DIET QUALITY, EATING BEHAVIORS, OTHER LIFESTYLE FACTORS AND CARDIOMETABOLIC RISK IN CHILDREN

Tany Elizabeth Garcidueñas Fimbres

UNIVERSITAT ROVIRA i VIRGILI

Diet quality, eating behaviors, other lifestyle factors and cardiometabolic risk in children

Tany Elizabeth Garcidueñas Fimbres

DOCTORAL THESIS
2023

Tany E. Garcidueñas Fimbres

DIET QUALITY, EATING BEHAVIORS, OTHER LIFESTYLE FACTORS AND CARDIOMETABOLIC RISK IN CHILDREN

DOCTORAL THESIS

This thesis has been supervised by Dr. Nancy Babio,

and Dr. Jordi Salas Salvadó

Human Nutrition Unit

Department of Biochemistry and Biotechnology

Universitat Rovira i Virgili

Reus, Tarragona

2023

I STATE:

That the present study entitled "**Diet quality, eating behaviors, other lifestyle factors and cardiometabolic risk in children**" is presented by *Tany Elizabeth Garcidueñas Fimbres* to obtain the degree of Doctor and has been accomplished under my supervision at the Department of Biochemistry and Biotechnology of the Universitat Rovira i Virgili.

Reus, 16 de mayo de 2023

Doctoral Thesis Supervisor

Dr. Nancy Elvira Babio Sánchez

Head of Nutrition and Dietetics Degree, Universitat Rovira i Virgili.
Associated Professor at the Department of Biochemistry and Biotechnology, Universitat Rovira i Virgili.

I STATE:

That the present study entitled "**Diet quality, eating behaviors, other lifestyle factors and cardiometabolic risk in children**" is presented by *Tany Elizabeth Garcidueñas Fimbres* to obtain the degree of Doctor and has been accomplished under my supervision at the Department of Biochemistry and Biotechnology of the Universitat Rovira i Virgili.

Reus, 16 de mayo de 2023

Doctoral Thesis Supervisor

Dr. Jordi Salas Salvadó

Distinguished Professor of Nutrition and Food Science, Universitat Rovira i Virgili.
Director of the Human Nutrition Unit of the Department of Biochemistry and Biotechnology, Universitat Rovira i Virgili.

The adventure of life is to learn.

The purpose of life is to grow.

The nature of life is to change.

The challenge of life is to overcome.

The essence of life is to care.

The opportunity of life is to serve.

The secret of life is to dare.

The spice of life is to befriend.

The beauty of life is to give".

- William Ward

Table of contents

ACKNOWLEDGMENTS 1

ABSTRACT 13

English 15

Castellano 17

Català 19

ABBREVIATIONS 21

I. INTRODUCTION 25

1. Human growth and development 27

1.1. The preschool-aged children 28

2. Obesity and cardiometabolic risk in childhood: definitions, epidemiology and clinical issues 28

2.1. Obesity 28

2.2. Cardiometabolic risk 30

3. Obesity and cardiometabolic risk in childhood: pathophysiology 31

4. Obesity and cardiometabolic risk in childhood: risk factors 32

4.1. Genetics 33

4.2. Family and early life 34

4.3. Environment 35

4.4. Lifestyle 36

4.5. Diet quality, eating speed and eating frequency 36

4.5.1. First article 36

4.6. Physical activity 67

4.7. Sedentary behaviors 68

4.8. Sleep duration 69
4.9. Lifestyle behaviors scores 70
5. Obesity and cardiometabolic risk in childhood: prevention 72
II. RATIONALE AND JUSTIFICATION 73
III. HYPOTHESIS AND OBJETIVES 77
IV. METHODOLOGY 81
1. Childhood Obesity Risk Assessment Longitudinal Study (CORALS) . 83
1.1. Study Design and Objectives 83
1.2. Study population 84
1.3. Data assessments 84
1.3.1. Early life and maternal factors 84
1.3.2. Diet and nutrients 85
1.3.3. Diet quality 85
1.3.4. Eating Speed 86
1.3.5. Physical activity 86
1.3.6. Sleep 87
1.3.7. Screentime 87
1.3.8. Adiposity 87
1.3.9. Blood pressure 88
1.3.10. Lipids and fasting plasma glucose 88
1.4. Statistical Analyses 88
2. Second article 89
2.1. Selection of participants 89
2.2. Specific data assessments 90
2.2.1. Statistical Analyses 90
3. Third article 90

