (13.5)	14.3 (10.7)	8.9 (9.0)	**0.020**[ac]

SCARED: Screen for Child Anxiety Related Emotional Disorders; YI-4: Youth's Inventory-4; EDI-2: Eating Disorder Inventory-2. SD: Standard Deviation; ns: non-significant.
Level of statistical significance: p value<0.05

Table 22 reports the association between psychological factors and the risk of low Mediterranean diet adherence. In specific terms, we performed two logistic regression multivariate models. Model 1 included psychological variables of preadolescence (baseline phase) and was adjusted for age, gender, SES, birthplace, family type, school type, BMI, and activity risk/control variable. Model 2 included psychological variables of the adolescence (follow-up phase) and was adjusted for age, gender, SES, birthplace, family type, school type, BMI, body fat, physical activity factor and risk/control variable. For model 1, the results showed that anxiety or depressive symptoms did not influence on low Mediterranean diet adherence, whereas, a high SES was a protective factor for presenting a low Mediterranean diet adherence level (OR=0.80, 95%CI (0.69-0.93), p=0.003). Meanwhile, the results of model 2 showed that a high SES was also a protective factor (OR=0.77, 95%CI (0.66-0.89), p=0.001) and depressive symptoms were related to the risk of presenting low Mediterranean diet levels (OR=1.06, 95%CI (1.01-1.13), p=0.021).

Table 22. Effect psychopathological factors in the preadolescence and adolescence on Mediterranean diet adherence in adolescence

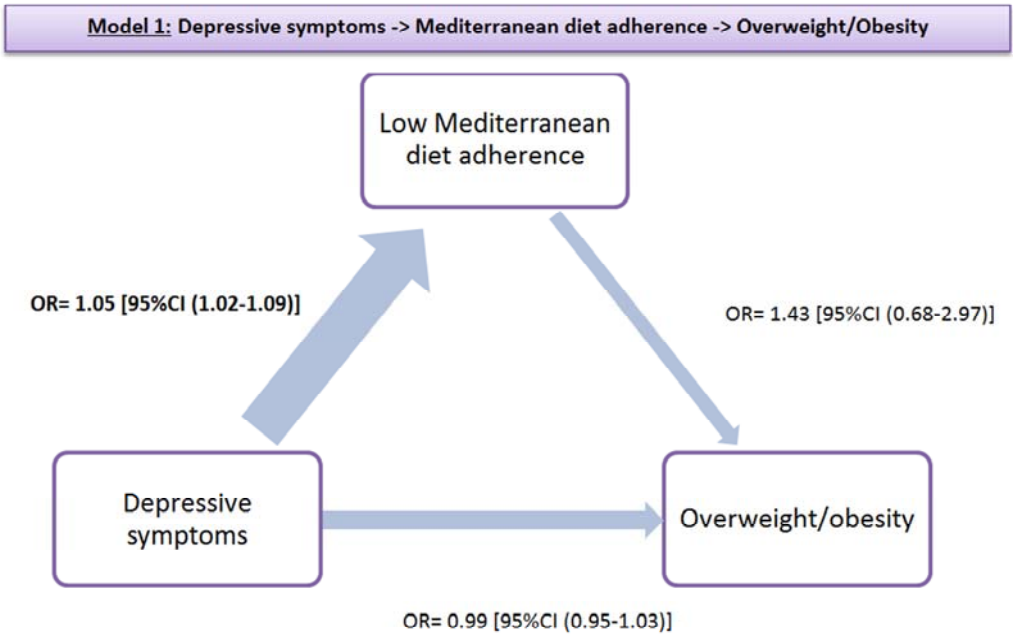
	Risk of low Mediterranean diet adherence in adolescence		
	ODDS RATIO (95% CI)	*p*	
Model 1: Preadolescence			
Anxiety symptoms[a] (score)	1.01 (0.97 – 1.04)	0.741	
Depressive symptoms[b] (score)	0.97 (0.93 – 1.02)	0.268	
Gender (0:boy; 1:girl)	0.80 (0.45 - 1.42)	0.452	
Age (years)	0.91 (0.66 - 1.25)	0.549	
Birthplace (0:foreign; 1:native)	2.14 (0.80 – 5.72)	0.130	***R^2Nagelkerke** * **100 = 10.8**
Family type (0:single parent; 1: nuclear)	1.41 (0.66 – 3.05)	0.377	**$\chi^2_{10.228}$ = 19.148**
School type (1: state school; 2: state-subsidized private school)	0.89 (0.49 – 1.61)	0.698	***p* = 0.038**
SES[c] (score)	0.80 (0.69 - 0.93)	**0.003**	
Risk/Control (0: risk; 1: control)	1.71 (0.76 – 3.85)	0.197	
BMI[d] (kg/m^2)	0.97 (0.89 – 1.06)	0.500	
Model 2: Adolescence			
Anxiety symptoms[e] (score)	0.93 (0.87 – 1.01)	0.072	
Depressive symptoms[e] (score)	1.07 (1.01 – 1.13)	**0.021**	
Eating disorders symptoms (score)[f]	1.02 (0.99 – 1.05)	0.291	***R^2Nagelkerke** * **100 = 17.3**
Conduct disorder symptoms[e] (score)	1.03 (0.95 – 1.13)	0.442	**$\chi^2_{12.231}$ = 32.072**
ADHD[g] symptoms[e]	0.99 (0.97 – 1.03)	0.921	***p* = 0.001**
Gender (0:boy; 1:girl)	0.50 (0.20-1.24)	0.132	
Age (years)	0.88 (0.65 – 1.20)	0.426	
SES (score)[c]	0.77 (0.66 – 0.89)	**0.001**	
Body fat (%)	0.97 (0.90 – 1.04)	0.411	
BMI (kg/m^2)	1.00 (0.85 – 1.18)	0.961	
Physical activity factor (score)	1.05 (0.92 – 1.21)	0.454	
Risk/Control (0: risk; 1: control)	1.54 (0.81 - 2.90)	0.187	

[a]SCARED: Screen for Child Anxiety Related Emotional Disorders. [b]CDI: Children's Depression Inventory. [c]SES: socioeconomic status. [d]BMI: body mass index. [e]YI-4:Youth's Inventory-4, [f]EDI-2: Eating Disorder Inventory-2. [g]ADHD: attention deficit hyperactivity disorder.

2.3.4 Mediational analyses between SES, depressive symptoms, Mediterranean diet and overweight/ obesity

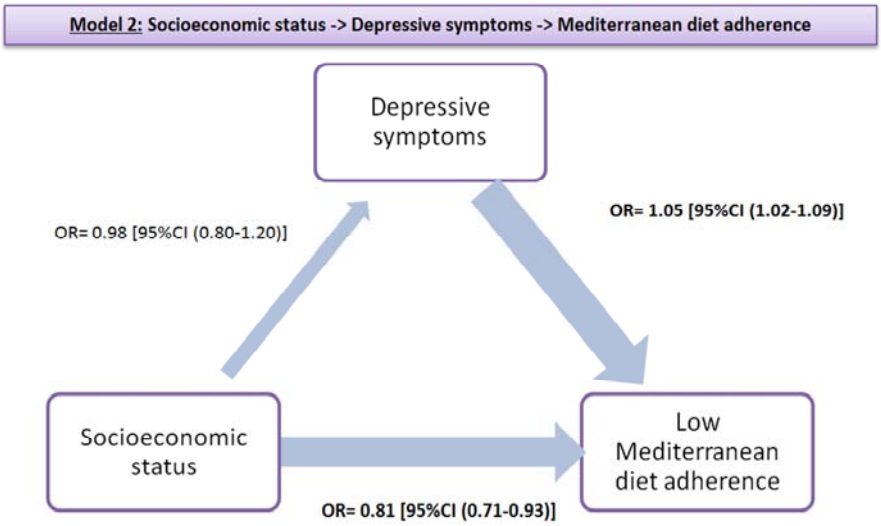
On the basis of the possible relationship between emotional symptoms and low SES with a high BMI, and in order to explore whether low Mediterranean diet adherence may be a mediator between these variables and overweight/obesity, we performed a mediational model adjusted by gender, age, and SES (figure 12). The results revealed that the risk of low Mediterranean diet adherence was not a mediator between depressive symptoms and overweight/obesity, or between SES and overweight/obesity. In model 1, we introduced the independent variable (depressive symptoms) and the mediator variable (Mediterranean diet adherence) to predict the risk of overweight and obesity. Depressive symptoms (OR=0.99, 95%CI (0.95-1.04), p=0.950) or Mediterranean Diet adherence (OR=1.42, 95%CI (0.67-3.02)) were not significant associated with low Mediterranean diet adherence. Only the high SES (adjusted variable) protected from risk of overweigh and obesity (OR=0.70, 95%CI (0.57-0.8), p=0.02). Furthermore, we tested another mediational model that positioned depressive symptoms as a partial mediator of the association between SES and risk of low Mediterranean diet adherence (model 2). According to the results of model 2, depressive symptoms were not a mediator variable since although depressive symptoms were related to Mediterranean diet adherence, SES were not associated with higher depressive symptoms. In the full model in which we included SES and depressive symptoms, both variables predicted the risk of low Mediterranean diet adherence (depressive symptoms (OR=1.05, 95%CI (1.02-1.09, p=0.001)); SES (OR=0.81, 95%CI (0.71-0.92, p=0.002)) (data not shown in the table). Therefore, in model 3 we tested another mediational model that positioned low Mediterranean diet adherence as a partial mediator of the association between SES and risk of overweight and obesity. The results revealed that lower SES was a predictor for the risk of low Mediterranean diet adherence and for overweight/obesity, whereas Mediterranean diet adherence did not predict risk of overweight/obesity.

Figure 12. Mediational analyses between socioeconomic status, depressive symptoms, Mediterranean diet and overweight/ obesity

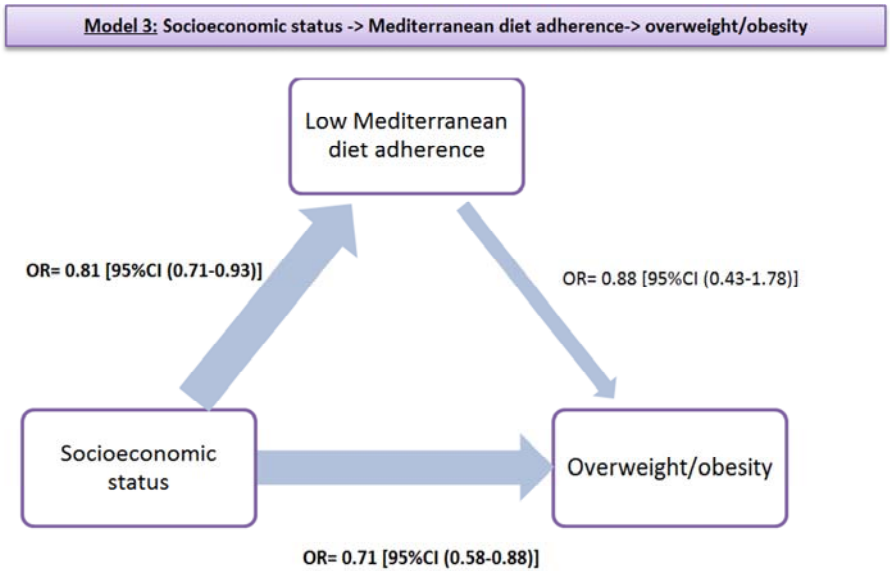


*Values expressed by OR (95% CI). Significant p values are in bold.
Model adjusted by age, gender and socioeconomic status.

Results



*Values expressed by OR (95% IC). Significant p values are in bold.
Model adjusted by age and gender.



*Values expressed by OR (95% CI). Significant p values are in bold.
Model adjusted by age and gender.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

3. EFFECT OFEMOTIONAL SYMPTOMS ON ANTHROPOMETRY AND BODY COMPOSITION

3.1 CROSS-SECTIONAL ASSOCIATION BETWEEN EMOTIONAL SYMPTOMS AND ANTHROPOMETRIC AND BODY COMPOSITION PARAMETERS IN THE BASELINE SAMPLE

The participants were classified as overweight/obesity or normoweight according to BMI and %BF.

The association of depressive and anxiety symptoms with weigh status (according to BMI values) is shown in table 23. There were no differences in frequency of overweight/obesity and presence or not of depressive symptoms (boys: X^2=1.775, p=0.446; girls: X^2=0.307, p=0.571) either presence or not of anxiety symptoms (boys: X^2=0.446, p=0.504; girls: X^2=1.461, p=0.227). Overweight/obese subjects also did not show differences in depressive and anxiety scores with respect to normal weight, either in girls or boys. The similar results were obtained whether participants were classified like normoweight or overweight/obese according to the %BF. We only found that overweight/obese girls showed higher scores of social phobia than girls with normal weight status (p=0.024) (table 24).There were no differences in frequency of overweight/obesity (according to the %BF) and presence or not depressive symptoms (boys: X^2=0.038, p=0.846; girls: X^2=0.402, p=0.526) either with presence or not of anxiety symptoms (boys: X^2=0.344, p=0.508; girls: X^2=1.997, p=0.158).

Table 23. Association between emotional symptoms and weight status (according to BMI values)

	Boys					Girls				
	Normoweight (n=440)		**Overweight/ obesity** (n=215)			**Normoweight** (n=520)		**Overweight /obesity** (n=223)		
	Mean‡	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Depressive symptoms (%)	10.0		13.4		ns	10.7		9.8		ns
CDI (score)	8.9	(5.9)	9.0	(6.3)	ns	8.8	(6.3)	9.3	(5.7)	ns
Anxiety symptoms (%)	42.3		39.5		ns	49.3		54.3		ns
SCARED (score)	23.5	(10.3)	22.6	(11.0)	ns	25.0	(10.1)	25.8	(9.2)	ns
Somatic panic (score)	3.8	(3.5)	3.6	(3.3)	ns	4.0	(3.6)	4.0	(3.2)	ns
Social phobia (score)	5.6	(2.8)	5.3	(3.0)	ns	6.2	(2.7)	6.4	(2.9)	ns
Generalized anxiety (score)	6.0	(3.0)	5.9	(3.0)	ns	6.3	(3.0)	6.4	(2.9)	ns
Separation anxiety (score)	7.6	(4.3)	8.0	(4.1)	ns	8.4	(4.0)	8.8	(3.7)	ns

‡ Expressed as mean (standard deviation), or percentage (where % shown). CDI: Children's Depression Inventory; SCARED: Screen for childhood Anxiety and Related Emotional Disorders; ns: non-significant. Level of statistical significance: p value<0.05

Table 24. Association between emotional symptoms and weight status (according to percentage of body fat)

	Boys					Girls				
	Normoweight (n=480)		**Overweight/ obesity** (n=167)			**Normoweight** (n=500)		**Overweight/ obesity** (n=204)		
	Mean‡	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Depressive symptoms (%)	10.8		11.4		ns	10.4		8.8		ns
CDI (score)	8.9	(5.9)	8.6	(6.0)		8.7	(6.1)	9.3	(5.8)	ns
Anxiety symptoms (%)	41.8		39.2		ns	48.8		54.7		ns
SCARED (score)	23.4	(10.3)	22.3	(10.9)	ns	24.9	(10.0)	25.9	(9.5)	ns
Somatic panic (Score)	3.8	(3.4)	3.5	(3.4)	ns	4.0	(3.6)	3.9	(3.1)	ns
Social phobia (score)	5.6	(2.9)	5.3	(2.9)	ns	6.1	(2.7)	6.7	(2.9)	**0.014**
Generalized anxiety (score)	6.0	(2.9)	5.7	(3.1)	ns	6.2	(3.0)	6.4	(2.9)	ns
Separation anxiety (Score)	7.9	(4.2)	7.7	(4.3)	ns	8.4	(3.9)	88	(3.8)	ns

‡ Expressed as mean (standard deviation), or percentage (where % shown)
CDI: Children's Depression Inventory; SCARED: Screen for childhood Anxiety and Related Emotional Disorders; ns: non-significant Level of statistical significance: p value <0.05

Among factors included in the depression test, it is important to highlight the low self-esteem factor. We observed that overweight/obese subjects showed higher scores of low self-esteem (girls: 1.7±1.4 scores; boys: 1.6±1.5 score) than normoweight group (girls: 1.4±1.3 scores; boys: 1.3± 1.4 score) in both genders (girls: p=0.015; boys: p=0.044) (Figure 13). Figure 14 depicts the association between body satisfaction and weight status (according to BMI). Body satisfaction was also lower in overweight/obese children than normal weight in boys (overweight/obesity: 30.7±5.6 score; normoweight: 32.2±5.1 score, p<0.001) and in girls (overweight/obesity: 28.8±5.5 score; normoweight: 31.7±5.5 score; p<0.001).

Figure 13. Association between low self-esteem and overweight/obesity

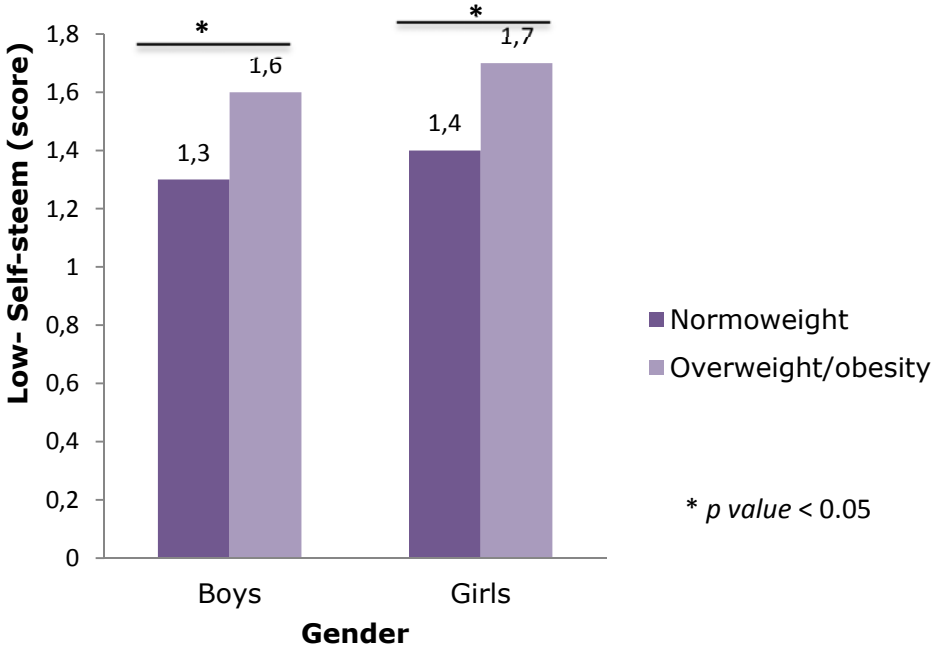
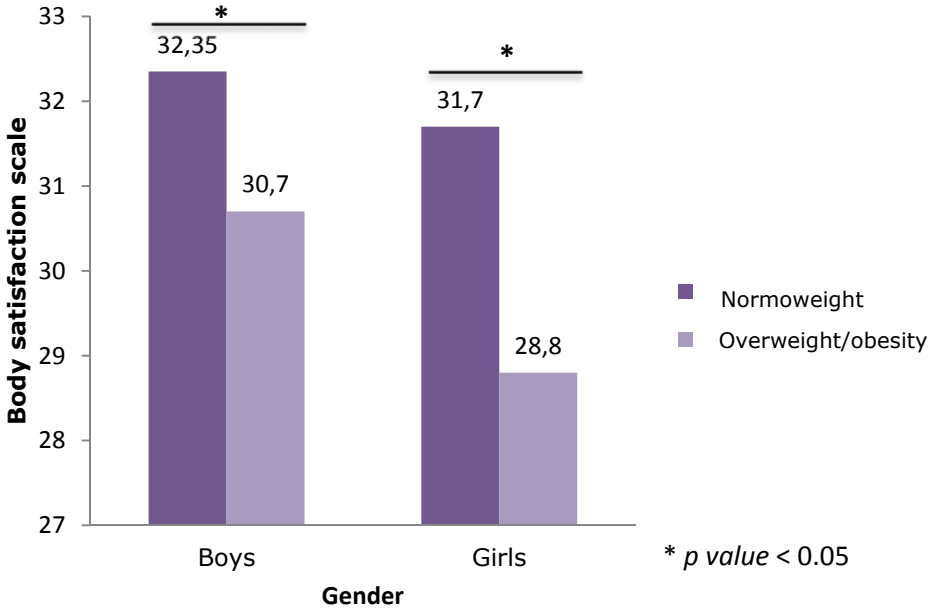


Figure 14. Association between body satisfaction and overweight/obesity



3.2 ASSOCIATION BETWEEN EMOTIONAL SYMPTOMS AND ANTHROPOMETRIC AND BODY COMPOSITION PARAMETERS IN THE FOLLOW-UP SAMPLE

We tested the association between the anthropometric and body composition parameters and the presence of any emotional symptoms during adolescence (table 25). There were no significant differences in anthropometric and body composition parameters between groups with emotional symptoms and control either in boys or in girls. In addition, there were no significant differences in the percentage of overweight/obesity between group with or without emotional symptoms. When we split the analysis by gender, we did not find differences either (data not shown).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Despite of this, based on the possible relationship between some emotional symptoms and anthropometry and body composition parameters, depressive symptoms and several types of anxiety symptoms were analyzed separately. Table 26 shows the correlation between the scores for anxiety and depression symptoms and the change in adiposity over the period of the study. A slight or moderate correlation was observed between separation anxiety and increased BMI ($r=0.220$) and %BF ($r=0.175$) in girls and between separation anxiety and increased WC in both gender (boys, $r=0.274$; girls $r=0.196$). Somatic symptoms were also found to be slightly or moderately associated with changes in WC ($r=0.269$), BMI ($r=0.187$) and %BF($r=0.210$) in girls. Scores for depressive symptoms were correlated with change in %BF in girls ($r=2.14$). In addition, the presence of depressive symptoms in preadolescence was associated with significant increases in BMI in boys ($p=0.040$) but not in girls ($p=0.150$) and with increases in %BF in both genders ($p<0.05$), compared to adolescents without these symptoms (measured by the t-test). Although the relationship between depressive symptoms and change in WC was not significant in either boys or girls, those adolescents who presented depressive symptoms showed a greater increase in WC than adolescents without depressive symptoms.

Table 25. Association between emotional symptoms and anthropometric and body composition characteristics

	Boys					Girls				
	Control (n= 29)		Emotional symptoms (n= 56)			Control (n= 50)		Emotional symptoms (n= 89)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Preadolescence[a]										
Weight (kg)	38.5	(9.2)	35.8	(6.4)	ns	40.8	(10.1)	40.1	(10.0)	ns
Height (cm)	142.9	(8.0)	141.1	(6.6)	ns	144.8	(8.4)	145.4	(8.7)	ns
BMI ((kg/m^2)	18.6	(2.8)	17.9	(2.5)	ns	19.4	(3.7)	18.7	(3.2)	ns
zBMI (score)	0.1	(0.7)	-0.03	(0.8)	ns	0.2	(1.0)	0.06	(0.8)	ns
Waist Circumference (cm)	66.8	8.0	65.8	6.6	ns	66.4	(8.7)	65.7	(7.6)	ns
Body fat (%)	20.5	(5.7)	18.8	(5.6)	ns	24.9	(9.0)	23.6	(8.0)	ns
Adolescence[b]										
Weight (kg)	53.5	(9.8)	51.2	(9.0)	ns	52.7	(10.4)	52.3	(9.9)	ns
Height (cm)	164.3	(9.3)	162.8	(8.8)	ns	160.0	(6.4)	160.2	(6.7)	ns
BMI (kg/m^2)	19.7	(2.9)	19.2	(2.9)	ns	20.5	(3.8)	20.2	(3.03)	ns
zBMI (score)	-0.04	(0.7)	-0.2	(0.8)	ns	0.04	(1.1)	-0.05	(0.8)	ns
Waist Circumference (cm)	73.7	(8.5)	72.3	(6.6)	ns	71.3	(8.0)	71.8	(7.8)	ns
Biceps skinfold (mm)	9.5	(5.4)	8.7	(5.0)	ns	11.3	(5.8)	10.0	(4.8)	ns
Triceps skinfold (mm)	142	(7.3)	13.2	(6.9)	ns	18.4	(6.4)	17.7	(5.8)	ns
Subscapular skinfold (mm)	12.1	(6.5)	11.2	(6.1)	ns	14.7	(6.7)	13.8	(5.6)	ns
Body fat (%)	14.3	(6.1)	13.0	(6.0)	ns	24.8	(7.9)	24.7	(7.1)	ns
Change[a] from preadolescence to adolescence										
Change in BMI (kg/m^2)	1.1	(1.2)	1.2	(1.7)	ns	1.1	(1.8)	1.4	(1.8)	ns
Change in Waist Circumference (cm)	6.9	(4.3)	7.4	(5.3)	ns	4.9	(5.1)	5.9	(6.1)	ns
Change in Body Fat (%)	-6.1	(4.4)	-5.4	(4.7)	ns	-0.3	(4.9)	1.2	(5.7)	ns

[a]Preadolescence: baseline phase; [b]Adolescence; follow-up phase. [c]Change: difference between the baseline point (preadolescence) and the endpoint (adolescence).
SD: Standard Deviation. ns: non-significant BMI: Body Mass Index; zBMI: z-score of BMI.
Level of statistical significance: p value<0.05

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Table 26. Association between emotional symptoms in preadolescence and changes in anthropometric and body composition parameters in adolescence

	CHANGE[a] FROM PREADOLESCENCE TO ADOLESCENCE								
	Total			**Boys**			**Girls**		
	CHANGE IN WC	CHANGE IN BMI	CHANGE IN %BF	CHANGE IN WC	CHANGE IN BMI	CHANGE IN %BF	CHANGE IN WC	CHANGE IN BMI	CHANGE IN %BF
Total SCARED[e](score)									
♦ *r*	0.144	0.086	0.061	0.108	-0.012	0.030	0.174	0.163	0.125
p	**0.037**	0.194	0.373	0.348	0.912	0.795	**0.045**	0.052	0.150
Somatic panic[e](score)									
r	-0.171	0.081	0.055	0.008	-0.044	-0.047	0.269	0.187	0.210
p	**0.013**	0.221	0.427	0.944	-0.47	0.679	**0.002**	0.026	**0.015**
Social phobia[e](score)									
r	-0.006	-0.031	0.055	<0.001	-0.110	0.004	0.039	0.024	-0.048
p	0.933	0.640	0.427	0.996	0.310	0.974	0.653	0.773	0.581
Generalized anxiety[e](score)									
r	-0.025	-0.29	0.042	0.002	-0.097	0.030	-0.023	0.018	0.002
p	0.717	0.665	0.538	0.986	0.310	0.793	0.796	0.834	0.979
Separation anxiety[e](score)									
r	0.231	0.196	0.101	0.274	0.131	0.115	0.196	0.220	0.175
p	**0.001**	**0.003**	0.141	**0.015**	0.163	0.313	**0.023**	**0.009**	**0.043**
Total CDI [f](score)									
r	0.130	0.138	0.117	0.176	0.133	0.109	0.085	0.138	0.214
p	0.059	**0.037**	0.089	0.124	0.218	0.339	0.332	0.101	**0.013**
Depressive symptomatology[g]									
<17	5.9(5.4)◊	1.2(1.7)	-0.2(5.7)	7.1(4.6)	1.1(1.6)	-4.1(4.0)	5.3(5.7)	1.1(1.8)	2.0(5.3)
≥17	8.1(6.7)	1.9 (1.8)	2.4(6.4)	10.4(7.9)	2.2(2.1)	-1.6(5.6)	6.6(5.6)	1.7(1.7)	5.0(5.6)
p	**0.030**	**0.017**	**0.010**	0.116	**0.040**	**0.044**	0.306	0.150	**0.015**

WC: waist circumference. BMI: body mass index. %BF: body fat percentage. SCARED: Screen for Childhood Anxiety and Related Emotional Disorders. CDI: Children's Depression Inventory.

[a]Change: difference between the endpoint (adolescence) and the baseline point (preadolescence).[b]Depressive symptoms score≥17.

♦*r:* Pearson coefficient ◊Mean (standard deviation). Level of statistical significance: p value<0.05.

3.3 PSYCHOPATHOLOGICAL PREDICTORS OF ADIPOSITY

Tables 27 and 28 show the multiple linear regression models adjusted for the various lifestyle variables, baseline anthropometry and body composition, body satisfaction and age for boys and girls, respectively.