3.1. Selection of participants 91
3.2. Specific data assessments 91
3.2.1. Composite score comprised of 6 healthy lifestyle behaviors 92
3.2.2. Statistical Analyses 93
V. RESULTS 95
Second article 97
Third article 109
VI. DISCUSSION 159
General discussion 161
Limitations and Strengths 167
VII. CONCLUSIONS 169
English 171
Castellano 173
VIII. FUTURE PERSPECTIVES 177
IX. REFERENCES 183
X. APPENDIXES 225
Scientific contributions 227
Publications derived from the present doctoral thesis 227
Participation in national and international conferences 227
Research stay 228

ACKNOWLEDGMENTS

Agradecimientos

Para mis padres, cuyo gran sacrificio por verme realizar mis sueños y superarme nunca se podrá comparar, por ser mi brújula. Para Vivian, Gary, Dulce y Jessica porque siempre han abrazado mis sueños y ayudado a perseguirlos. Para mis niños porque necesitan recordar siempre que nunca habrá nada que no puedan lograr con voluntad y esfuerzo.

Acknowledgments

English

"We all have an unsuspected reservoir of strength inside that comes to the surface when life tests us"
– Isabel Allende

It will never cease to amaze me the ability of human beings to adapt to almost any situation in which life places us. I believe that success in life depends greatly on this, but also on the people around you and those who you decide to surround yourself with. Another fundamental part of success are the experiences but, even more important, the interpretation you decide to give to them. With all this I am not referring exclusively to success in the professional environment, but to success that includes physical and emotional well-being.

"If I have seen further, it is by standing upon the shoulders of giants"
– Isaac Newton

I am convinced that parents, or those who play this role, are the first fundamental piece in the success of an individual's life and personally I could not be more grateful for my parents. They are the ones who have patiently taught me to fly, encouraged me to fly high and pursue my dreams, even though this meant to contribute to emptying the nest. They help me to understand that life is much

Acknowledgments

more than living in fear, that life is made of struggle, sweat and effort. They have made me see that I also can be giant. When you mature enough you understand that life was wise enough to give you older brothers because sometimes, they teach you how to confront life in such a way that hardly others are able to, thank you from the bottom of my heart. I also thank my sisters from a different mother because their love and support could not be more authentic and like that of a blood sister. Thank you to my children because although they are very little now and do not understand, they represent a constant reason to overcome, in case one day they need an additional example to that of their parents. My gratitude also extends to those friends and family members who have held my hand and to those whose company was temporary but left a print on this process.

"In the midst of every crisis, lies a great opportunity and those who overcome the crisis overcomes themselves without being overcome"
– Albert Einstein

All merit had first great challenges that indeed, are great opportunities. This professional achievement has not been the exception and for this reason my gratitude is to my thesis supervisors Dr. Nancy and Dr. Jordi, who with great patience have guided me through the chaos that my mind can be sometimes. This acknowledgment is also extended to the CORALS staff, as well as to

Acknowledgments

the parents and children who participate in the study because this thesis has been possible thanks to their support, dedication and effort. I also want to express my appreciation to Dr. Catherine Birken, for the opportunity to collaborate in the TARGet Kids! study, and for being together with Xuedi and Charlie, a guide in this learning stage because although it has been a challenging process, their support is felt across the distance.

As aforementioned, people around you greatly contribute to merits and because this, my gratitude extends to all the members of the Human Unit Nutrition and colleagues who sometimes dealt with the biggest chaos in my mind and yet were always willing to help me and solve my endless doubts. After what sometimes seemed like endless hours of work, we learned to be a family, thank you.

"The future belongs to those who believe in the beauty of their dreams"
– Eleanor Roosevelt

Castellano

"Todos tenemos una insospechada reserva de fortaleza por dentro que sale a la superficie cuando la vida nos pone a prueba"
– Isabel Allende

Quizá nunca deje de sorprenderme la capacidad que tiene el ser humano a adaptarse a casi cualquier situación en la que la vida lo sitúa. Considero que gran parte del éxito en la vida depende de esto pero también de las personas que te rodean y de aquellas de las que decides rodearte. Otra parte fundamental son las vivencias pero, aún más importante, la interpretación de decides darle a las mismas. Con todo esto no hago referencia exclusivamente al éxito en el ámbito profesional sino aquel que incluye el bienestar físico y emocional.

"Si he logrado ver lejos, ha sido porque he subido a hombros de gigantes"
– Isaac Newton

Estoy convencida de que los padres o quienes fungen este papel, son la primera pieza fundamental en el éxito de la vida de un individuo y personalmente no puedo estar más agradecida porque no he podido ser más afortunada por mis padres. Son ellos los que con bastante paciencia me enseñaron a volar y me han alentado a volar alto, a perseguir mis sueños. Me ayudaron a entender que la

Agradecimientos

vida es mucho más que vivir con miedo, que está hecha de lucha, sudor y esfuerzo. Me ayudaron a ver que yo también puedo ser un gigante. Cuando maduras lo suficiente entiendes lo sabia que ha sido la vida al darte hermanos mayores porque algunas veces, te enseñan a enfrentar algunos aspectos de la vida de una manera que difícilmente pueden otros, gracias de todo corazón. Agradezco también a mis hermanas de distinta madre porque su amor y apoyo no puede ser más auténtico y parecido al de una hermana de sangre. Gracias a mis niños porque aunque ahora son muy pequeños y no lo comprenden, ellos representan un constante motivo de superación, por si algún día necesitan un ejemplo adicional al de sus padres. Mi agradecimiento se extiende también a todas aquellas amigas, amigos y familiares que me han sostenido la mano y también a quienes cuya compañía ha sido temporal pero que han dejado su huella en este proceso.

"En medio de toda crisis, se encuentra una gran oportunidad y quien supera la crisis se supera a sí mismo"
– Albert Einstein

Todo mérito tuvo primero grandes desafíos que se convirtieron en grandes oportunidades. Este logro profesional no ha sido la excepción y por ello mi agradecimiento es hacia mis supervisores de tesis, la Dra. Nancy y el Dr. Jordi, quienes con mucha paciencia han sabido encaminar el caos que a veces puede ser mi mente. Este agradecimiento se extiende a todo el equipo de trabajo de CORALS,

Agradecimientos

así como a los padres y niños que participan en el estudio porque esta tesis ha podido realizarse gracias a su apoyo, dedicación y esfuerzo. Gracias a la Dra. Catherine Birken por la oportunidad de colaborar en el estudio TARGet Kids!, y también por ser, junto a Xuedi y Charlie, una guía en esta fase de aprendizaje que aunque no ha sido un proceso fácil, su apoyo se siente incluso a la distancia. Como ya he mencionado, son todas aquellas personas con las que diariamente convives las que también tienen un rol importante en alcanzar tus metas y es por dicha razón que mi agradecimiento se extiende a todos los miembros de la Unidad de Nutrición Humana, quienes a veces lidiaron con el mayor caos de mi mente y aun así siempre estuvieron dispuestos a ayudarme y resolver mis infinitas dudas. Porque después de lo que a veces parecían interminables horas de trabajo, aprendido a ser familia, muchas gracias.

"El futuro pertenece a quienes creen en la belleza de sus sueños"
– Eleanor Roosevelt

ABSTRACT

Abstract

English

Childhood obesity is one of the major concerns of Public Health, mainly due to its potential detrimental effects not only in this life stage but also in adulthood, the risk of which has been suggested to increase the earliest is the onset. In Spain, around 41% of children aged 6-9 years had overweight or obesity in 2019 and, at present, is one of the European countries with higher prevalence. Childhood obesity has been related to several comorbidities including cardiometabolic disorders. Etiology of obesity comprises a wide network of factors, which is mainly extended to diet, physical activity, sleep and sedentary behaviors however, other potential causal factors have been suggested recently, such as eating speed, eating frequency and adherence to the Mediterranean diet (MedDiet). Nevertheless, evidence on children is limited, especially regarding associations with cardiometabolic risk factors.