For boys (Table 27), model 1 shows that the presence of depressive symptoms significantly accounts for the increase in WC, BMI and %BF. Model 3, which was adjusted for the anxiety symptoms, confirms the results for the increase in WC (b=3.50, p=0.029), BMI (b=1.25, p=0.022) and %BF (b=3.32, p=0.024). Model 4, which was adjusted for the diagnostic category variables, shows that diagnosis of dysthymia was a highly significant predictor of increased WC (b=9.25, p=0.001) and BMI (b=3.50, p<0.001). However, diagnosis of major depressive episode was found to be inversely related to BMI (b=-2.98, p=0.020). With regard to anxiety in boys, we can also observe, in model 3, that the symptoms of separation anxiety were associated with increased WC (b=0.43, p=.006) and BMI (b=0.10, p=0.041). Of the anxiety disorders (model 4), social phobia was associated with increased WC (b=9.59, p=0.006) and BMI (b=2.90, p=0.019), and panic disorder was related to increased BMI (b=2.83, p=0.043). In addition, lifestyle variables were found to be significant predictors of WC or BMI in all models (p<0.05).

For girls (Table 28), no significant relationship was observed between depressive symptoms and WC, BMI or %BF (models 1 and 3). However, model 4 shows that a clinical diagnosis of dysthymia significantly influenced the increase in WC (b=7.86, p=0.017). With regard to anxiety in girls, model 2 shows that anxiety symptoms led to the increase in WC, BMI and %BF. More specifically, model 3 shows that somatic/panic symptoms contributed to the increase in WC (b=0.34, p=0.035) and %BF (b=0.30, p=0.045). However, we found no relationship between diagnosis of anxiety disorders and changes in anthropometric and body composition parameters. Anthropometric and body composition variables in preadolescence have a highly significant effect on the same parameters in adolescence (p<0.001).

Table 27. Effect of emotional psychopathology in preadolescence on anthropometric and body composition parameters in adolescence in boys

	CHANGE[d] IN WC[e]				CHANGE[d] IN BMI[f]				CHANGE[d] IN %BF			
	B[h]	SE[i]	p	Model	B	SE	p	Model	B	SE	p	Model
MODEL 1: without adjusting for anxiety				$R^2_{c.100}$				$R^2_{C.100}$				$R^2_{C.100}$
Depressive symptoms[a] (0: no, 1: yes)[#]	4.1	1.50	**0.007**	20.9	1.27	0.50	**0.013**	11.7	3.44	1.27	**0.009**	16.6
Baseline variable: WC, BMI or %BF	-0.03	0.83	0.640	F^6_{78} 4.174	0.04	0.06	0.518	F^6_{87} 2.766	-0.23	0.08	**0.007**	F^6_{79} 3.430
Age (years)	-1.39	0.63	0.033	p	-0.23	0.21	0.279	p	-0.68	0.57	0.239	p
Dietary quality (score)	-0.68	0.24	0.008	**0.001**	-0.19	0.83	**0.023**	**0.018**	-0.18	0.21	0.376	**0.005**
Physical activity (score)	-0.36	0.26	0.181		-0.37	0.08	0.680		-0.23	0.22	0.312	
Body satisfaction (score)	0.19	0.13	0.130		0.07	0.04	0.075		0.26	0.11	**0.024**	
MODEL 2: without adjusting for depression												
Symptoms of anxiety[b] (score)[#]	0.60	0.04	0.206	$R^2_{c.100}$	0.002	0.01	0.907	$R^2_{c.100}$	0.02	0.04	0.542	$R^2_{c.100}$
Baseline Variable: WC, BMI or %BF	-0.06	0.08	0.467	13.8	0.04	0.68	0.553	4.0	-0.23	0.08	**0.008**	8.1
Age (years)	-1.36	0.66	0.045	F^6_{78}	-0.24	0.22	0.279	F^6_{87}	-0.66	0.60	0.274	F^6_{79}
Dietary quality (score)	-0.65	0.25	**0.014**	2.925	-0.18	0.08	0.031	1.56	-0.17	0.22	0.428	2.07
Physical activity (score)	-0.47	0.27	0.089	p	0.06	0.09	0.481	p	-0.31	0.23	0.190	p
Body satisfaction (score)	0.10	0.13	0.415	**0.014**	0.04	0.04	0.307	0.171	0.17	0.11	0.141	0.068
MODEL 3: complete – fully adjusted												
Separation anxiety[b] (score)	0.43	0.15	**0.006**		0.10	0.51	**0.041**		0.12	0.13	0.382	
Generalized anxiety[b] (score)	-0.29	0.21	0.190		-0.98	0.07	0.165		-0.03	0.19	0.880	
Somatic panic[b] (score)	-0.15	0.15	0.306		-0.02	0.05	0.573		-0.11	0.13	0.404	
Social phobia[b] (score)	-0.09	0.20	0.634	$R^2_{c.100}$	-0.09	0.06	0.164	$R^2_{c.100}$	-0.05	0.18	0.759	$R^2_{C.100}$
Depressive symptoms[a](0: no, 1: yes)	3.50	1.61	**0.029**	26.4	1.25	0.53	**0.022**	26.4	3.32	1.44	**0.024**	13.1
Baselinel variable: WC, BMI or %BF	-0.03	0.08	0.662	F^{11}_{78}	0.02	0.06	0.660	F^{11}_{87}	-0.23	0.85	**0.008**	F^{11}_{79}
Age (years)	-1.14	0.63	0.770	3.576	-0.15	0.21	0.481	3.576	-0.66	0.60	0.275	2.099
Dietary quality (score)	-0.68	0.24	**0.006**		-0.18	0.08	**0.023**		-0.19	0.21	0.372	
Physical activity (score)	-0.28	0.26	0.281	p	-0.02	0.08	0.764	p	-0.20	0.23	0.386	p
Body satisfaction (score)	0.18	0.12	0.148	**0.001**	0.072	0.04	0.091	**0.001**	0.24	0.11	**0.038**	**0.038**

Results

	CHANGE[d] IN WC[e]				CHANGE[d] IN BMI[f]				CHANGE[d] IN %BF			
	B[h]	SE[i]	p	Model	B	SE	p	Model	B	SE	p	Model
MODEL 4: complete – fully adjusted				$R^2_{c.100}$ 37.5 F^7_{78} 4.933 p **<0.001**				$R^2_{c.100}$ 23.7 F^7_{87} 3.253 p **0.001**				$R^2_{c.100}$ 8.4 F^7_{79} 1.607 p 0.119
Diagnosis of separation anxiety disorder[c] (0: no, 1: yes)	1.54	2.64	0.560		-0.60	0.96	0.531		-0.95	2.70	0.724	
Diagnosis of generalized anxiety disorder[c] (0: no, 1: yes)	-0.47	2.01	0.813		-0.14	0.67	0.828		1.04	2.02	0.608	
Diagnosis of panic disorder[c] (0: no, 1: yes)	5.56	3.82	0.151		2.83	1.37	**0.043**		4.33	3.80	0.260	
Diagnosis of social phobia[c] (0: no, 1: yes)	9.59	3.35	**0.006**		2.90	1.21	**0.019**		5.92	3.38	0.085	
Diagnosis of major depressive episode[c] (0: no, 1: yes)	-6.55	3.51	0.067		-2.98	1.25	**0.020**		-0.30	3.56	0.933	
Diagnosis of dysthymia[c] (0: no, 1: yes)	9.25	2.62	**0.001**		3.50	0.95	**<0.001**		1.48	2.67	0.580	
Baseline variable: WC, BMI or %BF	-0.10	0.07	0.178		-0.03	0.06	0.596		-0.26	0.09	**0.006**	
Age (years)	-0.77	0.58	0.186		-0.10	0.20	0.616		-0.48	0.61	0.426	
Dietary quality (score)	-0.49	0.23	**0.037**		-0.16	0.08	**0.041**		-0.12	0.23	0.591	
Physical activity (score)	-0.52	0.24	**0.033**		-0.07	0.08	0.389		-0.32	0.24	0.188	
Body satisfaction (score)	0.14	0.11	0.205		0.05	0.04	0.166		0.19	0.11	0.106	

Multiple linear regression models adjusted for: baseline WC, baseline BMI or baseline %BF (according to outcome variable), age, quality of diet measured by the Krece Plus test, physical activity measured by the Krece Plus physical activity questionnaire and body satisfaction according to the Body Areas Satisfaction Scale. [a]Depressive symptoms measured by the Children's Depression Inventory. [b]Anxiety symptoms measured by the Screen for Childhood Anxiety and Related Emotional Disorders. [c]Anxiety and depression disorder diagnosis determined by the MINI-Kid interview. [d]Change: difference between the endpoint (adolescence) and the baseline point (preadolescence). [e] WC: waist circumference. [f] BMI: body mass index. [g] %BF: body fat percentage. [h]B: unstandardized coefficient.[i]SE: standard error.
Level of statistical significance p value<0.05.

Table 28. Effect of emotional psychopathology in preadolescence on anthropometric and body composition parameters in adolescence in girls

	CHANGE[d] IN WC				CHANGE[d] IN BMI				CHANGE[d] IN %BF			
	B[h]	SE[i]	p	Model	B	SE	p	Model	B	SE	p	Model
MODEL 1: without adjusting for anxiety												
				$R^2_{c.100}$				$R^2_{c.100}$				$R^2_{c.100}$
Depressive symptoms[a] (0: no, 1: yes)[#]	1.08	1.24	0.382	10.7	0.53	0.39	0.117	8.2	2.23	1.14	0.053	22.0
Baseline variable: WC, BMI or %BF	-0.24	0.06	**<0.001**		-0.17	0.04	**<0.001**		-0.30	0.05	**<0.001**	
Age (years)	-0.28	0.55	0.601	F^6_{134}	0.11	0.17	0.914	F^6_{142}	0.83	0.48	0.092	F^6_{134}
Dietary quality (score)	0.18	0.21	0.400	3.557	0.02	0.06	0.720	3.038	0.17	0.19	0.384	7.032
Physical activity (score)	-0.10	0.22	0.652		0.008	0.07	0.914		-0.15	0.20	0.446	
Body satisfaction (score)	0.11	0.10	0.262	p **0.003**	0.03	0.03	0.290	p **0.008**	0.08	0.09	0.407	p **<0.001**
MODEL 2: without adjusting for depression												
				$R^2_{c.100}$				$R^2_{c.100}$				$R^2_{c.100}$
Symptoms of anxiety[b] (score)[#]	0.13	0.05	**0.012**	14.70	0.03	0.01	**0.026**	10.40	0.09	0.04	**0.050**	22.00
Baseline variable: WC, BMI or %BF	-0.24	0.06	**<0.001**		-0.17	0.04	**<0.001**		-0.30	0.05	**<0.001**	
Age (years)	-0.28	0.53	0.592	F^6_{134}	0.13	0.17	0.426	F^6_{142}	0.84	0.48	0.086	F^6_{134}
Dietary quality (score)	0.21	0.21	0.312	4.686	0.03	0.06	0.603	3.643	0.21	0.19	0.272	7.026
Physical activity (score)	-0.12	0.21	0.552		-0.005	0.06	0.946		-0.21	0.20	0.290	
Body satisfaction (score)	0.17	0.10	0.100	p **<0.001**	0.05	0.03	0.144	p **0.002**	0.10	0.09	0.298	p **<0.001**
MODEL 3: complete – fully adjusted												
Separation anxiety[b] (score)	0.24	0.14	0.101		0.08	0.04	0.102		0.12	0.13	0.370	
Generalized anxiety[b] (score)	-0.17	0.17	0.310		-0.03	0.05	0.522		-0.02	0.16	0.876	
Somatic panic[b] (score)	0.34	0.16	0.035		0.06	0.05	0.240		0.30	0.15	0.045	
Social phobia[b] (score)	0.05	0.17	0.777	$R^2_{c.100}$	0.004	0.05	0.944	$R^2_{c.100}$	-0.17	0.16	0.309	$R^2_{c.100}$
Depressive symptoms[a](0: no, 1: yes)	-0.21	1.27	0.867	16.10	0.18	0.41	0.651	10.0	1.31	1.19	0.274	24.30
Baselinel variable: WC, BMI or %BF	-0.23	0.06	**<0.001**		-0.16	0.04	**0.001**		-0.29	0.05	**<0.001**	
Age (years)	-0.10	0.55	0.844	F^{11}_{134}	0.14	0.17	0.408	F^{11}_{142}	0.93	0.49	0.063	F^{11}_{134}
Dietary quality (score)	0.22	0.21	0.290	3.450	0.03	0.06	0.602	2.519	0.18	0.19	0.356	5.103
Physical activity (score)	-0.11	0.22	0.606		0.002	0.07	0.975		-0.17	0.20	0.386	
Body satisfaction (score)	0.16	0.10	0.125	p **0.001**	0.04	0.03	0.162	p **0.009**	0.10	0.09	0.289	p **<0.001**

Results

	CHANGE[d] IN WC				CHANGE[d] IN BMI				CHANGE[d] IN %BF			
	B[h]	SE[i]	p	Model	B	SE	p	Model	B	SE	p	Model
MODEL 4: complete - fully adjusted												
Diagnosis of separation anxiety disorder[c](0: no, 1: yes)	-0.05	2.13	0.980		-0.58	0.71	0.414		-2.34	2.01	0.247	
Diagnosis of generalized anxiety disorder[c](0: no, 1: yes)	1.04	1.54	0.502		-0.001	0.52	0.998		1.31	1.46	0.370	
Diagnosis of panic disorder[c] (0: no, 1: yes)	0.58	3.88	0.988	$R^2_{c.100}$ 14.2	0.39	1.31	0.763	$R^2_{c.100}$ 6.9	3.09	3.68	0.403	$R^2_{c.100}$ 20.7
Diagnosis of social phobia[c] (0: no, 1: yes)	-0.22	2.10	0.914	F^{11}_{134} 2.930	-0.14	0.70	0.834	F^{11}_{134} 1.916	-2.13	1.99	0.286	F^{11}_{134} 4.030
Diagnosis of major depressive episode[c](0: no, 1: yes)	-1.61	4.99	0.747		-0.03	1.68	0.931		-3.12	4.71	0.509	
Diagnosis of dysthymia[c] (0: no, 1: yes)	7.86	3.23	**0.017**	p **0.002**	1.93	1.09	0.079	p **0.043**	5.47	3.05	0.076	p **<0.001**
Baseline variable: WC, BMI or %BF	-0.26	0.06	**<0.001**		-0.19	0.049	**<0.001**		-0.32	0.057	**<0.001**	
Age (years)	-0.35	0.54	0.523		0.09	0.07	0.600		0.74	0.50	0.143	
Dietary quality (score)	0.23	0.21	0.279		0.02	0.07	0.685		0.25	0.20	0.209	
Physical activity (score)	-0.14	0.22	0.521		-0.01	0.07	0.890		-0.25	0.20	0.224	
Body satisfaction (score)	0.16	0.10	0.124		0.03	0.03	0.248		0.09	0.09	0.359	

[#] Multiple linear regression models adjusted for: baseline WC, baseline BMI or baseline %BF (according to outcome variable), age, quality of diet measured by the Krece Plus test, physical activity measured by the Krece Plus physical activity questionnaire and body satisfaction according to the Body Areas Satisfaction Scale (BASS). [a]Depressive symptoms measured by the Children's Depression Inventory . [b]Anxiety symptoms measured by the Screen for Childhood Anxiety and Related Emotional Disorders. [c]Anxiety and depression disorder diagnosis determined by the MINI-Kid interview. [d]Change: difference between the endpoint (adolescence) and the baseline point (preadolescence). [e]WC: waist circumference. [f]BMI: body mass index. [g]%BF: body fat percentage.[h]B: unstandardized coefficient.[i]SE: standard error. Level of statistical significance p value <0.05.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

4. EFFECT OF EMOTIONAL SYMPTOMS ON DIETARY PATTERNS AND ANTHROPOMETRY AND BODY COMPOSITION ACCORDING TO GENETICS FACTORS

4.1 ALLELE AND GENOTYPE DISTRIBUTION OF MAOA AND 5-HTTLPR POLYMORPHISMS

MAOA alleles and genotype distributions are depicted in table 29. In girls, genotype frequencies were within the Hardy–Weinberg equilibrium (X^2= 2.127; p=0.345). Due to hemizygosity in male subjects, the Hardy–Weinberg equilibrium could not be calculated for boys.

Also, table 30 showed 5-HTTLPR alleles and genotype distributions. The genotype frequencies were also within the Hardy-Weinberg equilibrium (X^2=2.426; p=0.1128).

Table 29. Allele and genotype distribution of the MAOA polymorphism

	Alleles, n (%)					Genotypes, n (%)													
	2	**3**	**3.5**	**4**	**5**	**2/2**	**2/3**	**2/3.5**	**2/4**	**2/5**	**3/3**	**3/4**	**3/3.5**	**3/5**	**3.5/3.5**	**3.5/4**	**3.5/5**	**4/4**	**4/5**	**5/5**
Boys	1 (1.2)	30 (35.3)	3 (3.5)	50 (58.8)	1 (1.2)	1 (1.2)	-	-	-	-	30 (35.2)	-	-	-	3 (3.5)	-	-	50 (59.0)	-	1 (1.2)
Girls	1 (0.7)	87 (60.8)	4 (2.8)	118 (82.5)	4 (2.8)	0	1 (0.7)	0	0	0	19 (13.3)	62 (43.4)	2 (1.4)	3 (2.1)	0	2 (1.4)	0	53 (37.1)	1 (0.7)	0
Total	2 (.9)	117 (51.3)	7 (3.1)	168 (73.7)	5 (2.2)	1 (0.4)	1 (0.4)	0	0	0	49 (21.4)	62 (27.1)	2 (0.9)	3 (1.3)	3 (1.3)	2 (0.9)	0	103 (45.2)	1 (0.4)	1 (0.4)

Table 30. Allele and genotype distribution of the 5HTTLPR polymorphism

	Alleles, n (%)		Genotypes, n (%)		
	L	**S**	**LL**	**SL**	**SS**
Boys	45 (60.0)	59 (78.7)	16 (21.1)	29 (38.2)	31 (40.8)
Girls	89 (67.9)	99 (75.6)	32(24.1)	58 (43.6)	43 (32.3)
Total	134 (65.0)	158 (76.0)	48 (23.0)	87 (41.6)	74 (35.4)

L: Long allele; S: Short allele

4.2 GENETIC FACTORS AND EMOTIONAL SYMPTOMS

The percentage of MAOA and 5-HTTLPR polymorphism was not different between presence of emotional symptoms and control groups either in boys (X^2= p<0.05) or in girls (p<0.05) (table 31)

Table 31. Presence of emotional symptoms according to MAOA genotype and 5-HTTLPR genotype

	Boys					Girls				
	control		Emotional symptoms			control		Emotional symptoms		
	n	(%)	n	(%)	p	n	(%)	n	(%)	p
MAOA										
MAOA-L	11	(39.3)	20	(35.7)	ns	7	(13.7)	12	(13.5)	ns
MAOA-H	17	(60.7)	36	(64.3)		44	(86.3)	77	(86.5)	
5-HTTLPR										
LL	6	(25.0)	10	(19.6)	ns	14	(28.6)	18	(22.2)	ns
SS/SL	18	(75.0)	41	(80.4)		35	(71.4)	63	(77.8)	

MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism; 5-HTTLPR LL: serotonin transporter polymorphism with long alleles; 5-HTTLPR SS/SL: serotonin transporter polymorphism with short alleles or short/long alleles; ns: non-significant. Level of statistical significance: p value<0.05

Table 32 and 33 show the association of anxiety and depressive symptoms in adolescence (follow-up phase) with MAOA and 5-HTTLPR polymorphism respectively. We found that score social phobia in boys were higher in MAOA-L than MAOA-H polymorphism (MAOA-H group: 4.7±3.2 score; MAOA-L group: 6.4±3.3 score, p=0.026), whereas we did not observe differences in girls. SCARED factor scores for both genders were higher in somatic/panic, generalized anxiety and separation anxiety for MAOA-H subjects than for MAOA-L subjects but these results were not statistically significant (table 32). In contrast, girls with 5-HTTLPR SS/SL polymorphism showed higher scores in depressive and anxiety symptoms than girls with 5-HTTLPR LL polymorphism. However there were no differences among boys (Table 33).

Table 32. Association between the MAOA genotype and anxiety and depressive symptoms in adolescence

	Boys			Girls		
	MAOA-L (n=31)	**MAOA-H** (n=54)		**MAOA-L** (n=20)	**MAOA-H** (n=123)	
	Mean (SD)	Mean (SD)	p	Mean (SD)	Mean (SD)	*p*
Depressive symptoms (YI-4 score)	12.6 (5.9)	14.3(7.7)	ns	14.0(8.9)	15.1 (8.6)	ns
Anxiety symptoms (SCARED score)	18.2 (6.4)	17.9 (9.6)	ns	19.9 (6.3)	21.4 (10.3)	ns
Somatic panic (score)	2.1 (1.6)	2.4 (2.5)	ns	3.0 (2.4)	3.8 (3.5)	ns
Social phobia (score)	6.4 (3.3)	4.7 (3.2)	**0.026**	6.3 (2.8)	5.2 (3.2)	ns
Generalized anxiety (score)	5.8 (3.0)	5.8 (3.6)	ns	5.3 (2.7)	6.8 (3.4)	ns
Separation anxiety(Score)	4.0 (2.6)	4.8 (2.8)	ns	5.2 (2.4)	5.6 (3.8)	ns

MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism; YI-4: Youth's Inventory-4; SCARED: Screen for Childhood Anxiety and Related Emotional Disorders
SD: Standard deviation; ns: non-significant
Level of statistical significance: p <0.05

Table 33. Association between the 5-HTTLPR genotype and anxiety and depressive symptoms in adolescence

	Boys			Girls		
	5-HTTLPR LL (n=16)	**5-HTTLPR SS/SL** (n=59)		**5-HTTLPR LL** (n=32)	**5-HTTLPR SS/SL** (n=98)	
	Mean (SD)	Mean (SD)	*p*	Mean (SD)	Mean (SD)	*p*
Depressive symptoms (YI-4 score)	14.5 (5.3)	13.5 (7.5)	ns	11.9 (9.0)	15.8 (8.2)	**0.021**
Anxiety symptoms (SCARED score)	17.0 (6.4)	17.6 (9.2)	**ns**	18.4 (8.7)	22.3 (9.9)	**0.046**
Somatic panic (score)	1.5 (1.3)	2.3 (2.2)	ns	3.2 (3.0)	3.9 (3.5)	ns
Social phobia (score)	5.3 (2.8)	5.1 (3.3)	ns	5.3 (2.9)	5.5 (3.2)	ns
Generalized anxiety (score)	5.9 (2.8)	5.6 (3.6)	ns	5.2 (2.9)	7.0 (3.3)	**0.006**
Separation anxiety(Score)	4.1 (2.0)	4.4 (2.8)	ns	4.5 (3.4)	5.8 (3.7)	ns

5-HTTLPR LL: serotonin transporter polymorphism with long alleles; 5-HTTLPR SS/SL: serotonin transporter polymorphism with short alleles or short/long alleles; SD: Standard deviation; ns: non-significant ; YI-4: Youth's Inventory-4; SCARED: Screen for Childhood Anxiety and Related Emotional Disorders.
Level of statistical significance: p value<0.05

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

4.3 GENETIC FACTORS AND DIETARY PATTERNS

4.3.1 Genetic factors and dietary patterns

Table 34 and 35 describe the scores of dietary pattern, Mediterranean diet and physical activity according to MAOA-L and MAOA-H polymorphism. However we did not find differences in these scores between MAOA-L and MAOA-H polymorphism either in girls or boys. Only, we observed that girls with MAOA-H obtained higher scores in the television and game factor than MAOA-L polymorphism (table 35).

Table 34. Association between the MAOA genotype and dietary patterns

	Boys					**Girls**				
	MAOA-L (n= 23)		**MAOA-H** (n= 32)			**MAOA-L** (n=14)		**MAOA-H** (n=87)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Sweet and Fatty Food Pattern (score)	0.5	(1.5)	0.01	(0.8)	ns	-0.01	(0.8)	-0.1	(0.8)	ns
Western Pattern (score)	0.06	(1.0)	-0.1	(0.7)	ns	0.2	(0.8)	0.007	(1.0)	ns
Healthy Pattern (score)	0.2	(1.4)	-0.2	(0.9)	ns	0.1	(0.8)	0.008	(0.9)	ns

MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism; SD: Standard deviation; ns: non-significant. Level of statistical significance: p value<0.05

Table 35. Association between the MAOA genotype and Mediterranean diet adherence and physical activity

	Boys					**Girls**				
	MAOA-L (n= 23)		**MAOA-H** (n= 32)			**MAOA-L** (n=14)		**MAOA-H** (n=87)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Mediterranean Diet (Score)	6.0	(2.4)	5.5	(2.1)	ns	5.4	(2.7)	5.5	(2.2)	ns
Physical activity Test (score)	6.4	(2.0)	6.4	(2.3)	ns	4.8	(2.4)	5.3	(2.7)	ns
Physical activity factor (score)	3.3	(1.9)	3.2	(1.9)	ns	2.4	(2.1)	2.3	(1.8)	ns
Television and Games factor (score)	3.1	(1.9)	3.2	(1.9)	ns	2.4	(1.4)	3.1	(1.1)	**0.029**

MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism; SD: Standard deviation; ns: non-significant. Level of statistical significance: p value<0.05

Regarding the 5-HTTLPR genotype, we did not find statistical differences on the dietary patterns, Mediterranean diet and physical activity scores (tables 36 and 37). In girls, sweet and fatty food pattern tended to be higher in the group of 5-HTTLPR SS/SL than in the 5-HTTLPR group LL polymorphism, whereas in the group of 5-HTTLPR SS/SL showed lower scores in western and healthy pattern than 5-HTTLPR LL, but these results were not statistically significant (table 37). There were no differences in food consumption, energy and nutrient intake between MAOA-H and MAOA-L polymorphism and neither between 5-HTTLPR SS/SL and LL (data not shown).

Table 36. Association between the 5-HTTLPR genotype and dietary patterns

	Boys					**Girls**				
	5-HTTLPR LL (n= 8)		**5-HTTLPR SS/SL** (n=40)			**5-HTTLPR LL** (n= 28)		**5-HTTLPR SS/SL** (n=67)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Sweet and Fatty Food Pattern (score)	0.05	(0.9)	0.07	(1.0)	ns	-0.3	(0.6)	0.06	(0.9)	ns
Western Pattern (score)	-0.3	(1.0)	-0.06	(0.7)	ns	0.1	(1.2)	-0.4	(1.0)	ns
Healthy Pattern (score)	0.4	(0.8)	-0.2	(0.9)	ns	0.03	(0.7)	-0.3	(0.9)	ns

5-HTTLPR LL: serotonin transporter polymorphism with long alleles; 5-HTTLPR SS/SL: serotonin transporter polymorphism with short alleles or short/long alleles; SD: Standard deviation; ns: non-significant.
Level of statistical significance: p value<0.05

Table 37. Association between the 5-HTTLPR genotype and Mediterranean diet adherence and physical activity

	Boys					**Girls**				
	5-HTTLPR LL (n= 8)		**5-HTTLPR SS/SL** (n=40)			**5-HTTLPR LL** (n= 28)		**5-HTTLPR SS/SL** (n=67)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Mediterranean Diet (score)	5.6	(2.8)	5.9	(2.2)	ns	5.8	(2.1)	5.5	(2.3)	ns
Physical activity Test (score)	6.4	(2.3)	6.4	(2.2)	ns	5.2	(2.1)	5.3	(2.2)	ns
Physical activity factor (score)	3.1	(1.9)	3.3	(1.9)	ns	2.3	(1.9)	2.3	(1.8)	ns
Television and Games factor (score)	3.3	(1.0)	3.1	(1.0)	ns	2.9	(1.1)	3.0	(1.2)	ns

5-HTTLPR LL: serotonin transporter polymorphism with long alleles; 5-HTTLPR SS/SL: serotonin transporter polymorphism with short alleles or short/long alleles; SD: Standard deviation; ns: non-significant
Level of statistical significance: p value<0.05

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

4.3.2 Association between emotional symptoms and dietary patterns according to genetic factors

To observe whether there were differences in dietary patterns, Mediterranean diet and physical activity scores between presence or not of emotional symptoms, the analyses were conducted separately according to the MAOA and 5-HTTLPR polymorphism.

Regarding the MAOA polymorphism, we found that, in MAOA-L group, girls with emotional symptoms showed higher scores of sweet and fatty food pattern (p=0.032) (table 38). In addition, they also showed significantly less Mediterranean diet score (p=0.043) and physical activity score (physical activity test, p=0.002; and physical activity factor, p=0.010) than control group (table 39). There were no differences between presences of emotional symptoms or control group in girls with MAOA-L. Boys with or without emotional symptoms did not show differences in scores of dietary patterns, Mediterranean diet and physical activity in either group of MAOA polymorphism (tables 40-41).