In overall, the present thesis aimed to summarize evidence on the associations between certain eating behaviors such as eating speed and eating frequency, and adiposity, cardiometabolic risk factors, as well as diet quality in children and adults. In addition, the association between eating speed and cardiometabolic risk as well as adherence to the MedDiet was assessed. Additionally, another objective was to create a composite score which assessed the concomitant associations of certain lifestyle behaviors, including adherence to

Abstract

the MedDiet, eating speed, breastfeeding, sleep duration, physical activity and sedentary behaviors with adiposity and several cardiometabolic risk factors in children. The individual contribution of each lifestyle behavior on the associations between the composite score and cardiometabolic outcomes was also assessed for the purposes of the present thesis.

The results of this thesis show that eating speed and adherence to the MedDiet are related to adiposity and the levels of certain cardiometabolic risk factors. Furthermore, although the co-occurrence of the lifestyle behaviors assessed was associated with adiposity and cardiometabolic risk, the individual contribution of each of them shown not to be equal so that eating speed contributes greatly to a beneficial cardiometabolic health, especially for fasting plasma glucose concentration. Meanwhile, adherence to the MedDiet shown to be a great contributor to serum triglycerides levels. Nevertheless, further research is warranted in order to confirm these associations observed, which if confirmed, eating speed, in tandem with adherence to MedDiet, could be considered modifiable eating behaviors with a relevant role in alleviating the consequences of this challenging Public Health issue.

Resumen

Castellano

La obesidad infantil es uno de los mayores problemas de Salud Pública, principalmente por su potencial riesgo a la salud en la infancia y la edad adulta, cuyo riesgo se ha sugerido que aumenta cuanto más precoz es su aparición. En España, alrededor del 41% de los niños de 6 a 9 años presentaron sobrepeso u obesidad en 2019. La obesidad infantil se ha relacionado con algunas comorbilidades, incluyendo los trastornos cardiometabólicos. La etiología de la obesidad comprende una amplia red de factores que incluye la dieta, la actividad física, el sueño y las conductas sedentarias; sin embargo, recientemente se han sugerido otros posibles factores causales como la velocidad de ingesta alimentaria, la frecuencia de las comidas y la adherencia a la dieta Mediterránea. No obstante, la evidencia en niños es limitada, especialmente en lo que respecta a las asociaciones con factores de riesgo cardiometabólico.

El objetivo de la presente tesis fue la de resumir la evidencia que existe sobre las asociaciones entre algunos comportamientos alimentarios, tales como la velocidad y frecuencia de ingesta, y la adiposidad, el riesgo cardiometabólico y la calidad de la dieta en niños y adultos. También se evaluó la asociación entre la velocidad de ingesta alimentaria y adiposidad, factores de riesgo cardiometabólico y la adherencia a la dieta Mediterránea. Además, otro objetivo fue el de crear un sistema de puntuación para explorar

Resumen

las asociaciones conjuntas de la adherencia a la MedDiet, la velocidad de ingesta alimentaria, la lactancia materna, la duración del sueño, la actividad física y las conductas sedentarias con la adiposidad y los factores de riesgo cardiometabólico en niños. La contribución individual de cada uno de estos comportamientos de estilo de vida en estas asociaciones también fue evaluada para los objetivos de la presente tesis.

Los resultados de esta tesis muestran que la velocidad de ingesta alimentaria y la adherencia a la MedDiet están relacionadas con la adiposidad y con algunos factores de riesgo cardiometabólico. Además, aunque la co-ocurrencia de los comportamientos de estilo de vida explorados se asoció con la adiposidad y el riesgo cardiometabólico, la contribución individual de cada uno de ellos mostró no ser igual, de modo que la velocidad de alimentación contribuyó en gran medida a una mejor salud cardiometabólica, especialmente con respecto a la concentración de glucosa plasmática en ayunas. Mientras tanto, la adherencia a la MedDiet mostró ser un gran contribuyente en los niveles séricos de triglicéridos. No obstante, es necesario seguir investigando para confirmar las asociaciones observadas, que de confirmarse, la velocidad de ingesta alimentaria así como la adherencia a la dieta Mediterránea podrían considerarse factores potencialmente modificables con un papel relevante en la disminución de las consecuencias de este difícil problema de salud pública.