Table 38. Dietary patterns according to MAOA genotype and emotional symptoms in girls

	MAOA-L					**MAOA-H**				
	Control (n= 5)		**Emotional symptoms** (n= 9)			**Control** (n= 33)		**Emotional symptoms** (n= 54)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Sweet and Fatty Food Pattern (score)	-0.2	(0.5)	0.1	(1.0)	ns	-0.4	(0.7)	0.1	(0.9)	**0.031**
Western Pattern (score)	0.1	(0.3)	0.4	(1.1)	ns	-0.1	(1.1)	0.1	(1.1)	ns
Healthy Pattern (score)	-0.3	(0.8)	0.4	(0.8)	ns	0.1	(0.9)	-0.4	(1.0)	ns

MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism; SD: Standard deviation; ns: non-significant. Level of statistical significance: p value <0.05

Table 39. Mediterranean diet and physical activity according to MAOA genotype and emotional symptoms in girls

	MAOA-L					MAOA-H				
	Control (n= 5)		Emotional symptoms (n= 9)			Control (n= 33)		Emotional symptoms (n= 54)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Mediterranean Diet (Score)	5.6	(2.8)	5.3	(2.7)	ns	5.9	(1.8)	5.1	(2.3)	**0.043**
Physical activity Test (score)	5.0	(2.8)	4.7	(2.2)	ns	6.1	(2.1)	4.9	(2.0)	**0.002**
Physical activity factor (score)	2.3	(2.4)	2.4	(2.0)	ns	2.8	(1.8)	2.0	(1.7)	**0.010**
Television and Games factor (score)	2.7	(1.4)	2.3	(1.5)	ns	3.3	(0.8)	2.9	(1.3)	ns

MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism; SD: Standard deviation; ns: non-significant. Level of statistical significance: p value<0.05

Table 40. Dietary patterns according to MAOA genotype and emotional symptoms in boys

	MAOA-L					MAOA-H				
	Control (n= 7)		Emotional symptoms (n= 16)			Control (n= 10)		Emotional symptoms (n= 22)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Sweet and Fatty Food Pattern (score)	0.7	(1.7)	0.4	(1.5)	ns	-0.05	(0.7)	0.05	(0.8)	ns
Western Pattern (score)	-0.2	(0.9)	0.1	(1.1)	ns	-0.2	(0.9)	-0.1	(0.5)	ns
Healthy Pattern (score)	0.1	(2.4)	0.2	(0.8)	ns	-0.03	(1.0)	-0.3	(0.8)	ns

MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism; SD: Standard deviation; ns: non-significant. Level of statistical significance: p value<0.05

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Table 41. Mediterranean diet and physical activity according to MAOA genotype and emotional symptoms in boys

	MAOA-L					MAOA-H				
	Control (n= 7)		Emotional symptoms (n= 16)			Control (n= 10)		Emotional symptoms (n= 22)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Mediterranean Diet (Score)	5.3	(1.9)	6.3	(2.6)	ns	5.7	(2.4)	5.3	(2.0)	ns
Physical activity Test (score)	5.5	(1.6)	6.8	(2.0)	ns	7.0	(2.1)	6.1	(2.2)	ns
Physical activity factor (score)	2.9	(2.3)	3.5	(1.6)	ns	3.2	(1.5)	2.9	(1.9)	ns
Television and Games factor (score)	2.6	(1.1)	3.3	(0.9)	ns	3.2	(1.0)	3.1	(1.1)	ns

MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism SD: Standard deviation; ns: non-significant. Level of statistical significance: p value<0.05

In regard to 5-HTTLPR polymorphism, table 42 and 43 show the association between emotional symptoms and score of dietary pattern score, Mediterranean diet and physical activity. In groups of girls with 5-HTTLPR LL polymorphism, healthy pattern scores were higher in emotional symptoms than control group (p=0.038). Meanwhile, girls with 5-HTTLPR SS/SL polymorphism and emotional symptoms obtained higher scores in sweet and fatty food pattern than control group (p=0.030, table 43). Moreover, they also showed significantly lower Mediterranean diet score (p=0.030) and physical activity score (total test, p=0.027; and physical activity factor, p=0.002) than control group (table 42). In boys, significant differences were not found (table 44 and 45).

Table 42. Dietary patterns according to 5-HTTLPR genotype and emotional symptoms in girls

	5-HTTLPR LL					5-HTTLPR SS/SL				
	Control (n= 13)		Emotional symptoms (n= 15)			Control (n=23)		Emotional symptoms (n= 44)		
	Mean	(SD)	Mean	(SD)	*p*	Mean	(SD)	Mean	(SD)	*p*
Sweet and Fatty Food Pattern (score)	-0.5	(0.6)	-0.3	(0.7)	ns	-0.3	(0.8)	0.2	(1.0)	**0.030**
Western Pattern (score)	0.2	(0.1)	0.2	(1.2)	ns	-0.3	(0.8)	0.1	(1.1)	ns
Healthy Pattern (score)	-0.3	(0.7)	0.3	(0.8)	**0.038**	0.2	(0.9)	-0.2	(0.9)	ns

5-HTTLPR LL: serotonin transporter polymorphism with long alleles; 5-HTTLPR SS/SL: serotonin transporter polymorphism with short alleles or short/long alleles; SD: Standard deviation; ns: non-significant.
Level of statistical significance: p value<0.05

Table 43. Mediterranean diet and physical activity according to 5-HTTLPR genotype and emotional symptoms in girls

	5-HTTLPR LL					5-HTTLPR SS/SL				
	Control (n= 13)		Emotional symptoms (n= 15)			Control (n=23)		Emotional symptoms (n= 44)		
	Mean	(SD)	Mean	(SD)	*p*	Mean	(SD)	Mean	(SD)	*p*
Mediterranean Diet (Score)	5.6	(1.7)	5.9	(2.4)	ns	6.1	(2.1)	5.2	(2.3)	**0.030**
Physical activity Test (score)	5.6	(1.8)	4.8	(2.3)	ns	6.0	(2.4)	4.9	(1.9)	**0.027**
Physical activity factor (score)	2.5	(1.7)	2.1	(2.0)	ns	2.8	(1.9)	2.1	(1.7)	**0.002**
Television and Games factor (score)	3.1	(0.8)	2.7	(1.2)	ns	3.2	(1.0)	2.9	(1.3)	ns

5-HTTLPR LL: serotonin transporter polymorphism with long alleles; 5-HTTLPR SS/SL: serotonin transporter polymorphism with short alleles or short/long alleles; SD: Standard deviation; ns: non-significant.
Level of statistical significance: p value<0.05

Table 44. Dietary patterns according to 5-HTTLPR genotype and emotional symptoms in boys

	5-HTTLPR LL					5-HTTLPR SS/SL				
	Control (n= 3)		Emotional symptoms (n= 5)			Control (n=11)		Emotional symptoms (n= 29)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	*p*
Sweet and Fatty Food Pattern (score)	0.5	(1.1)	-0.2	(0.7)	ns	-0.2	(0.5)	0.1	(1.1)	ns
Western Pattern (score)	-0.9	(1.1)	0.06	(0.8)	ns	0.07	(0.7)	-0.1	(0.7)	ns
Healthy Pattern (score)	0.6	(1.3)	0.3	(0.5)	ns	-0.3	(1.0)	-0.1	(0.9)	ns

5-HTTLPR LL: serotonin transporter polymorphism with long alleles; 5-HTTLPR SS/SL: serotonin transporter polymorphism with short alleles or short/long alleles; SD: Standard deviation; ns: non-significant.
Level of statistical significance: p value<0.05

Table 45. Mediterranean diet and physical activity according to 5-HTTLPR genotype and emotional symptoms in boys

	5-HTTLPR LL					5-HTTLPR SS/SL				
	Control (n= 5)		Emotional symptoms (n= 5)			Control (n=11)		Emotional symptoms (n= 29)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Mediterranean Diet (score)	6.0	(2.0)	5.3	(3.3)	ns	5.7	(2.4)	5.9	(2.0)	ns
Physical activity Test (score)	5.3	(3.0)	7.1	(1.5)	ns	7.0	(1.6)	6.1	(2.3)	ns
Physical activity factor (score)	2.3	(2.0)	3.6	(1.7)	ns	3.7	(1.8)	3.0	(1.9)	ns
Television and Games factor (score)	3.0	(1.5)	3.5	(0.5)	ns	3.2	(0.6)	3.0	(1.1)	ns

5-HTTLPR LL: serotonin transporter polymorphism with long alleles; 5-HTTLPR SS/SL: serotonin transporter polymorphism with short alleles or short/long alleles; SD: Standard deviation; ns: non-significant.
Level of statistical significance: p value<0.05

4.3.3 Effect of emotional symptoms on dietary pattern according to genetic factors

We performed several multiple linear regression models to examine the main effect of emotional symptoms and genetic factor and their interaction on sweet and fatty food pattern score adherence. Table 46 describes the five multiple linear regression models, unadjusted and adjusted, of the main effect of emotional symptoms and 5-HTTLPR and MAOA polymorphism on sweet and fatty food pattern in girls. The models were adjusted for age, SES, BMI and energy intake (kcal).

Model 1 (unadjusted) and model 2 (adjusted) of 5-HTTLPR showed that the presence of emotional symptoms and 5-HTTLPR SS/SL increased significantly the sweet and fatty food pattern adherence in girls. Otherwise, model 3 and 4 showed that while emotional symptoms increased the sweet and fatty food score adherence, the MAOA polymorphism did not have any effect. When we combined adjusted models of 5-HTTLPR and MAOA (in model 5), the association remained. The presence of emotional symptoms increased in 0.4 score of sweet and food pattern as well as 5-HTTLPR SS/SL increased in 0.4 score of sweet and food pattern, whereas, MAOA polymorphism were not significant. In addition, a high age and energy intake were associated with higher scores of sweet and fatty food pattern and a high SES was associated with lower scores of sweet and fatty food pattern.

In males, 5-HTTLPR polymorphism (b=0.001; p=0.998) and emotional symptoms (b=0.179; p=0.597) did not predict higher scores of sweet and fatty food pattern (R^2_{c*100}=-3.8; $F_{2,45}$=0.143; p=0.867); likewise, MAOA polymorphism (b=-0.53; p=0.112) and emotional symptoms (b=-0.05; p=0.873) did not have an effect on higher scores of sweet and fatty food pattern (R^2_{c*100}=1.2; $F_{2,52}$=1.314; p =0.277) (data not shown).

Table 46. Effect of emotional symptoms and 5-HTTLPR and MAOA polymorphisms on sweet and fatty food pattern in girls

	B	SE	p	Model
Model 1: 5-HTTLPR				
Intercept	-0.5	0.1	**0.002**	R^2_{c*100}=8.3
5-HTTLPR (0: LL; 1: SS-SL)	0.3	0.1	**0.046**	$F_{2,92}$=5.23
Emotional symptoms (0: No; 1: yes)	0.4	0.1	**0.025**	p =0.007
Model 2: 5-HTTLPR adjusted				
Intercept	-4.4	1.2	**0.001**	R^2_{c*100}=24.8
5-HTTLPR (0: LL; 1: SS-SL)	0.4	0.1	**0.020**	$F_{6,87}$ =6.1
Emotional symptoms (0: No; 1: yes)	0.4	0.1	**0.016**	p <0.001
Age (years)	0.2	0.08	**0.026**	
Socioeconomic status (score)	-0.1	0.04	**<0.001**	
zBMI (score)	-0.08	0.09	0.359	
Energy intake (kcal)	0.001	0.09	**<0.001**	
Model 3: MAOA				
Intercept	-0.2	0.2	0.294	R^2_{c*100}=3.2
MAOA (0: Low; 1: High)	-0.08	0.2	0.730	$F_{2,98}$=2.67
Emotional symptoms (0: No; 1: yes)	0.4	0.1	**0.025**	p =0.074
Model 4: MAOA Adjusted				
Intercept	-0.38	1.2	**0.003**	
MAOA (0: Low; 1: High)	-0.1	0.2	**0.673**	R^2_{c*100}=18.6
Emotional symptoms (0: No; 1: yes)	0.4	0.1	0.011	$F_{6,93}$ =4.7
Age (years)	0.2	0.08	**0.020**	p <0.001
Socioeconomic status (score)	-0.1	0.04	**0.007**	
zBMI (score)	-0.1	0.09	0.178	
Energy intake (kcal)	0.01	0.001	**0.002**	
Model 5: 5-HTTLPR + MAOA				
Intercept	-4.1	1.3	**0.002**	
5-HTTLPR (0: LL; 1: SS-SL)	0.4	0.1	**0.018**	
MAOA (0: Low; 1: High)	-0.2	0.2	0.386	
Emotional symptoms (0: No; 1: yes)	0.4	0.1	**0.024**	R^2_{c*100}=23.7
Age (years)	0.1	0.09	**0.033**	$F_{7,83}$=4.9
Socioeconomic status (score)	-0.1	0.04	**0.022**	p <0.001
zBMI (score)	-0.09	0.09	0.343	
Energy intake (kcal)	0.001	0.001	**0.001**	

B: unstandardized coefficient; SE: standard error; 5-HTTLPR LL: serotonin transporter polymorphism with long alleles; 5-HTTLPR SS/SL: serotonin transporter polymorphism with short alleles or short/long alleles; MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism; zBMI: z-score of BMI.
Level of statistical significance: p value<0.05

Furthermore, we ran interaction models of two-way interaction 5-HTTLPR x emotional symptoms and MAOA-x-emotional symptoms and three-way interaction 5-HTTLPR-x-MAOA-x-emotional symptoms. In girls, interactions terms were not significant for 5-HTTLPR-x-emotional symptoms and 5-HTLPR-x-MAOA-x-emotional symptoms were no significant (b=0.32, p=0.344; b=0.4, p=0.216, respectively) (data not shown in the tables). However, MAOA-x-emotional symptoms was significant (table 47, b=0.5; p=0.022). In boys, there was no interaction between terms of 5-HTTLPR-x-emotional symptoms (b=0.8, p=0.170), MAOA-x-emotional symptoms (b=0.5, p=0.260) and 5-HTTLPR and MAOA-x-emotional symptoms (b=0.2; p=0.350) (data not shown).

Therefore, due to the significant interaction between MAOA and emotional symptoms in girls, we performed stratified analyses of MAOA-L and MAOA-H. The stratified analyses of MAOA polymorphism in girls can be observed in table 47. There was no effect of emotional symptoms on sweet and fatty food pattern score in girls with MAOA-L, whereas in girls with MAOA-H, emotional symptoms increased 0.3 points of sweet and fatty food pattern score (p= 0.036). Due to a lack of significant association in interaction terms in boys, stratified analysis were not performed.

Table 47. Effect of emotional symptoms on sweet and fatty food pattern interaction and stratified by MAOA genotype in girls

	B	SE	p	Model
Model: Interaction MAOA x emotional symptoms				
Intercept	-4.0	1.2	**0.002**	
interaction term (MAOA x emotional symptoms	0.5	0.2	**0.022**	
Emotional symptoms (0: No; 1: yes)	0.09	0.2	0.692	R^2_{c*100}=-22.1
MAOA (0: Low; 1: High)	-0.1	0.2	0.636	$F_{7,93}$=4.81
Age (years)	0.2	0.08	**0.020**	p <0.001
SES (score)	-0.1	0.04	**0.026**	
energy intake (kcal)	0.001	0.001	0.429	
zBMI (score)	-0.7	0.09	**0.429**	
Model: MAOA-H				
Intercept	-3.1	1.4	**0.028**	
Emotional symptoms (0: No; 1: yes)	0.3	0.1	**0.036**	R^2_{c*100}=-16.1
Age (years)	0.1	0.09	0.138	$F_{5,82}$=4.31
SES (score)	-0.1	0.04	**0.013**	p =0.002
zBMI (score)	-0.1	0.09	0.090	
Energy intake (kcal)	0.001	0.001	**0.007**	
Model: MAOA- L				
Intercept	-8.2	2.5	**0.014**	
Emotional symptoms (0: No; 1: yes)	0.8	0.4	0.086	R^2_{c*100}=-36.4
Age (years)	0.5	0.1	**0.022**	$F_{5,11}$=2.860
SES (score)	-0.1	0.1	0.193	p =0.054
zBMI (score)	0.3	0.3	0.344	
Energy intake (kcal)	0.001	0.001	**0.082**	

B: unstandardized coefficient; SE: standard error; MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism; zBMI: z-score of BMI.
Level of statistical significance: p value<0.05

4.4 GENETIC FACTORS AND ANTHROPOMETRIC AND BODY COMPOSITION PARAMETERS.

4.4.1 Genetic factors and anthropometric and body composition parameters

Table 48 shows the association between anthropometric and body composition parameters and MAOA genotype. Non statistical differences were found either in girls or boys. Girls tended to show higher increase in change in BMI and %BF in the MAOA-H group than the MAOA-L group.

The frequencies of overweight and obese by MAOA genotype and gender can be seen in table 49. Although there were no significant differences, we found a higher percentage of overweigh/obesity in girls with MAOA-H in comparison to girls with MAOA-L, both preadolescence and adolescence. A 30.4% of girls in preadolescence and a 17.4% of girls in adolescence in MAOA-H group were overweight or obese by contrast MAOA-L group had 17.6% of girls in preadolescence and 5.9% of girls in adolescence overweight and obese. In contrast, there was a lower percentage of overweight/obese boys in MAOA-H group than in the MAOA-L group, in both preadolescence and adolescence.

Table 48. Anthropometric and body composition parameters according to MAOA genotype

	Boys					Girls				
	MAOA-L (n= 29)		MAOA-H (n= 49)			MAOA-L (n=17)		MAOA-H (n=115)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Preadolescence[a]										
Weight (kg)	37.0	(6.7)	37.0	(8.2)	ns	38.2	(7.0)	41.0	(10.5)	ns
Height (cm)	142.5	(6.63)	141.8	(7.6)	ns	143.4	(6.3)	145.7	(9.0)	ns
BMI (kg/m^2)	18.3	(2.6)	18.3	(2.7)	ns	18.5	(2.9)	19.1	(3.5)	ns
zBMI (score)	0.1	(1.0)	-0.01	(0.8)	ns	0.002	(0.8)	0.1	(0.1)	ns
Waist Circumference (cm)	65.5	(6.2)	66.5	(7.1)	ns	65.2	(6.7)	66.2	(8.3)	ns
Body fat (%)	19.3	(5.2)	19.9	(6.0)	ns	23.5	(8.8)	24.3	(8.4)	ns
Adolescence[b]										
Weight (kg)	52.9	(8.4)	52.5	(9.6)	ns	50.0	(7.0)	53.0	(10.5)	ns
Height (cm)	164.6	(8.2)	163	(9.4)	ns	159.8	(4.7)	160.3	(6.7)	ns
BMI (kg/m^2)	19.5	(3.0)	18.8	(4.7)	ns	19.5	(2.5)	20.4	(4.0)	ns
zBMI (score)	-0.1	(0.9)	-0.1	(0.8)	ns	-0.2	(0.7)	0.01	(1.0)	ns
Waist Circumference (cm)	71.8	(6.8)	73.9	(7.6)	ns	70.7	(6.6)	71.8	(8.1)	ns
Hip Circumference (cm)	77.8	(7.3)	79.2	(7.0)	ns	79.3	(5.8)	81.7	(8.8)	ns
Waist-Hip ratio	0.9	(0.03)	0.9	(0.4)	ns	0.9	(0.03)	0.9	(0.04)	ns
Biceps skinfold (mm)	8.8	(5.4)	9.2	(5.1)	ns	9.2	(4.0)	10.7	(5.4)	ns
Tricep skinfold (mm)	13.7	(7.9)	13.7	(6.8)	ns	17.2	(4.2)	18.0	(6.3)	ns
Subscapular skinfold (mm)	11.4	(6.5)	11.8	(6.3)	ns	12.0	(5.6)	14.5	(6.2)	ns
Tricep-subscapular skinfold ratio	1.2	(0.4)	1.2	(0.3)	ns	1.6	(0.5)	1.3	(0.3)	**0.036**
Body fat (%)	13.0	(6.1)	13.1	(6.1)	ns	23.1	(7.1)	25.1	(7.6)	ns
Change[c] from preadolescence to adolescence										
Change in BMI (kg/m^2)	1.2	(1.9)	1.3	(1.5)	ns	0.9	(2.3)	1.3	(1.8)	ns
Change in Waist Circumference (cm)	6.7	(4.8)	7.7	(5.3)	ns	5.4	(6.1)	5.5	(5.7)	ns
Change in Body Fat (%)	-3.9	(5.2)	-3.6	(4.4)	ns	-1.3	(5.3)	1.1	(5.6)	ns

[a]Preadolescence: baseline phase; [b]Adolescence; follow-up phase. [c]Change: difference between the baseline point (preadolescence) and the endpoint (adolescence).
MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism; BMI: Body Mass Index; zbMI: z-score of BMI; SD: Standard deviation; ns: non-significant
Level of statistical significance: p value<0.05

Table 49. MAOA genotype and frequencies by normoweight and overweight/obesity

	Boys (n=78)					Girls (n=132)				
	MAOA-L		MAOA-H			MAOA-L		MAOA-H		
	n	(%)	n	(%)	p	n	(%)	n	%	p
Preadolescence[a]										
normoweight	21	(72.4)	38	(77.6)		14	(82.4)	80	(69.6)	
overweight /obesity	8	(27.6)	11	(22.4)	ns	3	(17.6)	35	(30.4)	ns
Adolescence[b]										
normoweight	24	(82.8)	42	(85.7)		16	(94.1)	95	(82.6)	
overweight /obesity	5	(17.2)	7	(14.3)	ns	1	(5.9)	20	(17.4)	ns

[a]Preadolescence: baseline phase; [b]Adolescence; follow-up phase.
MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism; ns: non-significant. Level of statistical significance: p value<0.05

The association between anthropometric and body composition parameters and 5-HTTLPR genotype is showed in table 50. We found a higher increase in change in BMI and WC in the 5-HTTLPR SS/SL group than the LL group in girls, although the association was not significant. The percentage of overweight and obesity did not show significant differences between 5-HTTLPR SS/SL and 5-HTTLPR LL (table 51).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Table 50. Anthropometric and body composition parameters according to 5-HTTLPR genotype

	Boys					Girls				
	5-HTTLPR LL (n= 16)		5-HTTLPR SS/SL (n=53)			5-HTTLPR LL (n= 30)		5-HTTLPR SS/SL (n=93)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Preadolescence[a]										
Weight (kg)	38.5	(11.6)	37.1	(6.4)	ns	40.3	(10.4)	40.5	(10.4)	ns
Height (cm)	142.6	(8.6)	142.2	(7.1)	ns	146.5	(10.1)	145.0	(8.5)	ns
BMI ((kg/m^2)	18.3	(2.5)	18.6	(3.3)	ns	18.7	(3.3)	19.1	(3.4)	ns
zBMI (score)	-0.07	(0.8)	0.1	(0.9)	ns	0.05	(0.9)	0.1	(0.9)	ns
Waist Circumference (cm)	65.9	(9.2)	66.3	(5.9)	ns	66.5	(8.6)	65.5	(8.0)	ns
Body fat (%)	19.2	(5.6)	19.7	(5.7)	ns	22.0	(8.4)	22.0	(8.5)	ns
Adolescence[b]										
Weight (kg)	54.5	(9.8)	52.6	(9.0)	ns	52.7	(10.0)	52.8	(10.5)	ns
Height (cm)	164.9	(8.4)	163.8	(9.1)	ns	161.2	(6.5)	159.9	(6.5)	ns
BMI (kg/m^2)	19.5	(2.7)	20.0	(3.5)	ns	20.2	(3.4)	20.4	(4.0)	ns
zBMI (score)	-0.06	(0.8)	-0.1	(0.9)	ns	-0.06	(1.0)	0.02	(1.0)	ns
Waist Circumference (cm)	72.7	(8.2)	73.5	(7.3)	ns	71.9	(8.1)	71.8	(8.1)	ns
Body fat (%)	11.0	(6.0)	11.6	(6.0)	ns	25.3	(7.4)	24.8	(7.9)	ns
Hip Circumference (cm)	79.0	(8.0)	78.9	(6.9)	ns	81.4	(8.8)	81.6	(8.5)	ns
Waist-Hip ratio	0.9	(0.03)	0.9	(0.04)	ns	0.9	(0.04)	0.9	(0.04)	ns
Biceps skinfold (mm)	8.3	(4.8)	9.4	(5.0)	ns	10.9	(5.9)	10.3	(5.3)	ns
Tricep skinfold (mm)	13.5	(7.2)	13.7	(7.0)	ns	17.2	(6.6)	18.3	(6.1)	ns
Subscapular skinfold (mm)	11.9	(6.1)	11.8	(6.4)	ns	13.8	(6.3)	14.6	(6.2)	ns
Tricep-subscapular ratio	1.1	(0.3)	1.2	(0.3)	ns	1.3	(0.3)	1.3	(0.4)	ns
Change[c] from preadolescence to adolescence										
Change in BMI (kg/m^2)	1.4	(1.6)	1.3	(1.7)	ns	1.4	(1.8)	1.4	(1.7)	ns
Change in Waist Circumference (cm)	7.1	(5.9)	7.5	(5.1)	ns	5.2	(4.8)	6.1	(5.8)	ns
Change in BodyFat (%)	-5.9	(5.6)	-5.6	(4.6)	ns	1.0	(5.2)	0.9	(5.4)	ns

[a]Preadolescence: baseline phase; [b]Adolescence; follow-up phase. [c]Change: difference between the baseline point (preadolescence) and the endpoint (adolescence).
5-HTTLPR LL: serotonin transporter polymorphism with long alleles; 5-HTTLPR SS/SL: serotonin transporter polymorphism with short alleles or short/long alleles; BMI: Body Mass Index; zbMI: z-score of BMI; SD: Standard deviation; ns: non-significant.
Level of statistical significance: p-value<0.05

Table 51. **5-HTTLPR genotype and frequencies by normoweight and overweight/obesity**

	Boys (n=69)					**Girls** (n=123)				
	5-HTTLPR LL		**5-HTTLPR SS/SL**			**5-HTTLPR LL**		**5-HTTLPR SS/SL**		
	n	(%)	n	(%)	p	n	(%)	n	%	p
Preadolescence[a]										
normoweight	13	(81.3)	39	(73.6)	ns	25	(27.8)	65	(72.2)	ns
overweight/obesity	3	(18.8)	14	(26.4)		5	(15.2)	28	(30.1)	
Adolescence[b]										
normoweight	13	(81.3)	45	(84.9)	ns	25	(83.3)	77	(75.0)	ns
overweight/obesity	3	(18.8)	8	(15.1)		5	(16.7)	16	(17.2)	

[a]Preadolescence: baseline phase; [b]Adolescence; follow-up phase.
5-HTTLPR LL: serotonin transporter polymorphism with long alleles; 5-HTTLPR SS/SL: serotonin transporter polymorphism with short alleles or short/long alleles; ns: non-significant.
Level of statistical significance: p value<0.05

4.4.2 Association between emotional symptoms and anthropometric and body composition parameters according to genetic factors

We examined the association between emotional symptoms and anthropometric and body composition variables stratified by genetic factor. Table 52 shows the association between emotional symptoms and anthropometric and body composition parameter according to MAOA genotype in girls. We found a mixed and non-significant trend in the anthropometric and body composition variables in preadolescence and adolescence. We only found that girls with MAOA-H and emotional symptoms showed higher increase of %BF (1.9±5.8%) than control (-0.48±4.9%, p=0.035). In addition, girls with MAOA-H and emotional symptoms showed higher values of change in %BF than girls with MAOA-L and emotional symptoms (presence of emotional symptoms, MAOA-H group: 1.9±5.8% vs MAOA-L group: -1.7±5.3%, p=0.049, data not shown in the table). Likewise, in MAOA-H group, changes in BMI and WC were higher in girls with emotional symptoms than control group, although they were no significant.

Table 52. Anthropometric and body composition parameters according to MAOA genotype and emotional symptoms in girls

	MAOA-L					MAOA-H				
	Control (n= 5)		Emotional symptoms (n= 12)			Control (n=43)		Emotional symptoms (n=72)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Preadolescence[a]										
Weight (kg)	36.5	(9.3)	39.0	(6.5)	ns	41.9	(10.3)	40.6	(10.8)	ns
Height (cm)	143.6	(8.7)	143.3	(5.2)	ns	143.6	(8.7)	143.3	(5.2)	ns
BMI (kg/m^2)	17.4	(2.5)	19.0	(3.0)	ns	19.8	(3.7)	18.6	(3.2)	ns
zBMI (score)	-0.29	(0.5)	0.2	(0.8)	ns	0.4	(1.0)	0.02	(0.9)	ns
Waist Circumference (cm)	62.5	(4.5)	66.6	(7.3)	ns	67.6	(9.0)	65.4	(7.9)	ns
Body fat (%)	20.6	(8.4)	25.9	(8.0)	ns	26.3	(8.8)	23.1	(8.1)	ns
Adolescence[b]										
Weight (kg)	47.6	(5.6)	51.2	(7.6)	ns	54.0	(10.8)	52.4	(10.5)	ns
Height (cm)	159.9	(3.2)	159.9	(5.4)	ns	159.7	(3.2)	159.9	(5.4)	ns
BMI (kg/m^2)	18.6	(2.1)	20.0	(2.7)	ns	21.0	(4.0)	20.3	(3.2)	ns
zBMI (score)	-0.5	(0.4)	-0.8	(0.8)	ns	1.2	(1.2)	-0.6	(0.9)	ns
Waist Circumference (cm)	67.7	(6.8)	72.2	(6.3)	ns	72.0	(8.3)	71.6	(8.2)	ns
Hip Circumference (cm)	76.9	(5.6)	80.5	(5.7)	ns	81.9	(9.7)	81.6	(8.3)	ns
Waist-Hip ratio	0.9	(0.03)	0.9	(0.03)	ns	0.9	(0.0)	0.9	(0.04)	ns
Biceps skinfold (mm)	8.6	(3.6)	9.5	(4.3)	ns	11.8	(6.2)	10.0	(4.8)	ns
Triceps skinfold (mm)	16.9	(3.2)	17.4	(4.8)	ns	18.76	(6.9)	17.6	(6.0)	ns
Subscapular skinfold (mm)	10.4	(5.0)	12.8	(5.8)	ns	15.41	(6.9)	13.9	(5.7)	ns
Triceps-subscapular skinfold ratio	1.8	(0.6)	1.5	(0.4)	ns	1.28	(0.3)	1.3	(0.3)	ns
Body fat (%)	20.3	(3.3)	24.2	(8.0)	ns	25.60	(8.2)	24.8	(7.3)	ns
Change[c] from preadolescence to adolescence										
Change in BMI (kg/m^2)	0.9	(2.0)	1.0	(2.6)	ns	1.09	(1.9)	1.5	(1.7)	ns
Change in Waist Circumference (cm)	5.0	(4.8)	5.6	(6.8)	ns	4.57	(5.1)	5.9	(6.1)	ns
Change in Body Fat (%)	-0.3	(5.9)	-1.7	(5.3)	ns	-0.48	(4.9)	1.9	(5.8)	**0.035**

[a]Preadolescence: baseline phase; [b]Adolescence; follow-up phase. [c]Change: difference between the baseline point (preadolescence) and the endpoint (adolescence).
MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism; BMI: Body Mass Index; zbMI: z-score of BMI. SD: Standard deviation; ns: non-significant Level of statistical significance: p value<0.05

In contrast, in boys with MAOA-H, several measures of adiposity showed lower values in emotional symptoms group than control group (table 53). Mainly, values of BMI (p=0.024), zBMIz (p=0.039 and WC (p=0.007) in preadolescence and BMI (p=0.024), zBMI (p=0.004), WC (p=0.001) and biceps skinfold (p=0.039) in adolescence were lower in emotional symptoms group than in control group.

Respect to 5-HTTLPR, there were no significant differences in anthropometric and body composition variable between 5-HTTLPR SS/SL and 5-HTTLPR LL in girls (table 54). However, girls with 5-HTTLPR SS/SL and emotional symptoms showed higher increase of change in %BF (1.6±5.6%) than control group (-0.7±4.9%) although it was not significant (p=0.052). There were no differences among emotional symptoms group and control in boys (table 55).

4.4.3 Effect of emotional symptoms on change in anthropometric and body composition parameters according to genetic factors

Several models of multiple linear regression models were performed to examine whether emotional symptoms and genetic factors or their interaction (emotional symptoms-x-genetic factor) were associated with the change in BMI, WC and %BF. Any significant effect of genetic factors and/or emotional symptoms on parametric and anthropometric parameters was observed in either boys or girls.

In addition, we did not found significant differences between depressive or types of anxiety symptoms and anthropometric and body composition parameter in stratified analysis by genetic factor (data not shown).

Table 53. Anthropometric and body composition parameters according to MAOA genotype and emotional symptoms in boys

	MAOA-L					MAOA-H				
	Control (n= 11)		Emotional symptoms (n= 18)			Control (n=15)		Emotional symptoms (n=34)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Preadolescence[a]										
Weight (kg)	36.2	(5.2)	38.0	(7.4)	ns	41.8	(10.9)	34.8	(5.5)	**0.031**
Height (cm)	141.8	(5.8)	142.9	(7.2)	ns	145.3	(9.0)	140.2	(6.4)	**0.030**
BMI ((kg/m^2)	17.9	(2.0)	18.5	(2.9)	ns	19.5	(3.2)	17.7	(2.2)	**0.024**
zBMI (score)	-0.2	(0.6)	0.1	(1.0)	ns	0.4	(0.9)	-0.1	(0.7)	**0.039**
Waist Circumference (cm)	64.5	(6.1)	66.3	(6.3)	ns	70.7	(8.4)	64.6	(5.5)	**0.007**
Body fat (%)	19.1	(4.4)	19.4	(5.8)	ns	22.3	(6.3)	18.8	(5.5)	ns
Adolescence[b]										
Weight (kg)	51.9	(7.4)	53.0	(9.4)	ns	57.9	(10.0)	50.1	(8.5)	**0.007**
Height (m)	165.3	7.8	164.3	8.7	ns	165.4	9.9	162.5	9.2	ns
BMI (kg/m^2)	18.9	(2.2)	19.9	(3.5)	ns	20.9	(3.2)	18.9	(2.5)	**0.024**
zBMI (score)	-0.26	(0.6)	-0.05	(1.0)	ns	0.2	(0.7)	-0.2	(0.7)	**0.030**
Waist Circumference (cm)	70.4	(7.1)	72.8	(6.8)	ns	78.6	(7.8)	71.9	(6.6)	**0.004**
Hip Circumference (cm)	76.3	(6.5)	78.4	(8.1)	ns	84.2	(6.3)	77.3	(6.3)	**0.001**
Waist-Hip ratio	0.9	(0.03)	0.9	(0.03)	ns	0.9	(0.05)	0.9	(0.03)	ns
Biceps skinfold (mm)	7.2	(4.4)	9.7	(5.9)	ns	11.7	(5.8)	8.4	(4.6)	**0.039**
Triceps skinfold (mm)	11.7	(7.3)	14.9	(8.3)	ns	16.4	(7.8)	12.5	(6.4)	ns
Subscapular skinfold (mm)	10.7	(7.3)	11.8	(6.2)	ns	14.1	(6.5)	11.2	(6.3)	ns
Triceps-subscapular skinfold ratio	1.1	(0.3)	1.2	(0.03)	ns	1.1	(0.3)	1.1	(0.2)	ns
Body fat (%)	12.3	(5.0)	13.4	(6.8)	ns	16.3	(6.3)	12.9	(5.7)	ns
Change[c] from preadolescence to adolescence										
Change in BMI (kg/m^2)	1.0	(1.0)	1.3	(2.3)	ns	1.3	(1.4)	1.2	(1.4)	ns
Change in Waist Circumference (cm)	5.9	(4.0)	7.1	(5.3)	ns	7.9	(5.0)	7.6	(5.4)	ns
Change in Body Fat (%)	-6.8	(2.9)	-5.2	(6.4)	ns	-5.5	(5.7)	-5.5	(3.8)	ns

[a]Preadolescence: baseline phase; [b]Adolescence; follow-up phase. [c]Change: difference between the baseline point (preadolescence) and the endpoint (adolescence).
MAOA-L: Low-activity MAOA polymorphism; MAOA-H: High-activity MAOA polymorphism; BMI: Body Mass Index; zBMI: z-score of BMI; SD: Standard deviation; ns: non-significant
Level of statistical significance: p value<0.05

Table 54. Anthropometric and body composition parameters according to 5-HTTLPR genotype and emotional symptoms in girls

	5-HTTLPR LL					5-HTTLPR SS/SL				
	Control (n=17)		Emotional symptoms (n= 13)			Control (n=33)		Emotional symptoms (n= 60)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Preadolescence[a]										
Weight (kg)	39.1	(9.4)	41.3	(11.4)	ns	42.5	(10.7)	39.3	(10.1)	ns
Height (cm)	143.7	(10.2)	148.8	(9.6)	ns	145.2	(8.1)	144.7	(8.7)	ns
BMI ((kg/m^2)	19.1	(2.6)	18.4	(3.8)	ns	20.0	(4.1)	18.5	(3.0)	ns
zBMI (score)	0.2	(0.8)	-0.04	(1.0)	ns	0.4	(1.1)	-0.1	(0.9)	ns
Waist Circumference (cm)	65.7	(6.3)	67.2	(10.3)	ns	67.7	(9.7)	64.4	(6.8)	ns
Body fat (%)	24.4	(9.3)	23.8	(7.9)	ns	26.0	(9.3)	22.9	(8.0)	ns
Adolescence[b]										
Weight (kg)	44.3	(4.8)	52.4	(6.7)	ns	54.3	(11.2)	51.9	(10.1)	ns
Height (cm)	158.9	(5.5)	162.9	(6.8)	ns	160.2	(6.9)	159.7	(6.3)	ns
BMI (kg/m^2)	20.3	(3.2)	20.2	(3.6)	ns	21.2	(4.1)	20.2	(3.1)	ns
zBMI (score)	-0.05	(1.0)	-0.06	(1.0)	ns	0.2	(1.2)	-0.1	(0.9)	ns
Waist Circumference (cm)	70.4	(6.3)	73.0	(9.2)	ns	72.3	(8.8)	71.5	(7.8)	ns
Hip Circumference (cm)	78.2	(9.9)	78.8	(10.6)	ns	82.2	(9.4)	81.2	(8.2)	ns
Waist-Hip ratio	0.9	(0.03)	0.9	(0.5)	ns	0.9	(0.04)	0.9	(0.04)	ns
Bicep skinfold (mm)	11.5	(6.8)	10.4	(5.5)	ns	11.7	(5.8)	10.0	(5.1)	ns
Tricep skinfold (mm)	17.7	(6.4)	16.9	(6.9)	ns	19.2	(6.5)	17.9	(5.9)	ns
Subscapular skinfold (mm)	13.3	(5.7)	14.1	(6.8)	ns	15.9	(7.1)	13.8	(5.5)	ns
Tricep-subscapular ratio	1.4	(0.4)	1.3	(0.3)	ns	1.3	(0.4)	1.4	(0.3)	ns
Body fat (%)	25.6	(6.9)	25.0	(8.8)	ns	25.2	(8.4)	24.5	(7.0)	ns
Change[c] from preadolescence to adolescence										
Change in BMI (kg/m^2)	0.8	(2.1)	1.8	(1.3)	ns	1.2	(1.8)	1.6	(1.7)	ns
Change in Waist Circumference (cm)	3.8	(3.4)	6.3	(5.5)	ns	5.0	(5.6)	6.6	(5.9)	ns
Change in Body Fat (%)	0.3	(5.8)	1.6	(4.9)	ns	-0.7	(4.9)	1.6	(5.6)	ns

[a]Preadolescence: baseline phase; [b]Adolescence; follow-up phase. [c]Change: difference between the baseline point (preadolescence) and the endpoint (adolescence).
5-HTTLPR LL: serotonin transporter polymorphism with long alleles; 5-HTTLPR SS/SL: serotonin transporter polymorphism with short alleles or short/long alleles; BMI: Body Mass Index; zBMI: z-score of BMI; SD: Standard deviation; ns: non-significant Level of statistical significance: p-value<0.05.

Table 55. Anthropometric and body composition parameters according to 5-HTTLPR genotypeand presence of any emotional symptoms in boys

	5-HTTLPR LL					5-HTTLPR SS/SL				
	Control (n=6)		Emotional symptoms (n= 10)			Control (n=16)		Emotional symptoms (n= 37)		
	Mean	(SD)	Mean	(SD)	p	Mean	(SD)	Mean	(SD)	p
Preadolescence[a]										
Weight (kg)	42.2	(17.7)	36.2	(4.3)	ns	38.6	(5.3)	36.4	(6.8)	ns
Height (cm)	144.0	(10.7)	141.8	(7.5)	ns	144.4	(7.3)	141.2	(6.9)	ns
BMI ((kg/m^2)	19.6	(4.9)	17.9	(1.9)	ns	18.5	(2.0)	18.2	(2.7)	ns
zBMI (score)	0.3	(1.4)	-0.03	(0.7)	ns	0.15	(0.7)	0.03	(0.9)	ns
Waist Circumference (cm)	69.9	(4.8)	63.8	(4.5)	ns	66.8	(5.3)	66.0	(6.1)	ns
Body fat (%)	21.2	(7.6)	18.1	(4.3)	ns	20.0	(4.8)	19.5	(6.1)	ns
Adolescence[b]										
Weight (kg)	55.6	(13.6)	53.7	(7.3)	ns	55.7	(8.2)	51.1	(9.1)	ns
Height (cm)	165.0	(5.5)	164.9	(10.0)	ns	165.8	(9.8)	163.0	(8.8)	ns
BMI (kg/m^2)	20.3	(4.8)	19.7	(2.5)	ns	20.0	(2.3)	19.3	(3.1)	ns
zBMI (score)	-0.03	(1.1)	-0.08	(0.8)	ns	0.02	(0.7)	-0.17	(0.9)	ns
Waist Circumference (cm)	73.0	(11.7)	72.5	(5.8)	ns	76.1	(7.2)	72.6	(7.2)	ns
Hip Circumference (cm)	78.6	(10.1)	79.1	(6.9)	ns	1.6	(0.1)	1.6	(0.1)	ns
Waist-Hip ratio	0.92	(0.04)	0.91	(0.03)	ns	0.92	(0.05)	0.93	(0.1)	ns
Bicep skinfold (mm)	7.8	(5.4)	8.5	(4.6)	ns	10.2	(4.4)	9.4	(5.3)	ns
Tricep skinfold (mm)	12.1	(9.0)	14.2	(6.3)	ns	14.1	(6.4)	13.6	(7.6)	ns
Subscapular skinfold (mm)	9.7	(5.1)	13.1	(6.5)	ns	13.4	(7.1)	11.2	(6.4)	ns
Tricep-subscapular ratio	1.18	(0.4)	1.12	(0.2)	ns	1.09	(0.2)	1.2	(0.3)	ns
Body fat (%)	12.7	(7.5)	13.2	(5.3)	ns	14.6	(5.0)	13.39	(5.1)	ns
Change[c] from preadolescence to adolescence										
Change in BMI (kg/m^2)	0.7	(1.5)	1.8	(1.5)	ns	1.52	(1.1)	1.15	(1.8)	ns
Change in Waist Circumference (cm)	3.9	(3.6)	8.7	(6.2)	ns	8.6	(4.5)	6.9	(5.2)	ns
Change in Body Fat (%)	-7.8	(7.5)	-4.9	(4.4)	ns	-5.5	(3.5)	-5.6	(5.1)	ns

[a]Preadolescence: baseline phase; [b]Adolescence; follow-up phase. [c]Change: difference between the baseline point (preadolescence) and the endpoint (adolescence).
5-HTTLPR LL: serotonin transporter polymorphism with long alleles; 5-HTTLPR SS/SL: serotonin transporter polymorphism with short alleles or short/long alleles; BMI: Body Mass Index; zBMI: z-score of BMI; SD: Standard deviation; ns: non-significant
Level of statistical significance: p-value<0.05

5. NARRATIVE REVIEW: THE ROLE OF EMOTION REGULATION IN THE PREVENTION AND TREATMENT OF CHILDHOOD OBESITY

Figure 15 depicts the conceptual framework developed to suggest the role of emotion regulation in the prevention and treatment of childhood obesity. Following, the conceptual framework model is briefly explained. The extended information about this conceptual model are included in the manuscript "The role of emotion regulation in childhood obesity: Implications for prevention and treatment", which has been accepted recently.

Our model was develop understanding that stress and negative emotions during childhood pose a major threat to public health, since they have been related not only to psychological disease but also to physiological disturbances such as obesity. Emotion regulation is the process used to cope with negative emotions and it start to develop since early ages. Emotion regulation is a growing field within psychology and holds a central role in all the psychological areas including health, development, clinical, social and personality as well as in a variety of other disciplines.

Only very few observational studies have assessed the relationship between emotion regulation and obesity in children. Therefore, related factors such as executive function and self-control, which include similar aspects of emotion regulation, have also been included in the overview shown in Table 56. Better emotion regulation-related skills were associated with a healthier diet (more fruit/vegetables and less snack food, although not all studies reported significant findings), higher physical activity and, in some cases, a healthier weight status

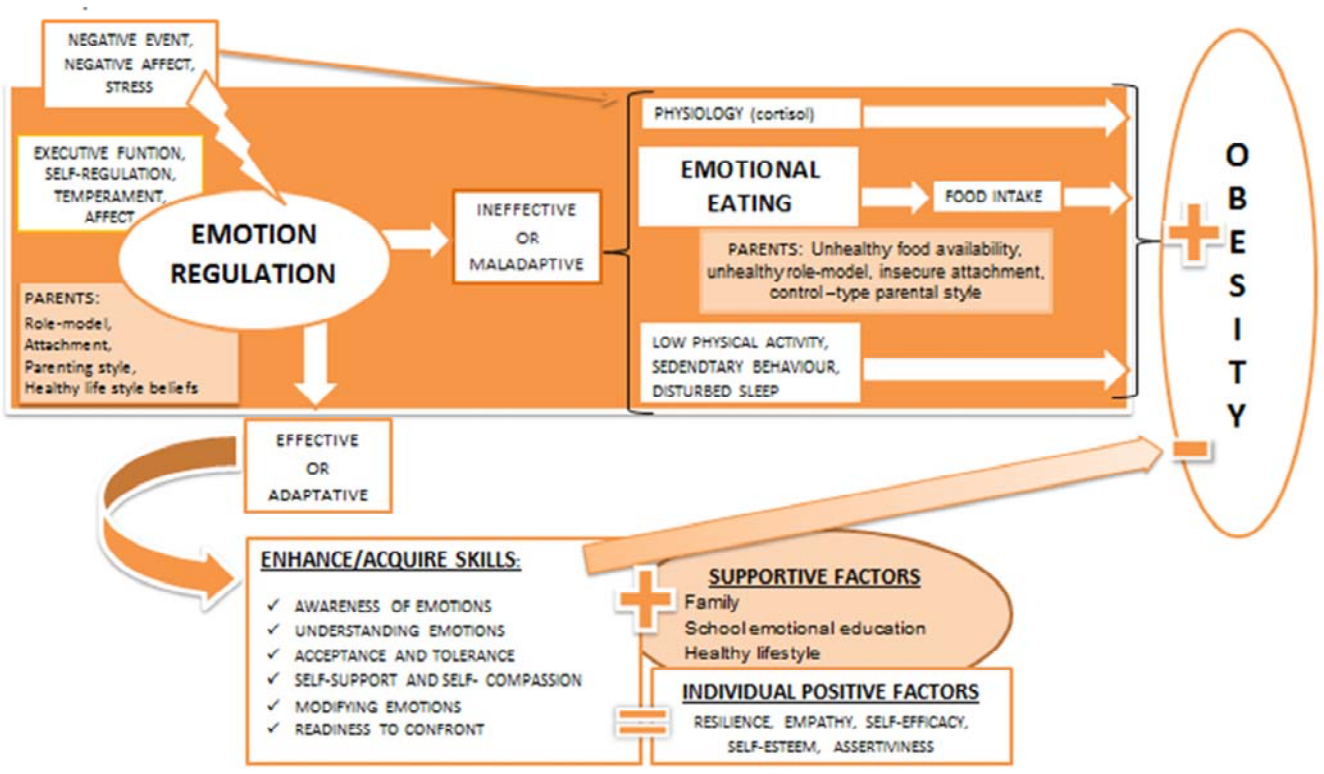
Our model posits that emotion regulation is a fundamental link between childhood stress and obesity. This review enhances knowledge of the mechanistic pathways between emotion regulation and eating behaviour and obesity by condensing existing studies to a visual research framework. Stress, combined with ineffective emotion regulation, could already be present in childhood and could cause abnormal cortisol patterns, emotional eating, decreased physical

activity, increased sedentary behaviour and the onset of sleep problems. A healthy lifestyle, such as physical activity and adequate sleep, could show benefits on emotion regulation and in developing adaptive emotion regulation strategies. Parents also influence the development of emotion regulation and obesity in children, as role models and through their parenting style and parental feeding practice.

Skills to develop an effective emotional regulation are: awareness of emotions (i.e. the ability to identify and label emotions), understanding emotions (i.e. the ability to identify the causes and maintaining factors of emotions); acceptance and tolerance (i.e. the ability to accept and tolerate negative emotions when necessary); self-support and self-compassion (i.e. the ability to provide effective self-support and self-compassion in distressing situations by self-soothing, self-encouragement and active self-coaching); modifying emotions (i.e. the ability to modify emotions in an adaptive way, which includes self-efficacy) and readiness to confront (i.e. the ability to address situations likely to cause negative emotions). Based on the literature addressing the adult population, possible successful therapies include mindfulness-based stress reduction therapy, acceptance and commitment therapy or self-compassion therapy. Furthermore, protective factors could be stimulated, such as resilience, assertiveness, empathy, self-efficacy and self-esteem.

Therefore, effective emotion regulation skills could decrease obesity-related unhealthy behaviour and enhance protective factors, which boost mental and physical health. As a result, effective emotion regulation could contribute to the prevention and treatment of childhood obesity. In children, some observational studies but few interventional studies on this relationship have been published. We proposed that psycho-educative intervention in emotion regulation training could increase the efficacy of prevention and treatment programs of obesity. Therefore, encouraging adaptive emotion regulation could be an effective new approach, along with nutritional and physical activity intervention, in the fight against and the treatment of childhood obesity.

Figure 15. Conceptual framework model of the role of emotion regulation in the prevention and treatment of childhood obesity



UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Table 56. Emotion regulation or related terms as predictors of childhood weight gain/overweight/obesity: Observational studies

Author, year	Country	Type of study	Sample (n) /gender (% girls)	Age or grade at baseline	Emotion regulation predictor: Measurement tool	Outcome: Adiposity-related measures or obesity-related behaviour measures	Results
Isasi *et al.*, (2013)	USA	Cross-sectional	N=612	12 years	Emotion regulation: soothability, sadness management, anger management, assessed by three scales Self-efficacy for healthy eating Self-efficacy for physical activity Depressive symptoms	Food intake: YAQ Physical activity: Youth Risk Behaviour Survey Time spent in sedentary behaviours Anthropometry: measured BMI	Stronger emotion regulation associated with higher intake of fruit/vegetables and physical activity; association mediated by self-efficacy. No association with BMI
Graziano *et al.*, (2010)	USA	Longitudinal 3.5-year follow-up	N=57 (43.8%)	2 years	Emotion regulation: laboratory task: videotapes of frustration tasks (Prize in the Box and High Chair). Inhibitory control/reward sensitivity: delay of gratification task, assessment of the overall total time touching gift Sustained attention: children were instructed to watch a 5-min segment of the videotape 'Spot', overall duration of attention was measured Child behaviour problems: child behaviour checklist (CBCL)	Measured: BMI	Maladaptive emotion regulation and inhibitory control/reward sensitivity predicted more extreme weight problems at 5.5 years
Graziano *et al.*, (2013)	USA	Longitudinal 7-year follow-up	N=195 (58.4%)	2 years	Laboratory task: as above, except that CBCL was replaced by the Toddler Behaviour Assessment Questionnaire Statistical method to reduce data: Factor 1: Self-regulation (including emotion regulation) Factor 2: Temperament; Pleasure	Measured: BMI	Toddlers with better self-regulation skills at age 2 years had lower BMI z-scores at 10 years old and were less likely to show an increase in BMI z-scores from 4 to 10 years

Results

Author, year	Country	Type of study	Sample (n) /gender (% girls)	Age or grade at baseline	Emotion regulation predictor: Measurement tool	Outcome: Adiposity-related measures or obesity-related behaviour measures	Results
Isasi and Wills (2011)	USA	Cross-sectional	N=1771	12 years	Self-regulation measures: Multiple indicators of effortful: planfulness, problem solving, soothability, self-reinforcement Multiple indicators of dysregulation: impulsivity, impatience, immediate gratification, anger ability, distractibility, self-criticism Self-efficacy for healthy eating Self-efficacy for physical activity	Dietary intake: short food frequency questionnaire Physical activity and sedentary behaviour: Youth Risk Behaviour Survey Anthropometry: measured BMI	Effortful control had a positive indirect effect on fruit and vegetable intake, mediated by self-efficacy Effortful control had a positive indirect effect on physical activity, mediated by self-efficacy Dysregulation had direct effects on higher intake of junk food/snacks and time spent in sedentary behaviours
Wills *et al.*, (2007)	USA	Cross-sectional	N=539	14.6 years (mean)	Self-control measures: Good self-control: soothability, planfulness, problem solving, cognitive effort, future time perspective, self-reinforcement Poor self-control: distractibility, impulsiveness, anger ability, tension maintenance, impatience, present time orientation, negative self-management	Dietary intake: Food Frequency Questionnaire Physical activity and sedentary behaviour: Youth Risk Behaviour Survey	Good self-control was related to more fruit and vegetable intake, more participation in sports, and less sedentary behaviour Poor self-control was related to more saturated fat intake and less vigorous exercise
Riggs et al. (2010)	USA	Longitudinal 4-month follow-up	N=184 (52%)	9.3 years (mean)	Executive cognitive function: BRIEF-SR (including a subscale of emotion control; inhibitory control; working memory; organization of materials)	Dietary intake: Food Frequency Questionnaire Physical activity: Physical Activity Questionnaire for Older Children	Baseline executive cognitive function was associated with more fruit/vegetable intake and physical activity four months later; no association with snack food intake.

Author, year	Country	Type of study	Sample (n) /gender (% girls)	Age or grade at baseline	Emotion regulation predictor: Measurement tool	Outcome: Adiposity-related measures or obesity-related behaviour measures	Results
Riggs *et al.*, (2012)	USA	Cross-sectional	N=1587	9.3 years	Executive cognitive function: BRIEF-SR (including a subscale of emotion control; inhibitory control; working memory; organization of materials)	Dietary intake: Food Frequency Questionnaire Physical activity: Physical Activity Questionnaire for Older Children	Executive cognitive function was negatively associated with high-calorie snack food intake and sedentary behaviour Executive cognitive function was positively associated with fruit and vegetable intake and physical activity
Riggs et al. (2010)	USA	Cross-sectional	N=107	9.4 years	Executive cognitive function: BRIEF-SR (including a subscale of emotional control; inhibitory control; working memory; organization of materials)	Dietary intake: Food Frequency Questionnaire Physical activity: Physical Activity Questionnaire for Older Children	Executive cognitive function was negatively related to snack food intake, but not significantly related to fruit and vegetable intake
Hughes *et al.*,(2015)	USA	Cross-sectional	n= 187	4.7 years	Eating self-regulation: Laboratory task: eating in the absence of hunger Questionnaire: CEBQ (satiety responsiveness and food responsiveness) Non-eating self-regulation (self-regulation, executive function, emotion regulation): Laboratory task: Tapping task, Flexible item selection task, Delay of gratification Gift delay task Questionnaire: CBQ (effortful control)	Anthropometry: measured BMI	Eating self-regulation was associated with BMI, but not other types of self-regulation.

CDC: Centers for Disease Control and Prevention; YAQ: Youth Adolescent Questionnaire; CBCL: Child Behaviour Checklist; BRIEF-SF: Behavioural Rating Inventory of Executive Function–Self-Report Version; CEBQ: Children's eating behaviour questionnaire; CBQ: Children's behaviour questionnaire.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Discussion

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

DISCUSSION

1. DESIGN, PARTICIPANTS AND METHODS

The prospective design in a non-clinical population enabled us to use a sample of schoolchildren at risk of emotional psychopathology and a group of control subjects. Also, using a three-year follow-up period, we were able to assess the effect of the psychopathology on nutritional status from preadolescence to adolescence.

Our study was conducted on a representative sample of schoolchildren from town's state schools and state subsidized private school of Reus, who were followed during three years. However, the sample rate diminishes drastically in follow-up phase. Therefore, due to the small sample size in the follow-up, the interpretation of the results should be prudent, particularly with regard to the boys.

We recollected a huge quantity of socio-demographic, psychological, anthropometric and body composition parameters, food consumption, physical activity and genetic variables, what contributed to be capable to execute our objectives.

The psychopathology assessment was done by screening test validated and adapted to our population and wide use in epidemiological studies. Also, we used a diagnostic interview to confirm the presence of the disorder. Other strength of this study is that the psychopathological information was provided by the children.

The dietary intake assessment was done by a food frequency questionnaire validated and adapted to our population (Trinidad-Rodríguez *et al.*, 2008). Also this is a semi quantitatively questionnaire which enable us to estimate the energy and nutrient intake. Moreover, we performed dietary pattern using principal component analysis.