Resum

Català

L'obesitat infantil és un important problema de Salut Pública, principalment pels seus potencials efectes perjudicials no sols en la infància sinó també en l'edat adulta, el risc de la qual ha augmentat com més precoç és la seva aparició. A Espanya, al voltant del 41% dels nens de 6 a 9 anys presentaven sobrepès o obesitat l'any 2019. L'obesitat infantil s'ha relacionat amb algunes comorbiditats, entre les quals s'inclouen els trastorns cardiometabòlics. L'etiologia de l'obesitat comprèn una àmplia xarxa de factors com la dieta, l'activitat física, el temps de son i les conductes sedentàries. Malgrat això, recentment s'han suggerit altres potencials factors causals de l'obesitat, com la velocitat d'ingesta, la freqüència dels àpats i l'adherència a la dieta mediterrània. Malgrat tot, l'evidència en poblacions de nens és limitada, especialment pel que fa a les associacions amb factors de risc cardiometabòlic. Un dels objectius de la present tesi és la de resumir l'evidència que existeix sobre les associacions entre alguns comportaments alimentaris, com ara la velocitat i freqüència d'ingesta, i l'adipositat, el risc cardiometabòlic i la qualitat de la dieta en nens i adults. També, es pretén avaluar l'associació entre la velocitat d'ingesta i adipositat, factors de risc cardiometabòlic i l'adherència a la dieta Mediterrània. A més, un altre dels objectius ha estat el d'explorar les associacions individuals i conjuntes entre l'adherència a la dieta mediterrània, la velocitat de

Resum

la ingesta, els antecedents de lactància materna, el tems de son, l'activitat física i les conductes sedentàries, i l'adiposita o diferents factors de risc cardiometabòlic en nens.

Els resultats d'aquesta tesi mostren que la velocitat d'ingesta i l'adherència a la dieta mediterrània estan relacionades amb l'adiposita i els nivells d'alguns factors de risc cardiometabòlic. A més, encara que la co-ocurrència dels comportaments d'estil de vida avaluats s'associen amb l'adiposita i el risc cardiometabòlic, la contribució individual de cadascun d'ells ha mostrat no ser igual. De manera que la velocitat en el menjar contribueix en gran manera a una bona salut cardiometabòlica, especialment relacionada amb les concentracions plasmàtiques de glucosa en dejú. D'altra part, l'adherència a la dieta mediterrània va mostrar ser una gran predictora dels nivells sèrics de triglicèrids. Malgrat tot, és necessari continuar investigant per a poder confirmar aquestes associacions. En cas de confirmar-se, la velocitat d'ingesta conjuntament amb l'adherència a la dieta Mediterrània, podrien considerar-se comportaments potencialment modificables amb un paper rellevant per pal·liar les conseqüències d'aquest difícil problema de salut pública.

ABBREVIATIONS

Abbreviations

AHA	American Heart Association
ANOVA	Analysis of variance
BMI	Body mass index
CEBQ	Child Eating Behavior Questionnaire
CESNID	Centre d'Enseyament Superior de Nutrició i Dietética
CHLS	Child healthy lifestyle score
CI	Confidence interval
CORALS	Childhood Obesity Risk Assessment Longitudinal Study
FMI	Fat mass index
HDL-c	High-density lipoprotein cholesterol
IQR	Interquartile range
LDL-c	Low-density lipoprotein cholesterol
MedDiet	Mediterranean diet
MetS	Metabolic syndrome
Non-HDL-c	Non-high-density lipoprotein cholesterol
OR	Odds ratio
SD	Standard deviation
WHO	World Health Organization

I. INTRODUCTION

1. Human growth and development

Human growth and development usually require about 21 years to be completed[1], during which time future health is established[2]. Although, the growth velocity varies according to the different stages of life[3], growth is a constant process and corresponds to one of the most important health indicators in childhood, any disturbance in it may be a sign of disease[4].

Human growth and development stages comprise:

- **Embryonic**: human body systems and structures are originated in this prenatal stage[5].
- **Fetal:** prenatal stage that greatly promotes the success in postnatal growth and development[3].
- **Postnatal:** corresponds to life after birth and is categorized in infancy (neonate to <1 year-old), toddler (1-5 year-old), early childhood (3-8 year-old), middle childhood (9-11 year-old), adolescence (12-18 year-old), adulthood (>18 year-old)[3].

A healthy growth and development begin with parental health, including their genetic characteristics, and continue with conception, prenatal and postnatal stages[6]. Several factors have also been associated to human growth and development, such as nutrition, diseases, parenting factors, environment exposures, among other risk factors[7].