Principal component analysis is a complex statistical technic that take into account the whole food consumption and their interaction which enables us to identify the usual diet that adolescents follow (Moeller *et al.*, 2007). Therefore, we were able to study the effect of emotional symptoms on whole diet instead of with isolated food items.

The anthropometric variables were measured by qualified personnel using a standardized methodology. The direct determination of weight and height gives our results greater precision and validity (Rhew *et al.*, 2008; Incledon *et al.*, 2011). Furthermore, most of the studies in the literature only consider BMI, yet this index does not provide scope for analysis of %BF or its distribution. The use of other measures that assess %BF, such as BIA, and abdominal fat distribution, such as WC, is therefore necessary. Both methods are simple, economical, fast and feasible at the population level. By contrast, other more sophisticated methods such as computed tomography or dual energy X-ray absorptiometry are more costly, more time-consuming and more difficult to implement.

2. MAIN CHARACTERISTICS OF PARTICIPANTS IN BASELINE AND FOLLOW-UP PHASE

In baseline phase, our results showed an 11.4% of depressive symptoms and a 46.7% of anxiety symptoms. The frequency of depressive symptoms is similar to other studies in adolescent population while the percentage of anxiety symptoms is slightly high (Merikangas *et al.*, 2010b; Coughlan *et al.*, 2014). This slight difference may be by screened methods used and that we assessed the symptoms instead of diagnosis which prevalence is less than symptoms. Also, we found that girls in baseline phase showed more anxiety symptoms than boys, like other authors showed (Romero *et al.*, 2010; Conley *et al.*, 2012; Abbo *et al.*, 2013; Coughlan *et al.*, 2014), although these differences were not observed in depressive symptoms, as not found in an epidemiological study in the same city 20 years ago (Canals *et al.*, 2002). However, Merikangas et al. (2010) found gender

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

differences among depressive disorders but not in anxiety disorders in children. These differences with the literature may be by the age of our participants since they are in onset of adolescence when girls start to sexual maturation. These initial body changes of puberty could be accompanied with more anxiety feeling in girls than in boys.

The frequency of overweight and obesity in our baseline sample was 31% which are similar to Spanish (Moreno *et al.*, 2005; Sánchez-Cruz *et al.*, 2013; Government of Catalonia Ministry of Health, 2014) and European studies (Brug *et al.*, 2012; Ahrens *et al.*, 2014) and is set in the upper range of worldwide prevalence (9-36%) (Lobstein and Frelut, 2003; Branca *et al.*, 2007; Lobstein and Millstone, 2007; Musa *et al.*, 2012; Kelishadi *et al.*, 2014). Some studies showed gender differences among prevalence of overweight and obesity (Serra Majem *et al.*, 2003; Ahrens *et al.*, 2014). However, we did not found it. It is likely that the gender differences in prevalence rates could vary across the lifespan, i.e girls aged 4-6 years showed more overweight and obesity and decreasing with age, whereas boys showed 4-6 aged showed less rate prevalence and it increase with age (Larrañaga *et al.*, 2007).

The percentage of overweight/obesity in adolescence is slightly lower than in preadolescence, although the percentage of overweight or obesity in girls were only gently lower than other Spanish study (Moreno *et al.*, 2005). This fact may be explained by several reasons. Firstly, the follow-up of the study coincided with first stages of pubertal development of the participants, the pubertal growth spurt which set at the age of 9 in girls and 11 in boys (González and Aguilar, 2012). The pubertal growth spurt results in a rapid growth along with by a reduction of body fat and increase free fat mass. Thus, those preadolescents who seem to have a gentle excess weight; they may be reducing their weight in adolescence. Secondly, our study ran into an intervention study to prevent obesity, which was conducted at the same period of time and on children at similar ages. As a consequence, the percentage of excess weight in our population may have diminished.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Referent to food consumption, our sample showed similar characteristics to adolescent population in Spanish and European studies (Aranceta *et al.*, 2003a; Diethelm *et al.*, 2012; Lynch *et al.*, 2014; Moreno *et al.*, 2014; Papadaki and Mavrikaki, 2015). Consistent with other studies, our sample of adolescents did not reach recommendation of vegetables and fruits serving per day. Several studies showed that adolescent consume fruit less than once per day (Diethelm *et al.*, 2012; Lynch *et al.*, 2014; Moreno *et al.*, 2014). Iannoti & Wang (2014) in United Stated found that only 34% of adolescents consume more than one serving per day. Also, in Europe, half of Greek adolescents consumed a second piece of fruit and nearly a 15% of adolescents consumed a second serving per day of vegetables (Papadaki and Mavrikaki, 2015). Also, consistent with literature (Diethelm *et al.*, 2012; Moreno *et al.*, 2014) the consumption of dairy products was also low to recommendation among our sample. In addition, the excessive consumption of snacks and food rich on sweet and fatty food was usual among adolescent population (Diethelm *et al.*, 2012). At first sight our results showed the consumption of sweet, pre-cooked meal, savoury snacks seem not to be excessively high, however the consumption of baked good and chocolates were almost six day a week. Indeed, whether we take into account all these unhealthy food together, the adolescents would consume unhealthy food at least twice a day.

Moreover, the dietary pattern identified was consistent with the usual dietary patterns in general population described by other studies (Newby and Tucker, 2004) and in children or adolescent population (Aranceta *et al.*, 2003a; Cutler *et al.*, 2011). We defined three well-known dietary patterns: pattern based on vegetables, fruit, beans and fish and a dietary pattern based on starchy and meat food called western, as other studies in our country (Sánchez-Villegas *et al.*, 2003; Serra-Majem *et al.*, 2009), and also we found a dietary pattern rich on sweet and fatty food which is usual pattern in adolescents population (Santaliestra-Pasías *et al.*, 2014a). However, other authors with large

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

samples showed more dietary pattern such as starchy food pattern (Cutler *et al.*, 2011), fast food pattern (Cutler *et al.*, 2011) or drinking pattern (Aranceta *et al.*, 2003a), pattern with typical food of breakfast (Santaliestra-Pasías *et al.*, 2014a). In addition, our results showed that although we live in a Mediterranean region, only a 10% of adolescents showed high levels of Mediterranean diet adherence in comparison to a 48% showed a low adherence. Similarity, in recent study by Tognon et al. (2014) with the participants of eight European Countries, the authors found that Spanish school-aged girls showed the lowest prevalence of Mediterranean diet adherence. However, in our result the percentage of Mediterranean diet adherence was considerably lower taking into account the Mediterranean diet prevalence found in the study by Serra-Majem et al. (2004) or a recent study conducted with by Spanish school–age children (Arriscado *et al.*, 2014). This may be because our study has a population with a considerable number of subjects at risk of emotional disorders.

The energy intake was similar to other studies has been reported (Neumark-Sztainer *et al.*, 2004; Serra-Majem *et al.*, 2006; Diethelm *et al.*, 2014). In this vein, consistently with literature (Serra-Majem *et al.*, 2006; Diethelm *et al.*, 2014; Moreno *et al.*, 2014), the percentage of protein seems to be adequate, while the percentage of carbohydrate is low to recommendation and percentage of fat are higher than recommendations (IOM, 2003). The high fat consumption particularity of monounsaturated could be due to the intake of oil was estimated by standard amount to all adolescents due to the difficult and complexity to measure the oil (Trinidad-Rodriguez *et al.,* 2008).

For the micronutrients the intake levels were mostly within an acceptable range, with the exceptions of calcium, iron, magnesium, vitamin D and folic acid intake in boys and/or girls. It is suggested that the adolescents between 14 -17 showed highest nutritional risk (Serra-Majem *et al.*, 2006). Some studies showed that nutrients more compromised in the food consumption of adolescents are vitamin D,

folates, calcium, iron, magnesium, vitamin A as well as vitamin C, E, B6 (Serra-Majem *et al*., 2003; Neumark-Sztainer *et al*., 2004; Aranceta *et al*., 2006). Whereas a large-cohort study across Europe is consistent with low adequacy of below two thirds of recommendations in vitamin D and folates and in calcium only in older girls but they did not observe iron and magnesium intakes were below to recommendation (Diethelm *et al.*, 2014).

In this vein, these vitamin and minerals are essential to correct growth and development. Calcium, magnesium and vitamin D are essential to the mineralization of the skeleton. Consequently, nutrient deficiency can affect the formation of optimum bone mass or may even accelerate bone loss at a crucial moment during bone growth (Serra-Majem *et al.*, 2006). In addition, a risk of iron intake inadequacy, with a larger percentage of the population at risk of iron deficiency than is reported for the general population (Price *et al.*, 2012). Iron deficiency has been associated with the risk of anaemia, reduced immune response and cognitive impairment, among other problems, because iron is involved in multiple metabolic functions (Eden and Sandoval, 2012). Likewise, deficient intake of other micronutrients such as vitamins A, C, B6, B2 and B3, folic acid and magnesium, among others, could be related to poorer mental health and lead to negative repercussions in the medium and long term (Davison and Kaplan, 2012).

In relation to physical activity and sedentary activity, our results that girls and boys showed a mid-low level of physical activity. Also, girls presented lower physical activity and spend more time in sedentary activities during their free time than boys which is consistent with data in recent studies which concluded that girls were less active and more sedentary than boys (Quiles-Marcos *et al.*, 2011; Al-Hazzaa *et al.*, 2014; Leech *et al.*, 2014).

Furthermore, recently, some authors showed that healthy dietary habits are known to be mostly associated with physical activity (Aranceta *et*

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

al., 2003a; Lissner *et al.*, 2012; Santaliestra-Pasías *et al.*, 2014a, 2014b). In this vein, although we did not show that physical activity scores were associated with Mediterranean diet adherence level, we found that boys who engage in more physical activity adhere less to sweet and fatty dietary patterns (in multivariate analysis). Likewise, Santaliestra-Pasías et al. (2014a) showed that boys who spent more time watching television, playing computer or video games and using internet had higher adherence to the snaking pattern and lower adherence to healthy pattern.

3. EFFECT OF EMOTIONAL SYMPTOMS ON DIETARY INTAKE AND PHYSICAL ACTIVITY

Our three-year follow-up study demonstrates that emotional symptoms during early adolescence lead to unhealthy lifestyle behaviors in terms of dietary patterns and sedentary behavior, and the relationship is different between genders. While girls with emotional symptoms during early adolescence deviate from the Mediterranean diet, they present a high adherence to unhealthy dietary patterns that are rich in sweet and fat foods, and low levels of physical activity; no association was observed in boys. At the same time, high SES was inversely related with sweet and fatty food pattern as well as low Mediterranean diet adherence.

Our results showed that although emotional symptoms did not increase energy intake, girls suffering from emotional symptoms presented a high consumption of sweet dairy desserts, sweets and a tendency to high consumption of baked goods and chocolates, pre-cooked meals, savory snacks and soft drinks. In contrast, their consumption of vegetables, fruit, beans, fish and seafood tended to be low. Their diet therefore was deviated from the Mediterranean diet and they acquired a dietary pattern rich in sweet and fat food. Indeed, almost 40% of girls with emotional symptoms presented a high adherence to a dietary

pattern of sweet and fatty foods, and the relation remained significant when we adjusted for potential confounder factors.

Several authors have suggested that there is a significant association between unhealthy dietary patterns and poor mental health in children and adolescents (van Kooten *et al.*, 2007; Jacka *et al.*, 2010, 2013; Michels *et al.*, 2012b, 2015a; El Ansari *et al.*, 2014). In this vein, our findings are consistent with the literature about stress shifting food choices from lower fat to higher fat and sweet food (Zellner et al., 2006). The association between higher consumption of sweet foods and higher perceived stress levels was also reported by several authors (Oliver *et al.*, 2000; Jenkins *et al.*, 2005; Kandiah *et al.*, 2006; Michels *et al.*, 2012b; Jääskeläinen *et al.*, 2014). Meanwhile, dishes or meal foods such as fruit and vegetables, meat, and fish were reported to be eaten less during stressful periods (Cartwright *et al.*, 2003). Other study also showed that the diets of European girls with more stress were of poor quality, limited variety and balanced (De Vriendt et al., 2012). Recently, a longitudinal study conducted on children at school age showed that certain stress predict higher consumption of sweet food (Michels *et al.*, 2015a).

In relation to emotional symptoms, a recent population-based study of young university students in the United Kingdom showed that depressive symptom scores were associated with a high consumption of unhealthy food (sweets, cookies, snacks, fast food) and a low consumption of healthy food (fresh fruits, salad, cooked vegetables) (El Ansari *et al.*, 2014), and that depression was associated with poor diet quality in Australian adolescents (Jacka *et al.*, 2010). Although some authors found this association also in boys, we only observed it in girls. Elsewhere, in other school-based adolescent samples, the consumption of snacks, sweets and fast food was associated with stress or behavior disorder but not with emotional symptoms (van Kooten *et al.*, 2007; Oddy *et al.*, 2009). It is possible that the emotional symptoms were related to eating disorders, which occur more often during adolescence

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

(Sancho *et al.*, 2007; Stephen *et al.*, 2014), and could lead a decline in food consumption (Aparicio, Canals, Pérez and Arija, 2014; Aparicio-Llopis, Canals and Arija, 2014) and above all showed a low consumption of sweet and fat food (Allen et al., 2012; Larson, Neumark-Sztainer and Story, 2009). As a result, the relation between emotional symptoms and higher consumption of palatable food may be not found. In this vein, our results showed that girls with emotional symptoms scored higher in the eating disorder symptoms test. Despite this, the relationship between emotional symptoms and sweet and fatty dietary patterns remained significant.

In addition, when we studied the psychopathological characteristics of the sample according to their Mediterranean diet adherence level, the results showed that girls with low Mediterranean diet adherence reported high mean scores of depression and eating disorder symptoms. Regardless of gender, Fulkerson et al. (2004) found that poor dietary quality was cross-sectionally associated with depression symptoms in accordance with our data from multivariate analyses. As in Jacka et al. (2011), our results did not show that baseline depression was a predictive factor for healthy diet quality during follow-up. The relationship found leads us to believe and support our previous results in which girls with depressive symptoms tend more towards a greater consumption of comfort foods rather than healthy foods, such as sweet snack foods, as a way to reduce feelings of sadness (Wurtman and Wurtman, 1995; Mooreville *et al.*, 2014).

It was therefore hypothesized that emotions would influence appetite in college students and that it would stimulate an increased preference for sweet food over other comfort food (Macht, 2008). It is proposed that eating may act as an emotional relief and a form of maladaptive coping. People eat "comfort food", typically with high fat and carbohydrate content, such as sweets, in an attempt to reduce the stress (Dallman *et al.*, 2003, 2005; Tryon *et al.*, 2013). This hypothesis was supported by our data since the presence of emotional symptoms has been related to

higher consumption of a dietary pattern rich in high-fat and sweet food. The biological mechanism is explained by the fact that in non-human animal models these palatable foods have a calming effect on the hypothalamic-pituitary-adrenal axis stress response (Adam and Epel, 2007; Torres and Nowson, 2007). Sugar and fat target the brain in a similar manner to opiates and are often sought during times of stress (Oliver *et al.*, 2000). These highly palatable foods with low nutrient density can provide short term pleasure and relief from discomfort (Dallman et al., 2005). Over time, this pattern may develop into a routine coping mechanism for dealing with emotional symptoms (Singh, 2014) and become a habitual dietary pattern in the future. Such increases of sweet and fatty food consumption would be expected to lead to excessive weight and fat gain overtime (Hooper *et al.*, 2012).

Although relation between depression and low Mediterranean diet adherence was independently of gender, we found no differences related to dietary patterns between those with or without emotional symptoms in adolescent boys. This provides evidence that the tendency for increased eating of high-fat, energy dense and palatable food was more pronounced among girls than boys. Indeed, the existing literature suggests that stress-vulnerable women tend to consume more sweet food and fast food (Epel *et al.*, 2001; Adam and Epel, 2007; Mikolajczyk *et al.*, 2009). Women are more likely to report the effect of disturbed mood on impaired control of overeating sweets (Kampov-Polevoy *et al.*, 2006). Other authors suggested that girls are more sensitive to palatable food and may be more susceptible to overeating. For instance, adult or laboratory studies observed that women may be more likely than men to increase food consumption, and particularly of sweet or fatty foods at times of negative emotion (Wansink *et al.*, 2003; Yannakoulia *et al.*, 2008a), while men tend to choose meal-related food at times of negative emotions (Wansink *et al.*, 2003) and increase food consumption at times of positive emotions (Dubé *et al.*, 2005). This may partly explain the differences in food preferences between the presence of emotional symptoms in girls and boys. The

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

differences between genders and the mechanisms involved are not sufficiently clear and more research is needed. In addition, our results should be interpreted with caution due to the small size of our sample.

Moreover, emotional symptoms could lead to increased intakes of sweet and fatty food, but some evidence suggests that eating behavior can affect mood states (Lai *et al.*, 2014). There are several potential biological pathways by which diet quality may be related to mental health (O'Neil *et al.*, 2014). While mood and emotional distress can induce a preference for sweet carbohydrate or fat-rich snacks food in order to enhance mood, these dietary pattern may increase the risk of depression (Sánchez-Villegas *et al.*, 2012) since dietary pattern rich in saturated fat may induce oxidative damage and inflammation and, consequently, interfere with neurotransmitter metabolism and reduce expression of brain-derived neurotropic factor (BDNF) associated with emotional disorders (Anisman, 2009). Furthermore, habitual dietary patterns with lower consumption of fish, olive oil, dry food and vegetables food (i.e. food rich in omega 3, oleic and vitamins and minerals) are also associated with an increase of mental disorders and it is suggested that Mediterranean diet could involve a preventive effect on risk of depression (Sánchez-Villegas *et al.*, 2013; Lai *et al.*, 2014). In specific terms, isolated nutrients of the Mediterranean diet, such as B vitamins, folate, and omega- 3 fatty acids are known to have preventive effects for depression in adults. For example, folate is required for the synthesis of methionine which is a precursor of S-adenosylmethionine, and acts in methylation reactions such as those involving neurotransmitters with antidepressant characteristics (Esposito *et al.*, 2004; Karatzi *et al.*, 2008; Salas-Salvadó *et al.*, 2008; Mena *et al.*, 2009; Sánchez-Villegas *et al.*, 2009). Indeed, Mediterranean diet adherence is also related to reductions in vascular, inflammatory, and metabolic processes related to patients with depression.

Several of the variables studied may be bidirectional related because of that we performed three mediational models to explain whether Mediterranean diet adherence or depressive symptoms were mediators of overweight/obesity and diet quality respectively. In this regard, despite depressive symptoms being related to Mediterranean diet adherence, this variable was not a mediator for high BMI. Also, other studies showed physical activity was a mediator factor between depression and BMI rather than dietary quality in adult population (Beydoun and Wang, 2010). Moreover, consistent with non-association between BMI factor and Mediterranean, other authors also did no observed association (Farajian *et al.*, 2011; Jennings *et al.*, 2011). In this regard, in adults, Rossi et al. (2008) showed that being classified as underweight, normal-weight, overweight and obesity had no significant effect on Mediterranean diet adherence. Therefore, due to adolescence is a critical period could be that adolescents just began to conduct eating behavior changes such as deviate healthy diet and acquire unhealthy dietary pattern which may lead to anthropometric effects in the long term, such as higher BMI (Schroder *et al.*, 2004; Martínez-González *et al.*, 2012).

We also found that high SES was a protective factor for presenting low Mediterranean diet adherence and overweight/obesity as well as high consumption sweet and fatty food. Hence, as in other studies it seems that families with higher SES informed better diet patterns than those with lower SES (Bonaccio *et al.*, 2012; Arriscado *et al.*, 2014; Grosso *et al.*, 2014). These results suggest that the diet pattern and high BMI of children depends on socioeconomic and educational level of their family and their parent's health awareness, as argued by Sotos-Prieto et al. (2014). This result is open to multiple interpretations. First, families with high incomes usually have high education levels which may be related to good knowledge of healthy dietary habits, and they may be more likely to follow healthy patterns. Second, their better economic opportunities may lead them to consume higher quality healthy products.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

By our results, we showed that emotional symptoms also influence lifestyles, reducing physical activity, in a manner that is especially significant in adolescent girls. Girls with depression or anxiety may develop apathy and have less motivation to do exercise. This could be involved in a more sedentary lifestyle. Indeed, the literature observed that emotional symptoms are linked to other aspects of obesity-related lifestyles, such as sedentary behaviour and limited physical activity. Children and adolescents with stress or emotional symptoms may perform less physical activity (Michels *et al.*, 2015a) and are likely to spend more time doing sedentary activities such video games, internet and watching television (Anton *et al.*, 2006; Holmes, Ekkekakis and Eisenmann, 2010; Reeves et al., 2008).

4. EFFECT OF EMOTIONAL SYMPTOMS ON ANTHROPOMETRIC AND BODY COMPOSITION PARAMETERS

In our cross-sectional view at baseline phase we did not show a strong association among depression and anxiety and weight status as others cross-sectional studies (Ali *et al.*, 2010). Although some studies found that obese children and adolescents suffer from anxiety and depression (Braet *et al.*, 1997; Needham and Crosnoe, 2005; Van Vlierberghe *et al.*, 2009; Goldfield *et al.*, 2010; Esposito *et al.*, 2014), we only observed that girls, who showed a % BF in overweight/obesity range, obtained higher scores of social phobia symptoms. Nevertheless, this is similar to others studies (Drukker *et al.*, 2009; Pirgon *et al.*, 2015), since social phobia, which characterized by distress in front of social situation by fear to being embarrassed, could be triggered by the negative relation with their peers. Most of overweight or obese children and adolescent often are stigmatised by their excess weight and are target of jokes and bulling by their peers (Lawler and Nixon, 2011; Barlösius and Philipps, 2015; Salwen *et al.*, 2015). Therefore, they could reject to go out home or avoid being alone in public situation. At

the same time, this may result in depressive symptoms. Goldfield et al. (2010) found that obese youth not only showed reported greater depressive symptoms but also anhedonia, negative self-esteem and body dissatisfaction. In this vein, like consistent literature (Erickson *et al.*, 2000; Pesa *et al.*, 2000; Ivarsson *et al.*, 2006; Goldfield *et al.*, 2010; Makinen *et al.*, 2012), we also found that overweight and obese girls showed low self-esteem and body dissatisfaction. This fact is triggered and could make worse by thinness ideal like the beauty stereotype in our society (Dragone and Savorelli, 2012).

Despite we did not observe a strong cross-sectional association between emotional symptoms and overweight or obesity, in a longitudinal view our results showed a certain effect on anthropometric and body composition variable over time. Recently, Jernigan et al. (2015) in a longitudinal study conducted in 756 adolescents showed that emotional symptoms increase BMI over two years. In this vein, we also observed a relationship between anxiety and depression in preadolescence and increased weight, adiposity and distribution of abdominal fat during adolescence. This relationship was observed in both sexes, although some differences were found according to the type and severity of psychopathology and relations were found predominantly in males.

We found that depressive symptoms led to increase BMI, WC and %BF in males only. Indeed, although some univariate associations were not observed, multiple regression adjusted for specific risk factors of overweight or obesity enabled us to identify the independent effects of depression, among others. The relationship between depression and increased adiposity is corroborated in individuals diagnosed with dysthymia but not in those diagnosed with major depression episode. This could be explained by the fact that dysthymia is a chronic disorder whose manifestations affect lifestyle and have long-term health effects. By contrast, a major depressive episode is a much more severe condition and is usually detected much earlier; furthermore, some authors suggest that this disorder may affect eating habits in different

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

ways, leading to different effects on weight status (McElroy *et al.*, 2004; Reeves *et al.*, 2008). As such, the effect of a major depressive episode on weight loss in males observed in our study is supported by previous research (Carpenter *et al.*, 2000). By contrast, we found that dysthymia leads to increase abdominal fat in both the male and female population. These findings are consistent with some research studies of adults with depressive disorder or depressive symptoms (Ahlberg *et al.*, 2002; Zhao *et al.*, 2009; Needham *et al.*, 2010). In this regard, a review in adults showed that depression may be associated with abdominal obesity in both men and women (McElroy *et al.*, 2004). In children and adolescents, a relationship has only been observed between depression and BMI (Goodman and Whitaker, 2002; Anderson *et al.*, 2006, 2011; Rofey *et al.*, 2009) and between depression and %BF in the specific case of adolescent girls (Hillman *et al.*, 2010). However, the results of Tanofsky-Kraff et al. (2006) for a sample of 146 American infants did not show greater increases in %BF, measured by dual energy X-ray absorptiometry, in subjects with depression.

We found that anxiety leads to increased anthropometric and body composition parameters, with differences observed according to sex and the type and severity of anxiety. Thus, although we found that the total anxiety score was related to an increase in WC, BMI, and %BF in girls, detailed analysis showed that only somatic/panic manifestations were related. In this respect, our results agree with those of Hillman et al. (2010), who associated anxiety symptoms with %BF measured using dual energy X-ray absorptiometry in a population of 198 female adolescents in the United States. However, Hillman et al. (2010) and Midei & Matthews (2009), who used the waist-hip-ratio in both genders, did not observe a significant relationship between anxiety and abdominal fat. Unlike girls, the boys with higher scores for separation anxiety showed a greater increase in WC and BMI. This increase in adiposity was also found in boys diagnosed with social phobia and the increase in BMI in boys diagnosed with panic disorder. It is difficult to find the reasons for these differences according to type and severity of anxiety. To our knowledge there are no studies of children or

adolescents that analyse the different subtypes of anxiety. One possible explanation is the method used to assess anxiety. The symptoms identified by SCARED are quantitative measures; however, the diagnosis obtained by MINI-Kid is a dichotomous variable and the level of the disorder that it establishes takes into account a minimum number of criteria from the DSM-IV-TR and clinical interference. Social phobia disorder causes limitations, major subjective discomfort and social isolation. Therefore, adolescents with this disorder usually stay at home more, eat more, are more inactive, and do not participate social activities and sports. Similarly, panic disorder can lead to avoidance behaviours such as not leaving home in order to avoid a stressful situation. Therefore, adolescents with this disorder may be more inactive or eat more to reduce anxiety manifestations.

However, it is difficult to explain why some of these relationships were observed in boys but not in girls in our study. In contrast, several studies conducted on adolescents found that the relationship between anxiety and depression and obesity appeared to be more evident in the female subjects (Anderson *et al.*, 2006). Our results show a consistent relationship between anxiety and WC for both genders, similar to the results of other authors who also observed the same relationship with abdominal fat in adults (Ahlberg *et al.*, 2002; Needham *et al.*, 2010; Zhao *et al.*, 2011). Likewise, Rofey et al. (2009) observed weight gain in both boys and girls with anxiety.

Our findings on psychopathology and increased WC could support the results of Ahlberg et al. (2002), which indicated that psychopathology is more closely related to abdominal fat reserves than obesity per se. Furthermore, assessment of WC is important because it is a diagnostic criterion for metabolic syndrome (Varda and Gregoric, 2009). In isolation, some research studies in adults suggest that depression and/or anxiety predict an increased risk of metabolic syndrome and cardiovascular diseases (Goldbacher and Matthews, 2007; Luppino *et al.*, 2011). Indeed, depressive symptoms were associated with low insulin sensitivity among healthy adolescents (Shomaker *et al.*, 2010).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

In the same vein, a recent review in children studied the relationship between chronic stress and metabolic syndrome (Pervanidou and Chrousos, 2012). Moreover, other longitudinal studies also found stress associated with greater adiposity (van Jaarsveld *et al.*, 2009; Michels *et al.*, 2015a).

There are various interpretations of these findings. On the one hand, as we supported before, the psychopathology may lead to changes in eating behaviour and lifestyle (Reeves *et al.*, 2008). It has been shown that a substantial proportion of people with depressive and anxiety symptoms have increased appetite and tend to overeat and reduce their levels of physical activity, leading to weight gain (McElroy *et al.*, 2004). On the other hand, there is evidence of a shared neurobiological mechanism between emotional psychopathology and weight gain. The emotional psychopathology affects the hypothalamic-pituitary-adrenal axis, leading to increased cortisol secretion. High cortisol levels are associated with obesity, especially abdominal obesity (Miller *et al.*, 2007; Reeves *et al.*, 2008; Pervanidou and Chrousos, 2011; Michels *et al.*, 2015b). This mechanism could account for the consistent observation of a relationship between emotional psychopathology and increased WC in both sexes in our study. On contrary, major depression which is characterized by acute and intense manifestation could be associated with acute stress and lead a hyperactive hypothalamic-pituitary adrenal axis which inhibit gastric motility and promote the release of sugar into the bloodstream, thereby suppressing feelings of hunger (Miller *et al.*, 2007; Torres and Nowson, 2007).

Additionally, our results show that greater baseline anthropometric and body composition measurements influence the change in anthropometric and body composition measurements in adolescent girls but not in adolescent boys. We are unsure of the reasons for these results, although one possible explanation would be the difference in age at onset of puberty between the genders. Girls in the age range considered in the study are likely to be in mid-puberty, whereas boys in the same age range are more likely to be at the onset of puberty. In

prepubertal boys, changes in body composition due to puberty are minimal, and the prepubertal weight and fat distribution may not be critical to the future development of these parameters. By contrast, in girls of the same age, changes in body composition due to puberty have just begun and their bodies are being modified and defined. Therefore, the development of body composition in mid-puberty may influence the subsequent progression of body fat and fat distribution. In addition, mid-pubertal girls are at the stage of becoming concerned about their weight, and many of them want to be thinner. Consequently, girls with higher anthropometric and body composition parameter values make a conscious effort not to gain weight or fat.

5. EFFECT OF EMOTIONAL SYMPTOMS AND GENETIC FACTORS ON NUTRITIONAL STATUS

Several studies about serotonin and dopamine regulation in the brain examined MAOA (the key enzyme responsible for degrading serotonin and also the dopamine) and 5-HTT (the transport serotonin from extracellular space).

5.1 MAOA POLYMORPHISM

Although traditionally MAOA-L was associated with mental disorder (Cicchetti *et al.*, 2007; Lavigne *et al.*, 2013; Priess-Groben and Hyde, 2013), our results are not completely consistent. We only observed that while boys with MAOA-L seem to be associated with higher scores of social phobia, MAOA-H only showed a tendency among anxiety in girls. Other studies also failed to find this association (Eley *et al.*, 2004). In contrast to traditional evidence, others authors found associations with MAOA-H or found differences regarding gender. Aggressive behaviours and impulsivity are associated with male subjects with MAOA-L, whereas anxious symptoms are associated with

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

MAOA-H female subjects (Huang *et al.*, 2004; McDermott *et al.*, 2009; Rivera *et al.*, 2009; Reif *et al.*, 2012). In this vein, a recent meta-analysis showed that women with MAOA-H showed risk of developing panic disorder. In addition, Rivera et al. (2009) showed MAOA-H polymorphism confers high risks of depression in a large community sample. Probably, our results could be not conclusive in the association of MAOA with emotional symptoms since the symptoms showed less severity and this could be less sensible to find associations.

Regarding the effect of MAOA genotype on eating behaviours and weight, in girls our findings showed that although MAOA polymorphism did not show a main effect on adherence to sweet and fatty food pattern, we observed that there was a gene interaction with emotional symptoms to predict sweet and fatty food patterns. Indeed, only girls with MAOA-H and presence of emotional symptoms showed an increase of sweet and fatty food, low adherence to Mediterranean Diet and reduce physical activity with regard to control. Also, we observed that MAOA-H is associated with a higher increased of body fat percentage, but there was not significant differences in change in BMI and WC. Therefore, we observed that MAOA-H in presence of emotional symptoms confers a vulnerability to develop an obesogenic pattern which could lead to body fat gain. In contrast, boys with MAOA-H and emotional symptoms showed a reduction of weight and fat.

In girls, the effect of MAOA–H along with emotional symptoms on higher adherence to sweet and fatty food pattern could be explained by the effects of serotonin as well as those of dopamine. MAOA–H increases the transcription of MAOA. As a consequence, it causes an increase of MAOA activity and neurotransmitter metabolism. The neurotransmitter metabolism by MAOA reduces serotonin and dopamine availability. This fact is not only linked to some of psychopathological problems, like depression or anxiety, but also to eating and weight problems.

On the one hand, serotonin activity in the brain has been found to be predisposed to a selective intake of carbohydrates. Whereas high levels of serotonin decrease the intake of carbohydrates compared with protein-rich food in animal models, the inverse effect was observed when the level of serotonin is reduced (Leibowitz and Alexander, 1998). These effects of brain serotonin on food intake constitute part of a negative feedback loop to control its own function through the determination of carbohydrate intake. Carbohydrates intake has been found to increase the plasma Tryptophan/large neutral amino acid ratio by the food content and especially by effect of elevation of insulin and glucose in blood (Markus, 2008). As a result, the concentration of transporter of tryptophan or large neutral amino acid increase to be transported into the skeletal muscles for conversion into protein and, simultaneously, this fact facilitates the entrance of tryptophan in the brain and finally the synthesis of serotonin (Wurtman and Wurtman, 1986). Therefore, this mechanism should generate a negative feedback loop and reduce the carbohydrates intake when the serotonin levels are restored. However, this effect could relatively display importance in chronic low serotonin levels (by genetic predispose or presence of mental disorders) since unhealthy eating behaviours may develop and persist (Shepers and Markus, 2015).

On the other hand, it is known that the available levels of dopamine tend to have a heightened sensitivity to reward, a higher hedonic capacity, and a strong motivation to consume palatable food that could result in overeating and obesity. The relation between MAOA polymorphism with dopamine availably is partially known, since it has been shown that women with a copy of a MAOA-H have higher levels of homovanilic acid the major dopamine metabolite (Jönsson *et al.*, 2000). This finding implies that MAOA-H allele has high transcriptionally efficiency of MAOA and as a result causes an increase of dopamine metabolism and drops the dopamine availability. In this vein the reward deficiency syndrome could be explained as dopamine availability was related to appetite behaviours. The reward deficiency syndrome was

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

initially based on additional research and more recently applied to obesity. Furthermore, it has been hypothesized that eating may restore low levels of dopamine signalling (Bassareo and Di Chiara, 1997). This theory suggests that an increased tendency to eat excessively leading to obesity is due to low levels of brain dopamine and these increase appetite behaviours are methods to stimulate dopamine levels and compensate deficiency in the reward.

Nevertheless, there are contradictory findings about which is the MAOA allele responsible for increasing weight and high palatable food consumption. Our results are in concordance with some authors who showed that MAOA-H was associated with higher weight gain and palatable food consumption (Galvão *et al.*, 2012; Goldfield *et al.*, 2013). Galvão et al. (2012) in a sample of 354 pre-schooler children showed that MAOA-H polymorphism was associated with higher consumption of lipid-dense food, but it was only observed in boys. However, others authors showed that MAOA-L seems to be associated with overweight and obesity or food intake (Camarena *et al.*, 2004; Need *et al.*, 2006; Fuemmeler *et al.*, 2008; Agurs-Collins and Fuemmeler, 2011). Also the study of Agurs-Collins & Fuemmeler (2011) conducted in 20,745 adolescents did not find a main effect of MAOA on food intake, but they observed an interaction among depressive symptoms and MAOA variant on food consumption. Their results showed that only males with MAOA-L and depressive symptoms predict higher consumption of dense calorie food, but not sweets. Other studies showed that carrying of MAOA-L showed a higher risk of overweight and obesity on adult population (Camarena *et al.*, 2004; Need *et al.*, 2006) as well as on adolescents (Fuemmeler *et al.*, 2008) which only observed the association in boys. In this vein, our results showed that boys who carry MAOA-H and emotional symptoms showed low weight and body fat. In accordance to this, Fuemmeler et al. (2009) in a cross-sectional study conducted on 20.275 adolescents showed that MAOA-H along with depression decreased risk of obesity and overweight in males. These finding could support and help to explain why males with major

depression showed decrease of weight as our previous results or literature showed in adult population (Carpenter *et al.*, 2000).

In addition under these mixed results it is probable that there are gender differences in the effect of MAOA variant on both psychological and nutritional status. Probably this could be explained by a genetic load of X dysbalance among genders since boys have only one X chromosome and MAOA is linked to it. As a consequence, girls with two copies of high allele were more vulnerable to consume palatable food, particularly in case of experiencing emotional symptoms. Moreover, gender and age differences could be explained since MAOA is affected by ovarian hormones which could vary with age (Gundlah *et al.*, 2002). It has been shown that ovarian steroids, partially oestrogen, can decrease MAOA expression, resulting in elevated serotonin levels (Barth *et al.*, 2015). In this vein, the existing literature has found an association between MAOA and obesity in post-menopausal women (Camarena *et al.*, 2004; Need *et al.*, 2006) but not in adolescent girls (Fuemmeler *et al.*, 2008). This could be due to adolescents having more oestrogen that protects against serotonin decrease and at the same time prevents weigh gain and mental disorders.

As far as we are concerned, there are very few studies in this topic and their findings are mixed and inconsistent. Despite of this, our findings contribute to this body of literature and encourage to continue researching in this topic, specially examining the effect of interaction among genetic factors and emotional symptoms on nutrition status.

5.2 5-HTTLPR POLYMORPHISM

Regarding to 5-HTTLPR polymorphism, our results showed that girls with SS polymorphism showed higher scores of depression and anxiety symptoms. Similarity, Lee et al. (2014) found the association mainly in adult females, but not in males. Our results are consistent with most of

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

the evidence, which shows that SS polymorphism confers vulnerability to develop mental disorders or to suffer stress (Eley *et al.*, 2004; López-León *et al.*, 2008; Priess-Groben and Hyde, 2013; Lee *et al.*, 2014).

Our results found that the polymorphism of serotonin transporter gene variant influenced on food consumption since girls who carried SS/SL polymorphism showed a higher adherence to palatable food. This association between 5-HTTLPR and unhealthy dietary pattern adherence occurs both in the presence of emotional symptoms and without. As far as we are concerned this was the first study showing a gene-x-emotional symptom interaction on dietary pattern in adolescents using a longitudinal community sample. Only in an experimental study, Capello & Markus (2014) in a sample of 94 ungraduated students showed that students who were SS carriers reported higher stress and this fact was accompanied by an increase of appetite mainly of sweet snacks. Indeed, mainly in girls, 5-HTTLPR genotype moderates the relation between depressive feelings and could cause an increase of emotional eating (van Strien *et al.*, 2010), which has been associated with higher consumption of palatable food rich in sugar and fat (Braet and van Strien, 1997; Moens and Braet, 2007; Nguyen-Michel *et al.*, 2007; Elfhag *et al.*, 2008; Wallis and Hetherington, 2009; Ouwens *et al.*, 2012).

These results seem highly interesting in light of findings of an association between 5-HTTLPR SS and increased weight in animal models (Uceyler *et al.*, 2010) and human studies in adults (Sookoian *et al.*, 2008; Wallmeier *et al.*, 2013) and adolescents (Sookoian *et al.*, 2007; Fuemmeler *et al.*, 2008; Marmorstein and Hart, 2011; Markus and Capello, 2012). However, our results did not find any association with anthropometric or body composition parameters either in the presence of emotional symptoms or without it. Consistent with this we found another study in adolescent population that did not observe an interaction among this genetic polymorphs and depressive symptoms

on BMI (Fuemmeler *et al.*, 2009). Other studies in adult population did not find any association (Hameed *et al.*, 2015; Uzun *et al.*, 2015) or they found contradictory association (Bah *et al.*, 2010; Shinozaki *et al.*, 2013). Indeed 5-HTTLPR SS was also frequent in underweight individuals (Bah *et al.*, 2010; Shinozaki *et al.*, 2013), and was associated with anorexia nervosa (Calati *et al.*, 2011; Castellini *et al.*, 2012; Chen *et al.*, 2013). Other researches even showed that L variant was associated with higher BMI (Peralta-Leal *et al.*, 2012). Therefore, the evidence in this topic is not clear and it could explain that 5-HTTLPR SS could be related with an alteration of weight regulation by excess or defect as well as the interaction of other environmental and psychological factors could modulate this relation (Chen *et al.*, 2015; Dick *et al.*, 2015).

Nevertheless, our findings are in line with observations of enhanced food consumption, especially food rich in carbohydrates in response to reduce serotonin (Leibowitz and Alexander, 1998). At biological levels, functionality of serotonin transporter was associated with risk of obesity in human and animal studies (Erritzoe *et al.*, 2010; Homberg *et al.*, 2010; Giannaccini *et al.*, 2013; Hesse *et al.*, 2014) since low levels of serotonin activity have been associated with more appetite and body weight. Therefore, given that food intake and safety are influenced by hypothalamic serotonin-mediated feedback process, serotonin vulnerability associated with SS genotype may negatively influence satiety signals, leading to an increased energy intake (Simansky, 1996; Leibowitz and Alexander, 1998; Halford *et al.*, 2007).

Paradoxically, although S-allele is the most common genetic variant associated with obesity and emotional disorders, this polymorphism implies a low transcriptional efficiency. As a consequence, this leads us to think that SS may cause the reduction of serotonin reuptake and therefore increase the serotonin availably in extracellular space, which is the opposite that we would expect to find in mental disorder cases or

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

increase eating behaviour. Therefore, the mechanism of 5-HTTLPR SS has been described by several explanations.

First, it may due to the way in which the brain adjusts the body to high serotonin levels early in development in relation to regulation other serotonin receptors. For instance, high serotonin concentrations could be producing a decreased regulation of certain serotonin receptors in order to relatively reduce the serotonin transmission despite high serotonin levels. Therefore, SS polymorphism could induce high serotonin levels early in development to cause that during development the brain acquires more vulnerability to stress across the lifespan (Nordquist and Oreland, 2010; Priess-Groben and Hyde, 2013). Hence, S allele could be associated with depression later in life, since high serotonin levels have made the brain develop in a way that is more reactive to stress (i.e through a down-regulation of serotonin receptors). However, the effect of genetic variants on adolescent brain development are unknown, and it may be important to bear in mind that gene expression could vary across the life course (Nordquist and Oreland, 2010).

Secondly, other authors showed that there seem to be poor correlation between polymorphism and availability of serotonin transport in adults (Lim *et al.*, 2006) and it is suggested that SS increases the amygdale reactivity and reduces the ability to cope with stress (Nordquist and Oreland, 2010). The amygdale is a brain region which has an important role in emotional regulation and processing such as adequate reactions to potential harmful situations. Individuals with 5-HTTLPR SS have low functional connectivity between regions, implying that differences in emotional processing of negative stimuli appear (Barzman *et al.*, 2015). A meta-analysis showed that 5–HTTLPR SS polymorphism is associated with amygdale activation (Munafò *et al.*, 2008) and reduced ability to cope with stress. In addition, increased amygdale reactivity has been associated with stress hormones secretion. As mentioned before, the chronic hypersecretion of stress hormones such as cortisol

concentrations in the circulation results in insulin hyper secretion and these effects could lead to long-term accumulation of fat especially in visceral adipose tissue and showed adverse health consequences (such as arterial hypertension, carbohydrate intolerance, dyslipidaemia, metabolic syndrome and type 2 diabetes mellitus (Pervanidou and Chrousos, 2011). Moreover, cortisol-linked cases could be responsible for increased adiposity and could also lead to consume sweet and fat rich food as has been observed in paediatric population (Michels *et al.*, 2013, 2015b). Therefore, individuals who carry the S allele 5-HTTLPR produce a significantly higher cortisol response to psycho-social stress (El Hage *et al.*, 2009; Agüero-tejado, 2014). By this mechanism, SS polymorphism could confer vulnerability to engage unhealthy eating habits, increase in weigh, and risk of metabolic disease (Way and Taylor, 2010). Indeed, the 5-HTTLPR variant is thought to influence on cardiovascular disease (Bondy, 2007). For instance, SS and SL genetic variants are more frequent in individuals with diabetis mellitus tipus 2 (Iordanidou *et al.*, 2010).

Therefore, as one might expect carriers of the S allele of the 5-HTTLPR shows an increased vulnerability to stress. For instance, according to the gen-x-environment hypothesis, Marmorstein & Hart (2011) showed that children carrying SS alleles, who received public assistance since they lived in adverse conditions, obtained higher scores of depressive symptoms as well as BMI values in adulthood. It seems that those who carried 5-HTTLPR SS were particular vulnerable to stress and later development of mental disorders.

Consistently, a recent review suggests the potential effect of 5-HTTLPR on emotional regulation which could lead emotional eating by the effect of 5-HTTLPR on amygdala reactivity or other brain regions such as the cortical-limbic circuit or cingulate essential to emotion regulation (Schepers and Markus, 2015). Given the 5-HTTLPR vulnerability on stress, more gene-x-environmental research is required (Barzman *et al.*, 2015).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Another factor that could support the mixed results in the literature is a new variant of the polymorphism 5-HTTLPR that affects to the expression of ARN messenger (Kraft *et al.*, 2005; Hu *et al.*, 2006). This new variant consist in a single nucleotide polymorphism (SNP, rs25531), with a substitution of one nucleotide of adenine (A) by a guanine nucleotide (G) in the L allelic variant of serotonin transporter gen. So, the 5-HTTLPR polymorphism could be functionally tri-allelic, S, long-A and long-G (Hu *et al.*, 2006). However, individuals with S allele and Long-G allele compared to Long-A carriers, have lower mRNA transcription of the serotonin transporter. Therefore, short and long-G allele act similarity, so it is underlined to include these alleles in future investigations (Gallinat *et al.*, 2007).

However, it is known that the vast majority of phenotypes are polygenic, which means that they are influenced by multiple genes (Afari *et al.*, 2010). Although we did not observe a gene-x-gene interaction (by 5-HTTLPR-x-MAOA), we performed adjusted analyses by two genes to observe if the relation was significantly maintained. Other studies found interactions of 5-HTTLPR and MAOA predicted anorexia nervosa (Urwin and Nunn, 2004) which could be related with nutritional status. Therefore, future genetic studies also could take into consideration the interaction between both genes in risk of psychopathology, obesity or craving sweet food.

6. ROLE OF EMOTION REGULATION IN THE PREVENTION AND TREATMENT OF CHILDHOOD OBESITY

Our conceptual model posits the role of ineffective emotion regulation in weight gain that enhancing emotion regulation skills could be useful for prevention and treatment of obesity. Several interventions have recently been conducted in adults to test the effectiveness of emotion regulation strategies in regulating obesity and food intake (O'Reilly *et*

al., 2014). The most frequent strategies are based on the regulatory skills and correspond to new approaches within the cognitive behavioural psychological therapies. So, mindfulness therapy, acceptance and commitment therapy, self-compassion therapy (loving-kindness therapy), emotionally-focused therapy and dialectical behaviour therapy lead to less psychological distress (Forman *et al.*, 2009; Dalen *et al.*, 2010; Daubenmier *et al.*, 2011; Compare *et al.*, 2013; Mantzios and Wilson, 2014), less binge eating episodes (Wiser and Telch, 1999; Dalen *et al.*, 2010; Compare *et al.*, 2013), less emotional eating (Alberts *et al.*, 2012), less food cravings (Alberts *et al.*, 2010, 2012; Forman *et al.*, 2013), healthier eating patterns (Dalen *et al.*, 2010; Miller *et al.*, 2012b), weight loss (Forman *et al.*, 2009, 2013; Dalen *et al.*, 2010; Mantzios and Wilson, 2014) less personal barriers to physical activity (Tapper *et al.*, 2009), and improved self-efficacy to weight loss (Kidd *et al.*, 2013). Despite a few non-significant findings (Kearney *et al.*, 2012), these strategies are a promising approach for obesity treatment and prevention.

However, later mentioned emotion regulation technics have been applied in children and adolescents in the field of mental disorders but not in the prevention and treatment of child obesity. Despite of this, we highlighted some studies which could content similar features of emotion regulation technics as calm down and awareness about your feelings and delay with emotion in a healthy way.

In the field of childhood obesity prevention focusing on emotion regulation, to our knowledge there is only one pilot study and two on-going studies. A school-based pilot intervention translated specific components of a violence and substance abuse program into a lifestyle intervention. This pilot program included seven lessons focusing on teaching tools for controlling impulsiveness, recognizing and adaptively dealing with stress, and analysing the effectiveness of possible solutions. The result was a significant change in positive attitudes toward self-regulation of appetite and positive changes in food and lifestyle (Riggs *et al.*, 2007; Jacobson and Melnyk, 2011; Miller *et al.*,

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

2012a; Boutelle *et al.*, 2014). Another intervention study conducted in adolescents and their parents applied a cognitive-behavioural skills intervention combined with nutrition education and physical activity. The program included 4 of 15 sessions on cognitive reappraisal, emotional and behavioural regulation, stress, coping, goal-setting and overcoming barriers to a healthy lifestyle. After six months, the participants had lower BMI (Melnyk *et al.*, 2013a). Another school-based intervention study is currently being carried out in toddlers, combining a traditional obesity prevention program with a program to enhance self-regulation. The study teaches children tools for behavioural self-regulation and includes a parental intervention (Miller *et al.*, 2012a).

In childhood obesity treatment, successful intervention studies have applied cognitive behavioural therapy to encourage the development of effective ER (Vignolo *et al.*, 2008; Sacher *et al.*, 2010; Jacobson and Melnyk, 2012; Kelly *et al.*, 2012; Halberstadt *et al.*, 2013; Melnyk *et al.*, 2013b). One interventional study applied a cognitive-behavioural skills-building program in primary care services. The program was carried out through seven telephone and clinical sessions, which included evaluation of emotional responses and behaviour. Although their results did not show effect on BMI, they observed an increase in physical activity, healthy lifestyle behaviours and nutrition knowledge. Long-term results were mainly attained in children with supportive families (Jacobson and Melnyk, 2012).

Therefore, given the positive and promising findings in adult population and some intervention with feature of emotion regulation in children, based on our new model future studies should urgently explore the impact of emotion regulation on obesity prevention and treatment by applying interdisciplinary interventions.

7. LIMITATIONS

7.1 FOLLOW-UP STUDY LIMITATIONS

The present study was subject to certain limitations which should be considered when interpreting the results. The main limitation is the reduced sample size and follow-up rate. The dropped of the follow-up rate could be due to several factors. Firstly, the follow-up studies of children over several years created difficulties regarding the appropriate sample size acquisition, which resulted mainly from children moving between schools and movement with their parents to other parts of town or even other parts of the country. Indeed, the majority of students in this period of age changed from primary school to high school. However, the researchers did a considerable effort to contact with all participants and conducted a complex work to find the new school of each participant. For instance, trying to obtain the maximum participation, the informed consent, signed or not should be returned to the researchers and explain the reasons why they rejected to participate. In cases in which, the informed consent was not returned, the researchers phoned personally the families, in order to obtain the maximum number of participants. Despite the work and the efforts, the response of the families and adolescents was negative in most of the cases. Secondly, during the follow-up phase, the parents as well as the adolescents were requested to return the informed consent and in most cases were the adolescents who rejected to participate. Due to the drop-out rate, several analyses were run to evaluate the baseline possible differences between subjects who followed up and subjects who dropped out.

Regarding the methods, firstly, we obtained information of different anxiety and depression disorders present in the study population. However, due to the high level of comorbidity between depression and anxiety (Esbjorn *et al.*, 2010; Kendall *et al.*, 2010; Romero *et al.*, 2010; Essau *et al.*, 2014) we had to adjust our statistical analyses for all of

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

these variables or to use only the emotional symptoms variable. Secondly, we have no data on the subjects' dietary intake in the baseline phase in order to examine the change on dietary patterns along the period of study or whether the dietary pattern has a potential role in preventing mental health problems in children. Also, to estimate the dietary pattern, we use a principal component analysis, that also enables us to identify the usual diet that adolescents follow, which has scarcely been studied. Nevertheless, this statistical method also has some limitations. Several of the components extracted from a principal component analysis are subjective. For example, based on the majority of studies we chose score coefficients from 0.3, but other authors chose score coefficients from 0.2.

Another limitation was the non-inclusion of other confounding variables such as ethnicity, pubertal stage, obesity and maternal depression, or social factors influencing eating patterns among others.

It is should also highlighted that other functional polymorphisms may also influence on this association. However, we examined two of the most related MAOA and 5-HTTLPR genotype in emotional symptoms as well as unhealthy-behaviour obesity related to try to explain one of the possible mechanisms involved. Our study represents a contribution to the growing body of studies on the relationship between the variant of the MAOA and 5-HTTLPR polymorphism, the most frequent in child and adolescent psychopathological symptoms and obesity or obesity-behaviour related.

7.2 NARRATIVE REVIEW LIMITATIONS

Since the model presented in this study is intended to illustrate the potential role of emotional regulation in the development of obesity, other risk factors in the stress-obesity pathway have not been mentioned. Firstly, it has been assumed that the associations between stress and obesity share genetic factors like polymorphism on the serotonin 5-HT-2C receptor gene, monoamine oxidase A, serotonin transporter gene or the fat mass and obesity associated gene (FTO gene) (Fuemmeler *et al.*, 2009a; Wermter *et al.*, 2010; Velders *et al.*, 2012; Barzman *et al.*, 2015). In addition, a recent review suggested that socioeconomic disadvantages and family disharmony are a common starting point for weight gain and psychological distress in children (Hemmingsson, 2014). At the same time, several prenatal, perinatal and postnatal factors (e.g. toxics exposure, maternal nutrition, maternal stress, maternal psychopathology and negative events during early life) have been identified as obesity and/or neurodevelopmental risk factors (Wermter *et al.*, 2010; Entringer *et al.*, 2012; Provençal and Binder, 2014).

Additionally, due to the multifaceted nature of the concept, emotional regulation has been discussed in the literature under other terms such as self-regulation, self-efficacy, effortful control, impulsivity and emotional eating. This lack of consistent terminology has hindered the literature search strategy and interpretation of studies.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

8. IMPLICATION AND FUTURE RESEARCH LINES

From a public health point of view, several implications are developed. Given adolescence is a time of increased risk of emotional, behavioral and weight problems as well as emergence of unhealthy eating and lifestyle behavior, is crucial to develop approaches to promote healthy dietary and physical habits and boost mental well-being. This becomes even more important since the relation between mental disorders and obesity and lifestyle is increasingly strong. Therefore, researchers should be encouraged to conduct effective and multidisciplinary novel approaches to foster early prevention and treatment of childhood obesity. Novel prevention and treatment strategies should focus on emotion regulation. Developing an effective emotion regulation during vulnerable stages of the development could result on health issues such as preventing unhealthy eating behaviors, weight gain, and psychological problems in children as well as boosting well-being. Thus, future emotional regulation interventions against childhood obesity are needed to confirm the validity of our model.

Our findings, therefore, boost to continue researching in these topics and confirm our results. Future prospective research should aim to elucidate the interrelationship between depression-anxiety and eating behaviour and obesity in terms of neurobiology and genetics, especially in a large community sample of children and young people. Revealing genetic mechanism implied an allowance to detect individuals who are at risk to develop obesity and unhealthy obesity-behaviours and are vulnerable to suffer mental disorders. Furthermore, studies ought to clarify which mechanisms play a role in gender differences. They also should include exteriorized disorder related to impulsivity such as hyperactivity and attention deficit disorder, which also has been recently associated with obesity and overeating.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Conclusions

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

CONCLUSIONS

1) The prevalence of anxiety and depressive symptoms in schoolchildren from 8 to 12 years old is 46.7% (95%CI: 44.1-49.2) and 11.4% (95%CI: 9.8-13.0) respectively, with girls showing more anxiety symptoms than boys. The prevalence of overweight and obesity in schoolchildren from to 8 to 12 years old is 31% (95%CI: 27.7- 32.4) with no gender differences.

At 13-15 years old, 15.6% of the adolescents monitored were overweight or obese, 36% performed low levels of physical activity and their daily intake of calcium, iron, magnesium, vitamin D, folic acid and vitamin A were below two-thirds of the reference dietary intake for boys and girls.

2) Girls with emotional symptoms during early adolescence consumed more sweets and sweet dairy desserts, but fewer dairy products and have lower levels of physical activity. 39.7% adhered strongly to a dietary pattern rich in sweet and fatty foods. In fact, emotional symptoms are predicted to increase the risk of adherence to this pattern more than fourfold. However, there were no differences among adolescent boys. In addition, there were no differences in energy and nutrient intake in relation to emotional symptoms between the two genders.

3) Depressive symptoms in adolescence and SES predict a risk of low adherence to the Mediterranean diet, although at this age we observed no significant association between Mediterranean diet adherence and overweight or obesity.

4) Emotional symptoms in preadolescence influence adiposity in adolescence. In boys, waist circumference and BMI increased with depressive and separation anxiety symptoms as well as social phobia

and/or dysthymia. However, in girls, waist circumference and the percentage of body fat increased with somatic panic and/or dysthymia.

5) High-activity MAOA polymorphism in girls with emotional symptoms may confer susceptibility to unhealthy behavior related to obesity, such as increased sweet and fatty food pattern adherence, reduced Mediterranean diet adherence and reduced physical activity, which could lead to an increased body fat percentage, as we observed. However, the high-activity MAOA polymorphism in boys with emotional symptoms is associated with lower weight status and waist circumference values. Furthermore, and only in girls, the SS/SL variant of the serotonin transporter could show an effect on increased sweet and fatty pattern adherence in adolescence, both in conjunction with and without emotional symptoms.

6) Our model posits that emotion regulation is a fundamental link between childhood stress and obesity since stress, combined with ineffective emotion regulation, could cause abnormal cortisol patterns, emotional eating, decreased physical activity, increased sedentary behavior and the onset of sleep problems.

Global conclusions:

During adolescence, the presence of emotional symptoms and genetic factors, together with socioeconomic status, has an influence on nutritional status, mainly among girls, pushing them towards unhealthy behaviors related to obesity. Emotional psychopathology in preadolescence is associated with increased weight gain and abdominal fat in adolescence, albeit with some differences in the precise relationship with each anxiety and depression disorder according to gender. Encouraging an emotion regulation could therefore be an effective new approach, as well as a nutritional and physical activity intervention, in the early prevention and treatment of childhood obesity.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

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UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

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UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

UNIVERSITAT ROVIRA I VIRGILI
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Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

SCIENTIFIC CONTRIBUTIONS

1. Scientific contributions related to the thesis

2. Other scientific contributions

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

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UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

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UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

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Emotional psychopathology and increased adiposity: Follow-up study in adolescents

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Keywords:
Depression
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Waist circumference
Longitudinal study

A B S T R A C T

Based on data from a three-year longitudinal study, we assess the effect, according to gender, of emotional psychopathology in preadolescence on anthropometric and body composition parameters in adolescence ($N = 229$). Psychopathology was assessed using the *Screen for Childhood Anxiety and Related Emotional Disorders*, the *Children's Depression Inventory* and the *MINI-International Neuropsychiatric Interview for Kids*. Body fat percentage (%BF), waist circumference (WC) and body mass index (BMI) were also determined. Following analysis with adjusted multiple regression models, the results indicated that symptoms of depression and separation anxiety were significantly associated with increased WC and BMI in boys, and that somatic symptoms were associated with increased WC and %BF in girls. Diagnosis of social phobia, panic disorder or dysthymia led to significantly increased WC and/or BMI in boys and dysthymia increased WC in girls. These findings suggest that emotional psychopathology in preadolescence is associated with increased weight gain and abdominal fat in adolescence.
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Overweight and obesity now affect 9–36% of the child and adolescent population in several developed and developing countries (Gupta, Goel, Shah, & Misra, 2012; Lobstein & Frelut, 2003; Ogden, Carroll, Kit, & Flegal, 2012; Valdes Pizarro & Royo-Bordonada, 2012). This pathology is associated with serious complications in childhood and adolescence and increased morbidity and mortality in adulthood. Overweight and obese children are also at risk of obesity in adulthood (Deckelbaum & Williams, 2001). Moreover, obesity is a chronic disease with a complex multifactorial nature. Numerous genetic and environmental factors have been found to contribute to the recent epidemic of obesity. Among these risk factors, psychological factors warrant particular attention.

Emotional psychopathology includes some of the most common psychiatric disorders among children and adolescents, such as depression or anxiety disorders (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Esbjorn, Hoeyer, Dyrborg, Leth, & Kendall, 2010). In adolescence, anxiety disorders are the most prevalent condition (31.4%), followed in third place by mood

Abbreviations: %BF, body fat percentage; WC, waist circumference; BMI, body mass index; BIA, bioelectrical impedance; CDI, Children's Depression Inventory; SCARED, Screen for Childhood Anxiety and Related Emotional Disorders; MINI-KID, Mini-International Neuropsychiatric Interview for Kids.

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UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

disorders (14.3%) (Merikangas et al., 2010). In Spain, symptoms of depression and anxiety affect 9% and 47%, respectively, of the school population (Canals, Marti-Henneberg, Fernández-Ballart, & Domenèch, 1995; Romero Acosta et al., 2010). Emotional psychopathology often shows comorbidity with other psychological disorders and is related to other childhood complications such as physical dysfunction, substance abuse, suicide attempts and hospitalization. Furthermore, anxiety and depression in childhood may predict adult depression and anxiety disorders (Bittner et al., 2007; Canals, Domenech-Llaberia, Fernandez-Ballart, & Marti-Henneberg, 2002; Kendall, Flannery-Schroeder, & Webb, 2004).

Therefore, obesity and emotional psychopathology have become critical public health problems.

The relationship between emotional psychopathology and obesity is the subject of considerable debate, both in adults (Ahlberg et al., 2002; Carpenter, Hasin, Allison, & Faith, 2000; Gariepy, Nitka, & Schmitz, 2010; Hach, Ruhl, Klotsche, Klose, & Jacobi, 2006; Needham, Epel, Adler, & Kiefe, 2010; Williams et al., 2009; Zhao et al., 2011) and in children and adolescents (Anderson et al., 2010; Duarte et al., 2010; Goodman & Whitaker, 2002; Hillman, Dorn, & Bin, 2010; Midei & Matthews, 2009; Rhew et al., 2008; Rofey et al., 2009; Tanofsky-Kraff et al., 2006). It has been suggested that early depression and/or anxiety may be predictive of obesity in adolescence (Anderson et al., 2010; Goodman & Whitaker, 2002; Hillman et al., 2010; Rofey et al., 2009) and in adulthood (Anderson, Cohen, Naumova, & Must, 2006; Vamosi, Heitman, & Kyvik, 2010). However, other authors have not observed this relationship (Duarte et al., 2010; Midei & Matthews, 2009; Rhew et al., 2008; Tanofsky-Kraff et al., 2006). Anderson et al. (2006) studied a community-based US cohort from childhood to adulthood and reported that anxiety and depression disorders were associated with higher weight status in females, whereas in males, depression was associated with lower BMI and childhood anxiety was not substantively associated with weight status. Similarly, among white adolescent girls studied over a two-year follow-up period, depression was related to a higher likelihood of obesity (Anderson et al., 2010). Goodman and Whitaker (2002) found that North American adolescents with symptoms of depression showed risk of obesity at 1-year follow-up in both genders. By contrast, among adolescents studied over a one-year follow-up period using height and weight measurements, depression was not associated with BMI in either gender (Rhew et al., 2008). According to a recent review, the relationship between psychological factors and obesity in children and adolescents has not been confirmed (Incledon, Wake, & Hay, 2011).

Although body mass index (BMI) is the most common measurement of overweight, methods measuring excess fat and its abdominal distribution allow us to study more important cardiovascular risk factors. However, few pediatric studies examining the relationship between psychopathology and obesity have applied these methods (Hillman et al., 2010; Midei & Matthews, 2009; Tanofsky-Kraff et al., 2006).

The transition from childhood to adolescence is a critical period involving both psychological and physical maturation, and as such various symptoms and changes in body composition may be presented. Gender and age are key modulators of emotional psychopathology and obesity. On the one hand, girls are found to experience more emotional problems in adolescence than boys (Canals et al., 1995, 2002; Conley, Rudolph, & Bryant, 2012; Moksnes, Espnes, & Lillefjell, 2012); however, there is also evidence that overweight and obesity have become more prevalent in males during puberty (Serra-Majem, Ribas-Barba, et al., 2003).

To date, there have been few longitudinal studies in adolescents that examine the influence of depression and anxiety on adiposity according to gender. Furthermore, none of the studies carried out has used an accurate methodology to assess adiposity in a non-clinical adolescent population.

Given the limitations of current knowledge, we decided to assess the effect, according to gender, of emotional psychopathology in preadolescence on anthropometric and body composition parameters in adolescence. We hypothesized that emotional psychopathology at baseline would contribute significantly to adiposity gain at three-year follow-up in adolescents, and that this relationship would be different between males and females.

Methods

Sample and study design

A three-year longitudinal study was conducted of 229 schoolchildren of preadolescent to adolescent age. The participants were recruited from a three-phase epidemiological study of anxiety and depression disorders that was begun in 2007 in the town of Reus (Catalonia, Spain).

The baseline sample in the study was a group of 1514 schoolchildren (720 boys and 794 girls) with a mean age of 10.2 years old (SD = 1.2) from 13 schools randomly chosen from the town's state schools and state-subsidized private schools. Screening questionnaires for anxiety and depression were used to select a sample at risk of emotional problems and a risk-free control sample. A child was considered to be at risk of emotional psychopathology if he/she had a score equal to or greater than 25 on the Screen for Children's Anxiety Related Emotional Disorders (SCARED; Birmaher et al., 1997) and/or a score equal to or greater than 17 on Kovacs' (1985) Children's Depression Inventory (CDI). For the control group, one child without risk of emotional psychopathology (SCARED score below 25 and CDI score below 17) was selected for every three children at risk of emotional psychopathology, matching for age, gender and type of school. Therefore, in the second phase, the participants were 562 children (254 boys and 308 girls), of which 405 were at risk of an emotional disorder (235 at risk of anxiety disorder and 170 at risk of depressive disorder) and 157 were controls. The mean age was 11.2 years old (SD = 1.0) (Romero Acosta et al., 2010; Vigil-Colet et al., 2009). At the follow-up three years after the baseline, all second-phase subjects were contacted. 245 adolescents (147 girls and 98 boys) agreed to participate, with a mean age of 13.5 years (SD = .9). Sixteen subjects

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

were excluded due to a lack of data. Therefore, in this paper we examine the subjects who participated in all three phases of the study. There were no psychopathological, baseline anthropometric and body composition differences three-phase participants and those who withdrew.

Procedure

At baseline, in the first phase, we assessed the presence of anxiety and depressive symptoms and recorded anthropometric, body composition, sociodemographic and body satisfaction data. One year later, in the second phase, we individually evaluated the presence or absence of a diagnosis of anxiety or depression disorder. In the third stage, we recorded anthropometric and body composition parameters and administered questionnaires on dietary quality and physical activity.

The project was approved by the Rovira i Virgili University ethics committee for research on individuals and received permission from the Ministry of Education of the Government of Catalonia. The board of governors of each school was subsequently asked to participate in the first and second phases, and informed consent was requested from the parents of the preadolescent and adolescent subjects participating in the third phase.

Measures

Demographic and sociocultural data

Sociocultural level was calculated according to parents' professions, using the Hollingshead index (Hollingshead, 2011).

Emotional psychopathology

Screen for Childhood Anxiety and Related Emotional Disorders (SCARED) (Birmaher et al., 1997)

This is a 41-item questionnaire used in the pediatric population to screen for anxiety symptoms. The questionnaire was designed from clinical studies of the anxiety disorders in the DSM-IV-TR. We used the validated Spanish version (Vigil-Colet et al., 2009), which considers four factors in the factorial analysis: somatic/panic, social phobia, generalized anxiety and separation anxiety. It has good levels of reliability (overall Cronbach's alpha of .86, and by factors: panic/somatic, alpha .78; social phobia, alpha .69; generalized anxiety, alpha .69; and separation anxiety, alpha .70). A score of 25 has been considered the cut-off point for risk of anxiety (Birmaher et al., 1997; Canals, Hernández-Martínez, Cosi, & Domènech, 2012).

Children's Depression Inventory (CDI) (Kovacs, 1985)

This is a 27-item questionnaire for people aged 7–17 years old. It assesses depressive symptoms in the cognitive, affective and behavioral spheres. The Spanish version has good internal consistency and good test–retest reliability (Cronbach's alpha between .70 and .94). We used a score of 17 as the cut-off point for depressive symptoms (Kovacs, Barrio, & Carrasco, 2004).

Personal interview. MINI-International Neuropsychiatric Interview for Kids (MINI-Kid) (Sheehan et al., 1998)

This is a structured diagnostic interview for children aged 6–17 years old, based on DSM-IV and ICD-10 criteria. The MINI-Kid is organized into diagnosis sections. All questions have a binary response format (yes/no). The administration time is approximately 30 min. The reliability and validity of this interview have been demonstrated in a recent study (Sheehan et al., 2010). Mood disorders and anxiety disorders present good psychometric properties (AUC = .81, k = .56, sensitivity = .85, specificity = .76; and AUC = .84, k = .59, sensitivity = .90, specificity = .77, respectively). This study assessed the diagnosis of major depressive episode and dysthymia, as well as anxiety disorders: panic disorder with or without agoraphobia, separation anxiety disorder, generalized anxiety disorder and social phobia.

Anthropometric and body composition measurements

Anthropometry

The anthropometric parameters evaluated in the initial and final phase were weight, height and waist circumference (WC). Body mass index (BMI) (kg/m^2) was then calculated. Weight was measured using the Tanita® TBF-300 scale, which has an accuracy of 100 g and a maximum weight of 200 kg. WC was measured using a flexible tape and height was measured using an inextensible tape measure, with a variation of 1 mm considered acceptable. WC was measured at the midpoint between the iliac crests and the lower costal margin, without clothes. Weight and height were measured with light clothing, barefoot and without heavy objects in pockets.

Bioelectrical impedance (BIA)

The TANITA® TBF-300 body composition analyzer was used to assess body composition. The results were expressed as follows: fat mass in kilograms (kg), body fat percentage (%BF), lean mass in kg, water content in kg and baseline metabolic rate in kilocalories.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Lifestyle: dietary and physical activity

Krece plus food questionnaire (Serra-Majem, Aranceta-Bartrina, Ribas-Barba, Sangil-Monroy, & Rérez-Rodrigo, 2003)

This test determines dietary quality. It consists of 16 items, with a score of 1 or −1 for each item. The maximum possible score is 11, and the minimum is −5.

Krece Plus physical activity questionnaire: the Krece Plus short physical activity test (Román-Viñas, Serra-Majem, Ribas-Barba, Pérez, & Aranceta-Bartrina, 2003)

This test consists of two questions. Each question has six possible responses, with a score of 0–5. The maximum score for the test is 10 and the minimum is 0.

Body satisfaction

Body Areas Satisfaction Scale (BASS) (Cash & Szymanski, 1995)

This scale assesses an individual's degree of satisfaction or dissatisfaction with 10 body areas. The scale rates satisfaction with each different body part with a score of 1–5.

Statistical analysis

We confirmed the normality of the variables and the criteria for application of the statistical tests. The degree of non-independence of observations from children nested within the same school can be estimated using intraclass correlation coefficients (ICC) (Kenny, Manneti, Pierro, Livi, & Kashi, 2002; Pardo, Ruiz, & San Martin, 2007). We found no evidence to suggest that observations were non-independent for the outcome variable: "change WC" (ICC = .0827), "change BMI" (ICC = .0001), and "change %BF" (ICC = .0192, $ps > .05$). Therefore, we applied traditional statistical analysis. The chi-square test, Student–Fisher *t*, analysis of variance adjusted for the Bonferroni multiple comparisons and Pearson correlations were used according to the types of variables compared. The values are expressed as the mean and standard deviation for the quantitative variables, and as percentages for the qualitative variables.

The change in anthropometric and body composition measurements from preadolescence to adolescence was calculated as the difference between the final values in adolescence and the initial values in preadolescence.

Various multiple linear regression models were applied to assess the effect of psychopathology on changes in anthropometry and body composition. The multiple linear regression models used the ENTER method for psychopathological variables and the STEPWISE method for the other adjustment variables. The psychopathological variables were as follows: depressive symptoms in model 1; anxiety symptoms in model 2; symptoms of depression, separation anxiety, generalized anxiety, somatic/panic and social phobia in model 3; and diagnosis of panic disorder, separation anxiety disorder, generalized anxiety disorder, social phobia, diagnosis of major depressive episode and dysthymia in model 4. The other adjustment variables were age (years), initial WC (cm), initial BMI (kg/m^2) and initial %BF (%), according to the dependent variable in the multiple linear regression model, the Krece Plus diet test and Krece Plus physical activity test scores, and the body areas satisfaction score.

The lower threshold for statistical significance was $p < .05$. Data were analyzed using SPSS 17.0 for Windows.

Results

Descriptive data

Table 1 shows the general, psychopathological, anthropometric and body composition characteristics in preadolescence (phase one) and adolescence (phase three).

Relation between emotional psychopathology and adiposity

Table 2 shows the correlation between the scores for anxiety and depression symptoms and the change in adiposity over the period of the study. A slight or moderate correlation was observed between separation anxiety and increased BMI ($r = .220$) and %BF ($r = .175$) in girls and between separation anxiety and increased WC in both gender (Boys, $r = .274$; Girls $r = .196$). Somatic symptoms were also found to be slightly or moderately associated with changes in WC ($r = .269$), BMI ($r = .187$) and %BF ($r = .210$) in girls. Scores for depressive symptoms were correlated with change in %BF in girls ($r = 2.14$). In addition, the presence of depressive symptoms in preadolescence was associated with significant increases in BMI in boys ($p = .040$) but not in girls ($p = .150$) and with increases in %BF in both genders ($p < .05$), compared to adolescents without these symptoms (measured by the *t*-test). Although the relationship between depressive symptoms and change in WC was not significant in either boys or girls, those adolescents who presented depressive symptoms showed a greater increase in WC than adolescents without depressive symptoms.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Table 1
Sociodemographic, psychopathological, anthropometric and body composition characteristics.

Subjects	Preadolescence				Adolescence			
	Total ($n = 229$)	Boys ($n = 87$)	Girls ($n = 142$)	p Value between boys and girls	Total ($n = 229$)	Boys ($n = 87$)	Girls ($n = 142$)	p Value between boys and girls
Age (years)[i]	10.2 (.9)[i]	10.1 (.9)	10.4 (.9)	.008	13.5 (.9)	13.4 (.9)	13.6 (.9)	.037
Sex (%)		38.0	62.0			38.0	62.0	
Socioeconomic level								
Low (%)	34.6	33.7	35.2	.909	34.6	33.7	35.2	.909
Medium (%)	44.7	44.2	45.1		44.7	44.2	45.1	
High (%)	20.6	22.1	19.7		20.6	22.1	19.7	
Emotional variables								
Score: total SCARED[a]	29.5 (10.9)	30.1 (12.8)	29.2 (9.7)	.554				
Score: somatic panic	5.5 (4.0)	6.4 (4.8)	5.0 (3.3)	.026				
Score: social phobia	6.7 (3.0)	6.2 (3.1)	7.0 (2.9)	.034				
Score: generalized anxiety	7.5 (3.3)	7.4 (3.5)	7.5 (3.1)	.775				
Score: separation anxiety	9.9 (4.3)	10.2 (4.8)	9.7 (3.9)	.440				
Body satisfaction scale (score): BASS[b]	26.9 (4.6)	27.6 (4.7)	26.4 (4.6)	.058				
Depressive symptoms (CDI test)[c] (%)	18.8	18.4	19.0	.907				
Diagnosis of major depressive episode[d] (%)	2.2	3.4	1.4	.305				
Diagnosis of dysthymia[d] (%)	4.4	5.7	3.5	.424				
Diagnosis of separation anxiety disorder[d] (%)	5.7	5.7	5.6	.971				
Diagnosis of generalized anxiety disorder[d] (%)	13.1	11.5	14.1	.573				
Diagnosis of anxiety disorder[d] (%)	1.7	2.3	1.4	.618				
Diagnosis of social phobia[d] (%)	5.7	3.4	7.0	.254				
Anthropometric and body composition measures								
Height (m)	1.4 (.08)	1.4 (.07)	1.5 (.08)	.001	1.6 (.1)	1.6 (.1)	1.6 (.06)	.007
Weight (kg)	39.2 (9.4)	37.2 (8.0)	40.6 (9.9)	.006	52.5 (10.1)	52.8 (10.4)	52.2 (9.9)	.662
BMI[e] (kg/m^2)	18.8 (3.2)	18.5 (2.9)	19.1 (3.3)	.171	20.1 (3.3)	19.8 (3.5)	20.3 (3.2)	.265
BF[f] (%)	20.7 (7.8)	18.0 (6.5)	22.2 (8.1)	<.001	20.9 (8.8)	14.1 (6.8)	24.7 (7.4)	<.001
WC[g] (cm)	66.0 (7.6)	66.1 (7.0)	66.0 (7.9)	.912	72.4 (8.1)	73.7 (8.8)	71.5 (7.5)	.038
Change[h] in BMI[e] (kg/m^2)					1.3(1.7)	1.4 (1.7)	1.3 (1.8)	.727
Change[h] in BF[f] (%)					.3 (5.9)	−3.6 (4.5)	2.5 (5.5)	.007
Change[h] in WC[g] (cm)					6.4 (5.7)	7.8 (5.6)	5.6 (5.7)	<.001
Lifestyle characteristics								
Food: Krece Plus test score					5.7 (2.2)	5.8 (2.2)	5.6 (2.2)	.442
Physical activity: Krece Plus physical activity questionnaire score					5.6 (2.2)	6.2 (2.1)	5.2 (2.2)	<.001

[a] SCARED: Screen for Childhood Anxiety and Related Emotional Disorderd.
[b] BASS: Body Areas Satisfaction Scale.
[c] CDI: Children's Depression Inventor.
[d] MINI-Kid: MINI-International Neuropsychiatric Interview for Kids.
[e] BMI: body mass index.
[f] BF: body fat.
[g] WC: waist circumference.
[h] Change: difference between the endpoint (adolescence) and the baseline point (preadolescence).
[i] Expressed as mean (standard deviation), except where % shown.

Psychopathological predictors of adiposity

Tables 3 and 4 show the multiple linear regression models adjusted for the various lifestyle variables, initial anthropometry and body composition, body satisfaction and age for boys and girls, respectively.

For boys (Table 3), model 1 shows that the presence of depressive symptoms significantly accounts for the increase in WC, BMI and %BF. Model 3, which adjusts for the anxiety symptoms, confirms the results for the increase in WC ($B = 3.50$, $p = .029$), BMI ($B = 1.25$, $p = .022$) and %BF ($B = 3.32$, $p = .024$). Model 4, which adjusts for the diagnostic category variables, shows that diagnosis of dysthymia was a highly significant predictor of increased WC ($B = 9.25$, $p = .001$) and BMI ($B = 3.50$, $p < .001$). However, diagnosis of major depressive episode was found to be inversely related to BMI ($B = -2.98$, $p = .020$). With regard to anxiety in boys, we can also observe, in model 3, that the symptoms of separation anxiety were associated with increased WC ($B = .43$, $p = .006$) and BMI ($B = .10$, $p = .041$). Of the anxiety disorders (model 4), social phobia was associated with increased WC ($B = 9.59$, $p = .0006$) and BMI ($B = 2.90$, $p = .019$), and panic disorder was related to increased BMI ($B = 2.83$, $p = .043$). In addition, lifestyle variables were found to be significant predictors of WC or BMI in all models ($p < .05$).

For girls (Table 4), no significant relationship was observed between depressive symptoms and WC, BMI or %BF (models 1 and 3). However, model 4 shows that a clinical diagnosis of dysthymia significantly influenced the increase in

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Table 2
Association between emotional symptoms in preadolescence and changes in anthropometric and body composition parameters in adolescence.

	Change[a] from preadolescence to adolescence								
	All			Boys			Girls		
	Change in WC[b]	Change in BMI[c]	Change in %BF[d]	Change in WC	Change in BMI	Change in %BF	Change in WC	Change in BMI	Change in %BF
Total SCARED[e] (score)									
r[h]	.144	.086	.061	.108	−.012	.030	.174	.163	.125
p	.037	.194	.373	.348	.912	.795	.045	.052	.150
Somatic panic[e] (score)									
r	−.171	.081	.055	.008	−.044	−.047	.269	.187	.210
p	.013	.221	.427	.944	.685	.679	.002	.026	.015
Social phobia[e] (score)									
r	−.006	−.031	.055	<.001	−.110	.004	.039	.024	−.048
p	.933	.640	.427	.996	.310	.974	.653	.773	.581
Generalized anxiety[e] (score)									
r	−.025	−.029	.042	.002	−.097	.030	.023	.018	.002
p	.717	.665	.538	.986	.310	.793	.796	.834	.979
Separation anxiety[e] (score)									
r	.231	.196	.101	.274	.131	.115	.196	.220	.175
p	.001	.003	.141	.015	.163	.313	.023	.009	.043
Depressive symptomatology[f](score)									
r	.130	.138	.117	.176	.133	.109	.085	.138	.214
p	.059	.037	.089	.124	.218	.339	.332	.101	.013
Depressive symptomatology[f,g]									
No	5.9 (5.4)[i]	1.2 (1.7)	−.2 (5.7)	7.1 (4.6)	1.1 (1.6)	−4.1 (4.0)	5.3 (5.7)	1.1 (1.8)	2.0 (5.3)
Yes	8.1 (6.7)	1.9 (1.8)	2.4 (6.4)	10.4 (7.9)	2.2 (2.1)	−1.6 (5.6)	6.6 (5.6)	1.7 (1.7)	5.0 (5.6)
p	.030	.017	.010	.116	.040	.044	.306	.150	.015

Level of statistical significance $p < .05$.
[a] Change: difference between the endpoint (adolescence) and the baseline point (preadolescence).
[b] WC: waist circumference.
[c] BMI: body mass index.
[d] %BF: body fat percentage.
[e] SCARED: Screen for Childhood Anxiety and Related Emotional Disorders.
[f] CDI: Children's Depression Inventory.
[g] Depressive symptoms score ≥ 17.
[h] *r*: Pearson coefficient.
[i] Mean (standard deviation).

WC ($B = 7.86$, $p = .017$). With regard to anxiety in girls, model 2 shows that anxiety symptoms led to the increase in WC, BMI and %BF. More specifically, model 3 shows that somatic/panic symptoms contributed to the increase in WC ($B = .34$, $p = .035$) and %BF ($B = .30$, $p = .045$). However, we found no relationship between diagnosis of anxiety disorders and changes in anthropometric and body composition parameters. Anthropometric and body composition variables in preadolescence have a highly significant effect on the same parameters in adolescence ($p < .001$).

Discussion

We observed a relationship between anxiety and depression in preadolescence and increased weight, adiposity and distribution of abdominal fat during adolescence. This relationship was observed in both sexes, although some differences were found according to the type and severity of psychopathology and relations were found predominantly in males.

We found that depressive symptoms led to increased in BMI, WC and %BF in males only. Indeed, although some univariate associations were not observed, multiple regression adjusted for specific risk factors of overweight or obesity enabled us to identify the independent effects of factors such as depression, age, diet and physical activity among others. The relationship between depression and increased adiposity is corroborated in individuals diagnosed with dysthymia but not in those diagnosed with major depression episode. This could be explained by the fact that dysthymia is a chronic disorder whose manifestations affect lifestyle and have long-term health effects. By contrast, a major depressive episode is a much more severe condition and is usually detected much earlier; furthermore, some authors suggest that this disorder may affect eating habits in different ways, leading to different effects on weight status (McElroy et al., 2004; Reeves, Postolache, & Snitker, 2008). As such, the effect of a major depressive episode on weight loss in males observed in our study is supported by previous research (Carpenter et al., 2000). By contrast, we found that dysthymia leads to increased abdominal fat in both the male and female population. These findings are consistent with some research studies of adults with depressive disorder or

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Table 3
Effect of emotional psychopathology in preadolescence on anthropometric and body composition parameters in adolescence in boys.

	Change[d] in WC[e]				Change[d] in BMI[f]				Change[d] in %BF[g]			
	B[h]	SE[i]	*p*	Model	*B*	SE	*p*	Model	*B*	SE	*p*	Model
Model 1: without adjusting for anxiety												
Depressive symptoms[a] (0: no, 1: yes)[j]	4.17	1.50	.007	$R^2_{c.100}$ 20.9	1.27	.50	.013	$R^2_{c.100}$ 11.7	3.44	1.27	.009	$R^2_{c.100}$ 16.6
Initial variable: WC, BMI or %BF	−.03	.83	.640		.04	.06	.518		−.23	.08	.007	
Age (years)	−1.39	.63	.033	F^6_{78} 4.174	−.23	.21	.279	F^6_{87} 2.766	−.68	.57	.239	F^6_{79} 3.430
Dietary quality (score)	−.68	.24	.008		−.19	.83	.023		−.18	.21	.376	
Physical activity (score)	−.36	.26	.181	*p* .001	−.37	.08	.680	*p* .018	−.23	.22	.312	*p* .005
Body satisfaction (score)	.19	.13	.13		.07	.04	.075		.26	.11	.024	
Model 2: without adjusting for depression												
Symptoms of anxiety[b] (score)[j]	.60	.04	.206	$R^2_{c.100}$ 13.8	.002	.01	.907	$R^2_{c.100}$ 4.0	.02	.04	.542	$R^2_{c.100}$ 8.1
Variable: Initial WC, BMI or %BF	−.06	.08	.467		.04	.68	.553		−.23	.08	.008	
Age (years)	−1.36	.66	.045	F^6_{78} 2.925	−.24	.22	.279	F^6_{87} 1.56	−.66	.60	.274	F^6_{79} 2.07
Dietary quality (score)	−.65	.25	.014		−.18	.08	.031		−.17	.22	.428	
Physical activity (score)	−.47	.27	.089	*p* .014	.06	.09	.481	*p* .171	−.31	.23	.190	*p* .068
Body satisfaction (score)	.10	.13	.415		.04	.04	.307		.17	.11	.141	
Model 3: complete – adjusted												
Separation anxiety[b] (score)	.43	.15	.006		.10	.51	.041		.12	.13	.382	
Generalized anxiety[b] (score)	−.29	.21	.190		−.98	.07	.165		−.03	.19	.880	
Somatic panic[b] (score)	−.15	.15	.306	$R^2_{c.100}$ 26.4	−.02	.05	.573	$R^2_{c.100}$ 26.4	−.11	.13	.404	$R^2_{c.100}$ 13.1
Social phobia[b] (score)	−.09	.20	.634	F^{11}_{78} 3.576	−.09	.06	.164		−.05	.18	.759	
Depressive symptoms[a] (0: no, 1: yes)	3.5	1.61	.029		1.25	.53	.022	F^{11}_{87} 3.576	3.32	1.44	.024	F^{11}_{79} 2.099
Initial variable: WC, BMI or %BF	−.03	.08	.662		.02	.06	.660		−.23	.85	.008	
Age (years)	−1.14	.63	.77	*p* .001	−.15	.21	.481		−.66	.60	.275	
Dietary quality (score)	−.68	.24	.006		−.18	.08	.023	*p* .001	−.19	.21	.372	*p* .038
Physical activity (score)	−.28	.26	.281		−.02	.08	.764		−020	.23	.386	
Body satisfaction (score)	.18	.12	.148		.072	.04	.091		.24	.11	.038	
Model 4: complete – adjusted												
Diagnosis of separation anxiety disorder[c] (0: no, 1: yes)	1.54	2.64	.560		−.60	.96	.531		−.95	2.70	.724	
Diagnosis of generalized anxiety disorder[c] (0: no, 1: yes)	−.47	2.01	.813	$R^2_{c.100}$ 37.5	−.14	.67	.828	$R^2_{c.100}$ 23.7	1.04	2.02	.608	$R^2_{c.100}$ 8.4
Diagnosis of panic disorder[c] (0: no, 1: yes)	5.56	3.82	.151		2.83	1.37	.043		4.33	3.80	.260	F^7_{79} 1.607
Diagnosis of social phobia[c] (0: no, 1: yes)	9.59	3.35	.006	F^7_{78} 4.933	2.90	1.21	.019	F^7_{87} 3.253	5.92	3.38	.085	
Diagnosis of major depressive episode[c] (0: no, 1: yes)	−6.55	3.51	.067	$p < .001$	−2.98	1.25	.020	*p* .001	−.30	3.56	.933	*p* .119
Diagnosis of dysthymia[c] (0: no, 1: yes)	9.25	2.62	.001		3.50	.95	<.001		1.48	2.67	.580	
Initial variable: WC, BMI or %BF	−.10	.07	.178		−.03	.06	.596		−.26	.09	.006	
Age (years)	−.77	.58	.186		−.10	.20	.616		−.48	.61	.426	
Dietary quality (score)	−.49	.23	.037		−.16	.08	.041		−.12	.23	.591	
Physical activity (score)	−.52	.24	.033		−.07	.08	.389		−.32	.24	.188	
Body satisfaction (score)	.14	.11	.205		.05	.04	.166		.19	.11	.106	

[a] Depressive symptoms measured by the Children's Depression Inventory.
[b] Anxiety symptoms measured by the Screen for Childhood Anxiety and Related Emotional Disorders.
[c] Anxiety and depression disorder diagnosis determined by the MINI-Kid interview.
[d] Change: difference between the endpoint (adolescence) and the baseline point (preadolescence).
[e] WC: waist circumference.
[f] BMI: body mass index.
[g] %BF: body fat percentage.
[h] *B*: unstandardized coefficient.
[i] SE: standard error. Level of statistical significance $p < .05$.
[j] Multiple linear regression adjusted for: initial WC, initial BMI or initial %BF (according to outcome variable), age, quality of diet measured by the Krece Plus test, physical activity measured by the Krece Plus physical activity questionnaire and body satisfaction according to the Body Areas Satisfaction Scale.

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

Table 4
Effect of emotional psychopathology in preadolescence on anthropometric and body composition parameters in adolescence in girls.

	Change[d] in WC[e]				Change[d] in BMI[f]				Change[d] in %BF[g]			
	B[h]	SE[i]	*p*	Model	*B*	SE	*p*	Model	*B*	SE	*p*	Model
Model 1: without adjusting for anxiety												
Depressive symptoms[a] (0: no, 1: yes)[j]	1.08	1.24	.382	$R^2_{c.100}$ 10.7	.53	.39	.117	$R^2_{c.100}$ 8.2	2.23	1.14	.053	$R^2_{c.100}$ 22.0
Initial variable: WC, BMI or %BF	−.24	.06	<.001		−.17	.04	<.001		−.30	.05	<.001	
Age (years)	−.28	.55	.601	F^6_{134} 3.557	.11	.17	.914	F^6_{142} 3.038	.83	.48	.092	F^6_{134} 7.032
Dietary quality (score)	.18	.21	.400		.02	.06	.720		.17	.19	.384	
Physical activity (score)	−.10	.22	.652	*p* .003	.008	.07	.914	*p* .008	−.15	.20	.446	$p < .001$
Body satisfaction (score)	.11	.10	.262		.03	.03	.290		.08	.09	.407	
Model 2: without adjusting for depression												
Symptoms of anxiety[b] (score)[j]	.13	.05	.012	$R^2_{c.100}$ 14.70	.03	.01	.026	$R^2_{c.100}$ 10.40	.09	.04	.050	$R^2_{c.100}$ 22.00
Initial variable: WC, BMI or %BF	−.24	.06	<.001		−.17	.04	<.001		−.30	.05	<.001	
Age (years)	−.28	.53	.592	F^6_{134} 4.686	.13	.17	.426	F^6_{142} 3.643	.84	.48	.086	F^6_{134} 7.026
Dietary quality (score)	.21	.21	.312		.03	.06	.603		.21	.19	.272	
Physical activity (score)	−.12	.21	.552	$p < .001$	−.005	.06	.946	*p* .002	−.21	.20	.290	$p < .001$
Body satisfaction (score)	.17	.10	.100		.05	.03	.144		.10	.09	.298	
Model 3: complete – adjusted												
Separation anxiety[b] (score)	.24	.14	.101		.08	.04	.102		.12	.13	.370	
Generalized anxiety[b] (score)	−.17	.17	.310		−.03	.05	.522		−.02	.16	.876	
Somatic panic[b] (score)	.34	.16	.035		.06	.05	.240		.30	.15	.045	
Social phobia[b] (score)	.05	.17	.777	$R^2_{c.100}$ 16.10	.004	.05	.944	$R^2_{c.100}$ 10.0	−.17	.16	.309	$R^2_{c.100}$ 24.30
Depressive symptoms[a] (0: no, 1: yes)	−.21	1.27	.867		.18	.41	.651		1.31	1.19	.274	
Initial variable: WC, BMI or %BF	−.23	.06	<.001		−.16	.04	.001		−.29	.05	<.001	
Age (years)	−.10	.55	.844	F^{11}_{134} 3.450	.14	.17	.408	F^{11}_{142} 2.519	.93	.49	.063	F^{11}_{134} 5.103
Dietary quality (score)	.22	.21	.290		.03	.06	.602		.18	.19	.356	
Physical activity (score)	−.11	.22	.606	*p* .001	.002	.07	.975	*p* .009	−.17	.20	.386	$p < .001$
Body satisfaction (score)	.16	.10	.125		.04	.03	.162		.10	.09	.289	
Model 4: complete – adjusted												
Diagnosis of separation anxiety disorder[c] (0: no, 1: yes)	−.05	2.13	.980		−.58	.71	.414		−2.34	2.01	.247	
Diagnosis of generalized anxiety disorder[c] (0: no, 1: yes)	1.04	1.54	.502		−.001	.52	.998		1.31	1.46	.370	
Diagnosis of panic disorder[c] (0: no, 1: yes)	.58	3.88	.988	$R^2_{c.100}$ 14.2	.39	1.31	.763	$R^2_{c.100}$ 6.9	3.09	3.68	.403	$R^2_{c.100}$ 20.7
Diagnosis of social phobia[c](0: no, 1: yes)	−.22	2.10	.914		−.14	.70	.834		−2.13	1.99	.286	
Diagnosis of major depressive episode[c] (0: no, 1: yes)	−1.61	4.99	.747	F^{11}_{134} 2.930	−.03	1.68	.931	F^{11}_{134} 1.916	−3.12	4.71	.509	F^{11}_{134} 4.030
Diagnosis of dysthymia[c] (0: no, 1: yes)	7.86	3.23	.017	*p* .002	1.93	1.09	.079	*p* .043	5.47	3.05	.076	$p < .001$
Initial variable: WC, BMI or %BF	−.26	.06	<.001		−.19	.049	<.001		−.32	.057	<.001	
Age (years)	−.35	.54	.523		.09	.07	.600		.74	.50	.143	
Dietary quality (score)	.23	.21	.279		.02	.07	.685		.25	.20	.209	
Physical activity (score)	−.14	.22	.521		−.01	.07	.890		−.25	.20	.224	
Body satisfaction (score)	.16	.10	.124		.03	.03	.248		.09	.09	.359	

[a] Depressive symptoms measured by the Children's Depression Inventory.
[b] Anxiety symptoms measured by the Screen for Childhood Anxiety and Related Emotional Disorders.
[c] Anxiety and depression disorder diagnosis determined by the MINI-Kid interview.
[d] Change: difference between the endpoint (adolescence) and the baseline point (preadolescence).
[e] WC: waist circumference.
[f] BMI: body mass index.
[g] %BF: body fat percentage.
[h] *B*: unstandardized coefficient.
[i] SE: standard error. Level of statistical significance $p < .05$.
[j] Multiple linear regression adjusted for: initial WC, initial BMI or initial %BF (according to outcome variable), age, quality of diet measured by the Krece Plus test, physical activity measured by the Krece Plus physical activity questionnaire and body satisfaction according to the Body Areas Satisfaction Scale (BASS).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

depressive symptoms (Ahlberg et al., 2002; Needham et al., 2010; Zhao et al., 2011). In this regard, a review in adults showed that depression may be associated with abdominal obesity in both men and women (McElroy et al., 2004). In children and adolescents, a relationship has only been observed between depression and BMI (Anderson et al., 2006, 2010; Goodman & Whitaker, 2002; Rofey et al., 2009) and between depression and %BF in the specific case of adolescent girls (Hillman et al., 2010). However, the results of Tanofsky-Kraff et al. (2006) for a sample of 146 American infants did not show greater increases in %BF, measured by dual energy X-ray absorptiometry, in subjects with depression. Despite the evidence described above, and in contrast to studies that indicate a relationship between depression and obesity primarily among the female population, our study shows that the relationship is stronger in boys than in girls. The differences between boys and girls could be explained by the findings of recent studies that applied a novel statistical approach based on spline function, in which it was found that the association between depression and BMI *z* score was non-linear and that the shape of the curve obtained varied according to gender (Cortese et al., 2009; Revah-Levy et al., 2011). Another study using the same analytical methodology showed that the relationship between BMI and body dissatisfaction was also different for boys and girls (Cortese et al., 2010). This fact may modulate the relationship between depression and BMI according to gender.

We found that anxiety leads to increased anthropometric and body composition parameters, with differences observed according to sex and the type and severity of anxiety. Thus, although we found that the total anxiety score was related to an increase in WC, BMI, and %BF in girls, detailed analysis showed that only somatic/panic manifestations were related. In this respect, our results agree with those of Hillman et al. (2010), who associated anxiety symptoms with %BF measured using dual energy X-ray absorptiometry in a population of 198 female adolescents in the United States. However, Hillman et al. (2010) and Midei and Matthews (2009), who used the waist–hip-ratio in both genders, did not observe a significant relationship between anxiety and abdominal fat.

Unlike girls, the boys with higher scores for separation anxiety showed a greater increase in WC and BMI. This increase in adiposity was also found in boys diagnosed with social phobia and the increase in BMI in boys diagnosed with panic disorder. It is difficult to find the reasons for these differences according to type and severity of anxiety. To our knowledge there are no studies of children or adolescents that analyze the different subtypes of anxiety. One possible explanation is the method used to assess anxiety. The symptoms identified by SCARED are quantitative measures; however, the diagnosis obtained by MINI-Kid is a dichotomous variable and the level of the disorder that it establishes takes into account a minimum number of criteria from the DSM-IV-TR and clinical interference. Social phobia disorder causes limitations, major subjective discomfort and social isolation. Therefore, adolescents with this disorder usually stay at home more, eat more, are more inactive, and do not participate social activities and sports. Similarly, panic disorder can lead to avoidance behaviors such as not leaving home in order to avoid a stressful situation. Therefore, adolescents with this disorder may be more inactive or eat more to reduce anxiety manifestations.

However, it is difficult to explain why some of these relationships were observed in boys but not in girls in our study, in contrast with several studies conducted with adolescents in which the relationship between anxiety and obesity appeared to be more evident in the female subjects (Anderson et al., 2006). Our results show a consistent relationship between anxiety and WC for both genders, similar to the results of other authors who observed the same relationship with abdominal fat in adults (Ahlberg et al., 2002; Needham et al., 2010; Zhao et al., 2011). Likewise, Rofey et al. (2009) observed weight gain in both boys and girls with anxiety.

In general, the differences in the observed effects of anxiety and depression on adiposity may be due in part to differences in the study design, such as the age range considered and the methodology used to assess psychological disorders and to determine weight, fat and fat distribution (Incledon et al., 2011).

Additionally, our results show that greater baseline anthropometric and body composition measurements influence the change in anthropometric and body composition measurements in adolescent girls but not in adolescent boys. We are unsure of the reasons for these results, although one possible explanation would be the difference in age at onset of puberty between the genders. Girls in the age range considered in the study are likely to be in mid-puberty, whereas boys in the same age range are more likely to be at the onset of puberty. In prepubertal boys, changes in body composition due to puberty are minimal, and the prepubertal weight and fat distribution may not be critical to the future development of these parameters. By contrast, in girls of the same age, changes in body composition due to puberty have just begun and their bodies are being modified and defined. Therefore, the development of body composition in mid-puberty may influence the subsequent progression of body fat and fat distribution. In addition, mid-pubertal girls are at the stage of becoming concerned about their weight, and many of them want to be thinner. Consequently, girls with higher anthropometric and body composition parameter values make a conscious effort not to gain weight or fat.

However, our results reveal inconsistencies in %BF measured by BIA. Although some studies support the use of this method among children, others argue that it has limitations at critical stages of development, does not detect small changes with sufficient accuracy, and shows varying validity according to adiposity (Eisenmann, Heelan, & Welk., 2004; Goldfield et al., 2006; Treuth, Butte, Wong, & Ellis, 2001). In this case, BMI and, in particular, WC may reflect changes in adiposity more accurately. Our findings on psychopathology and increased WC could support the results of Ahlberg et al. (2002), which indicate that psychopathology is more closely related to abdominal fat reserves than obesity per se. Furthermore, assessment of WC is important because it is a diagnostic criterion for metabolic syndrome (Varda & Gregoric, 2009). In isolation, some research studies in adults suggest that depression and/or anxiety predict an increased risk of metabolic syndrome and cardiovascular diseases (Goldbacher & Matthews, 2007; Luppino et al., 2011). In the same vein, a recent review in children studied the relationship between chronic stress and metabolic syndrome (Pervanidou & Chrousos, 2011).

UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
Estefania Aparicio Llopis
Dipòsit Legal: T 1593-2015

There are various interpretations of these findings. On the one hand, the psychopathology may lead to changes in eating behavior and lifestyle (Reeves et al., 2008). It has been shown that a substantial proportion of people with depressive and anxiety symptoms have increased appetites and tend to overeat and reduce their levels of physical activity, leading to weight gain (McElroy et al., 2004). On the other hand, there is evidence of a shared neurobiological mechanism between emotional psychopathology and weight gain. The emotional psychopathology affects the hypothalamic–pituitary–adrenal axis, leading to increased cortisol secretion. High cortisol levels are associated with obesity, especially abdominal obesity (Pervanidou & Chrousos, 2011; Reeves et al., 2008). This mechanism could account for the consistent observation of a relationship between emotional psychopathology and increased WC in both sexes in our study. The existence of a common genetic foundation has also been suggested (Wermter et al., 2010).

This study has a number of strengths. First, the prospective design in a non-clinical population enabled us to use a sample of schoolchildren at risk of emotional psychopathology and a group of control subjects. Second, by using a three-year follow-up period we were able to assess the effect of the psychopathology on the increase in adiposity from preadolescence to adolescence. Third, we not only evaluated emotional symptoms but also diagnosed the underlying emotional disorder on an individual basis according to standardized clinical criteria (DSM-IV-TR). We thus obtained diagnoses of the different anxiety and depression disorders present in the study population and were able to specifically assess the predictive ability of each one. However, due to the high level of comorbidity between depression and anxiety and between the different types of disorders (Essau, 2008; Polaino-Lorente, Canals, & Domènech-Llaberia, 2002) we adjusted our statistical analyses for all of these variables. Fourth, the anthropometric variables were measured by qualified personnel using a standardized methodology. The direct determination of weight and height gives our results greater precision and validity (Incledon et al., 2011; Rhew et al., 2008). Furthermore, most of the studies in the literature only consider BMI, yet this index does not provide scope for analysis of %BF or its distribution. The use of other measures that assess %BF, such as BIA, and abdominal fat distribution, such as WC, is therefore necessary. Both methods are simple, economical, fast and feasible at the population level. By contrast, other more sophisticated methods such as computed tomography or dual energy X-ray absorptiometry are more costly, more time-consuming and more difficult to implement.

Our study has certain limitations that should be considered when interpreting the results, including the limited sample size and follow-up rate, and the non-inclusion of other confounding variables such as ethnicity, pubertal stage, maternal obesity and maternal depression, among others.

Future research should therefore aim to elucidate the interrelationship between depression–anxiety and obesity and/or metabolic syndrome in terms of behavior, neurobiology and genetics, especially among children and young people, and using various measures of adiposity.

In light of the evidence presented above, to our knowledge this is one of the first data sets for a preadolescent population that describes the influence of depression (and specifically dysthymia) and the various types of anxiety according to DSM-IV on increased WC in adolescents. Depression and anxiety during childhood are common, treatable conditions, and as such, these findings may have significant implications for the prevention and treatment of obesity and metabolic syndrome. In addition, WC is a simple and economical measure that can be used at community and school level, in prevention programs and in clinical settings, enabling rapid monitoring of children with psychopathology to identify weight problems before they become pathological.

In conclusion, emotional psychopathology in preadolescence is associated with increased weight gain and abdominal fat in adolescence, albeit with some differences in the precise relationship with each anxiety and depression disorder according to gender. These factors could lead to disorders such as obesity or metabolic syndrome. Future research should seek to confirm these results and examine the possible mechanisms involved.

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Dipòsit Legal: T 1593-2015

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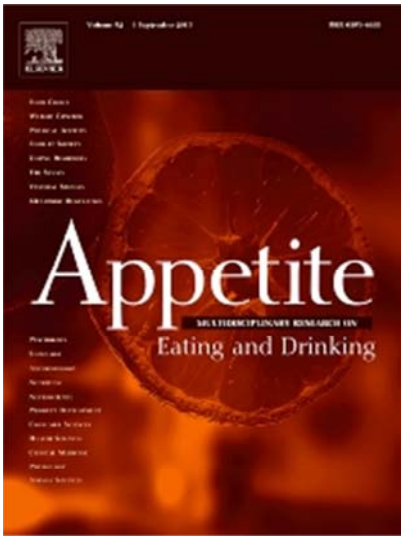
Do emotional symptoms affect dietary patterns in early adolescence? A school-based follow-up study



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Abstract: Introduction: Although stress could causes overeating and changes food choice, the relation between emotional symptoms and dietary pattern is less clear, especially in population-based studies during early adolescence. Our aim is to examine the prospective relationship, according to gender, between emotional symptoms and dietary patterns in a school-based sample followed for 3 years in early adolescence.
Methods: From a baseline sample of 1,514 adolescents, 165 were followed up over three years. Depression and anxiety symptoms were assessed at baseline and after one year and three years. The participants were classified as showing emotional symptoms in any of the phases (n=100) or in the control group (n=65). In the third year, food consumption was recorded and dietary patterns were created by principal component analysis. Tests of quality of diet (Mediterranean diet) and physical activity were administered.
Results: Girls with emotional symptoms scored significantly lower in the Mediterranean diet and physical activity tests than the control group. They presented a high consumption of sweet dairy desserts and sweets, and 39.7% of them showed high adherence to a sweet and fat dietary pattern. After adjusted logistic regression, girls with emotional symptoms were four times as likely to have a high adherence to a sweet and fatty food dietary pattern (OR: 4.79, IC (1.55-15.10). No differences were observed among boys.
Conclusion: Girls with emotional symptoms during early adolescence present a high adherence to a dietary pattern rich in sweet and fat foods, and engage in low levels of physical activity, while there are no differences among adolescent boys. These findings highlight the need to add negative emotion management to obesity and obesity-related diseases prevention programs.

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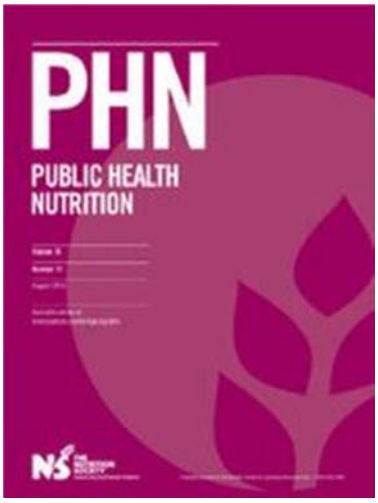
Longitudinal study of psychopathological, anthropometric and socio-demographic factors related to the level of Mediterranean diet adherence in a community sample of Spanish Adolescents



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Longitudinal study of psychopathological, anthropometric and socio-demographic factors related to the level of Mediterranean diet adherence in a community sample of Spanish Adolescents

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Longitudinal study of psychopathological, anthropometric and socio-demographic factors related to the level of Mediterranean diet adherence in a community sample of Spanish Adolescents

Objective The Mediterranean diet (MD) pattern has been shown to have important health benefits, although it seems that in recent years that Spanish school-age child have been abandoning this healthy pattern. The main aim of the study was to identify psychopathological, anthropometric and socio-demographic factors which may influence the risk of low MD adherence.

Design A longitudinal study in three phases. Adherence to the MD was assessed by the *Krece plus food questionnaire* and psychopathological symptoms by the *Screen for Childhood Anxiety and Related Emotional Disorders, Children's Depression Inventory, Youth Inventory-4 and* the *Eating Disorder Inventory-2*. Anthropometric data were collected in the first and third phase.

Settings All 5 representative areas of Reus (Catalonia, Spain).

Subjects 241 adolescents from 13 schools of Reus.

Results Results showed that regardless of past and current Body Mass Index (BMI), socioeconomic status (SES) was a protective factor for low MD adherence (OR = .805, p = .003) and a risk factor for high BMI (OR = .718, p = .002; OR = .707, p = .001). Regardless of SES, depression was involved with risk for low adherence (OR = 1.069, p = .021). Girls with low MD adherence presented significant higher scores for eating disorders and depression symptoms than girls with a high adherence.

Conclusions The results highlight the influence of psychosocial factors on the MD adherence level. Taking into account these factors is important when carrying out prevention and health promotion initiatives.

Keywords Mediterranean diet, adolescents, psychopathology, risk factors

For Peer Review

The role of emotion regulation in childhood obesity: Implications for prevention and treatment

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UNIVERSITAT ROVIRA I VIRGILI
THE EFFECT OF EMOTIONAL AND GENETIC FACTORS ON NUTRITIONAL STATUS IN A SCHOOL-BASED POPULATION.
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The role of emotion regulation in childhood obesity: Implications for prevention and treatment

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Abstract

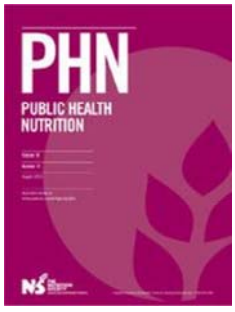
Stress and negative emotions pose a major threat to public health, by increasing the risk of obesity. Since the management process for emotions (emotion regulation, ER) is developed in childhood, we present a novel conceptual framework model for the role of ER in the prevention and treatment of childhood obesity. Narrative review of the literature by electronic database search (MEDLINE, Web of Knowledge and Scopus) was conducted of observational and interventional/experimental literature on ER and obesity and the underlying concepts. We also present an overview of ER intervention techniques. Our model indicates that childhood ER is a link between stress and obesity. Stress along with ineffective ER leads to abnormal cortisol patterns, emotional eating, sedentary lifestyle, reduction of physical activity, and sleep problems. Simultaneously, a healthy lifestyle could show benefits on ER and in developing adaptive ER strategies. In the development of obesity and ER, parents also play a role. By contrast, effective ER skills decrease obesity-related unhealthy behaviour and enhance protective factors, which boost health. The literature contains some observational studies of children but very few intervention studies, most of which are pilot or on-going studies. In conclusion, encouraging effective ER could be a useful new approach for combating and treating childhood obesity. Future ER intervention studies are needed to confirm the validity of this model in children.

For Peer Review

2. OTHER SCIENTIC CONTRIBUTIONS

2.1 JOURNAL PUBLICATIONS

Aparicio-llopis E, Canals J, Arija V. Dietary Intake According to the Course of Symptoms of Eating Disorders in a School-based Follow-up Study of Adolescents. *European Eating Disorders Review 2014. 22(6): 412-422*
Published



Aparicio E, Canals J, Pérez S, Arija V. Dietary intake and nutritional risk in Mediterranean adolescent in relation to the severity of the eating disorder. *Public Health Nutrition* 2015. 18(8): 1461-1473.
Published

Aparicio E, Canals J, Arija V. Predictors of and factors associated with the persistence of eating disorders in adolescent girls from a school-based sample: a three-year longitudinal study. *The journal of Early Adolescence 2015.*
Submitted

2.2 CONGRESSES

Aparicio E, Canals J, Voltas N, Hernández-Martínez C, Arija V**.** Psicopatología emocional e incremento de adiposidad. Estudio longitudinal en escolares. IX Congreso de la Sociedad Española de Nutrición Comunitaria. Cádiz, 2012.
Participation: Oral communication

Voltas N, Hernández-Martínez C, **Aparicio E,** Arija V, Canals J. Estudio prospectivo de los síntomas obsesivos compulsivos en escolares**.** 57º Congreso de la Asociación Española de psiquiatría del niño y del adolescente. Barcelona, 2012.
Participation: Poster

Aparicio E, Canals J, Domènech-Llaberia E, Voltas N, Hernández-Martínez C, Arija V. Els símptomes somàtics poden contribuir a l'increment de pes en adolescents. Estudi longitudinal**.** III Jornada de recerca en Salut Pública. Barcelona, 2013.
Participation: Poster.

Aparicio E, Canals J, Voltas N, Hernández-Martínez C, Arija V. Does depression increase the risk of overweight in adolescents? Longitudinal study. 1st World Forum for Nutrition Research Conference: Mediterranean Food on Health and Disease. Reus, 2013
Participation: Poster

Voltas N, Hernández-Martínez C, **Aparicio E**, Arija V, Canals J. Longitudinal study of the course of anxiety symptomatology in a Spanish sample. 15th International Congress of ESCAP. Dublin (Ireland), 2013.
Participation: Poster

Voltas N, Hernández-Martínez C, **Aparicio E**, Arija V, Canals J. Factores psicopatológicos asociados al rendimiento académico en el inicio de la adolescencia: estudio prospectivo de tres fases. 59º Congreso de la Asociación Española de psiquiatría del niño y del adolescente. Santander, 2014.
Participation: Poster

Aparicio E, Canals J, Voltas N, Hernández-Martínez C, Arija V**.** Does anxiety affect on diet quality in adolescents?**.** 2nd International Conference on Nutrition and Growth. Barcelona, 2014.
Participation: Poster.

Arija V, **Aparicio E**, Voltas N, Canals J**.** Does depression affect on diet quality in adolescents? III world Congress of public Health Nutrition, II Latin American congress of comunity nutrition, X congreso de la sociedad española de nutrición comunitaria (SENC). Barcelona, 2014
Participation: Poster.

Aparicio E, Canals J, Arija V, De Henauw S, Michels N. The role of emotional regulation in childhood obesity: implications for prevention and treatment. 22nd European Congress on Obesity. Praga, 2015.
Participation: Poster.

2.3 AWARDS

Oral communication award:

Aparicio E, Canals J, Voltas N, Hernández-Martínez C, Arija V. Psicopatología emocional e incremento de adiposidad. Estudio longitudinal en escolares. Congreso: IX Congreso de la Sociedad Española de Nutrición Comunitaria. Cádiz, 2012

Poster award:

Voltas N, Hernández-Martínez C, **Aparicio E**, Arija V, Canals J. Factores psicopatológicos asociados al rendimiento académico en el inicio de la adolescencia: estudio prospectivo de tres fases. 59º Congreso de la Asociación Española de psiquiatría del niño y del adolescente. Santander, 2014.

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