1.1. The preschool-aged children

At the end of the second year of life the growth slow down, children become more active, participate longer at playgrounds and daily sleep duration declines[4]. Additionally, the nutritional requirements and appetite decrease, and food fussiness appears however, in normal conditions children can regulate food intake and adapt it to their nutritional requirements, according to appetite and satiety [4].

Certain growth and development disorders may impact health at this age and may track into adolescence and adulthood[8]. For example, in early childhood, the onset of adiposity rebound, a premature increase in BMI[9], has the potential to increase the risk of obesity in adulthood and, at the same time, it could lead to the development of several comorbidities[4]andit also might predict cardiometabolic risk at older stages of life [9,10].

2. Obesity and cardiometabolic risk in childhood: definitions, epidemiology and clinical issues

2.1. Obesity

Obesity has existed since prehistoric times. Hippocrates, the “father of modern medicine”, first discussed the clinical problem of obesity and described it as an excess of fluids[11]. Over centuries, perception of obesity has changed from a symbol of wealth and prosperity to the called “epidemic of the 21st century”[11]. The World Health Organization (WHO) defines obesity as an anomalous or excessive

collection of body fat mass[12] however, it has also been defined as a status of low-grade systematic inflammation[13,14]. At present, it represents a major global public health concern mainly due to a rapid increase in prevalence since 1975[12]. In Europe, around 13% of children aged 5-9 year-old had obesity in 2020[15] and highest rates were reported in Mediterranean and eastern European countries[16]. In Spain, according to the ALADINO study[17], near to 41% of children aged 6-9 year-old had overweight or obesity in 2019. It is expected that around 9 million of children aged 5-9-year-old will have obesity for 2030 in European countries[15].

Idiopathic obesity is a multisystemic disease which could lead to short and long-term health consequences that may affect the cardiometabolic, cardiovascular, endocrine, renal, pulmonary, gastrointestinal, musculoskeletal, dermatologic, neurologic and psychosocial systems or dimensions[18–20]. Potential comorbidity of obesity in children includes, among others [19,21,22]:

- **Psychosocial**: depression, eating disorders, scarce self-esteem.
- **Cardiovascular**: dyslipidemia, hypertension, coagulopathy, chronic inflammation, endothelial dysfunction.
- **Gastrointestinal**: gallstones, non-alcoholic fatty liver disease, non-alcoholic steatohepatitis.
- **Endocrine**: type 2 diabetes, precocious puberty, polycystic ovary syndrome, male hypogonadism.

- **Neurologic**: pseudotumor cerebri.
- **Pulmonary**: sleep apnea, asthma, obesity hypoventilation syndrome.
- **Renal**: glomerulopathy.
- **Musculoskeletal**: forearm fracture, slipped capital femoral epiphysis, flat feet, genu valgo.

Additionally, obesity may track into adulthood, including the associated risk for certain comorbidities such as cardiometabolic disorders[23–29] and other chronic diseases, including cancer[30].

2.2. Cardiometabolic risk

In children, cardiometabolic risk has been a continuous concern[31] as well as the role of obesity[32]. In this life stage, it has been reported that cardiometabolic disorders could be favor by increased adiposity[33–35], especially central body fat accumulation[36]. In the last years, cardiometabolic risk has begun to be analyzed deeper at this stage of life[37] and certain scores have been created for this purpose, which are usually comprised of waist circumference, triglycerides, HDL-c, glucose, and systolic blood pressure[38,39]. However, waist-to-height ratio has also been reported as a potential predictor of cardiometabolic risk[40]. According to a Canadian Health Measures Survey conducted from 2007 to 2011[41], the most prevalent cardiometabolic risk factors in children and adolescents were abdominal obesity, low HDL-c or elevated triglycerides and the

combination of these three components accounted for 61.5% of the population. In this sense, there is increased evidence on the association between childhood obesity and cardiometabolic risk and the prevalence or incidence of cardiometabolic disorders in adulthood.[26–28,30,42]

Metabolic syndrome (MetS) is a complex interrelated network of cardiometabolic risk factors including central obesity, impaired plasma glucose, hypertension and dyslipidemia that has been defined in adults[43] but not in children[44]. Nevertheless, the American Heart Association (AHA) has suggested that MetS in children may also include abdominal obesity, abnormal lipid profile, high blood pressure and glucose[44]. In this sense, a recent systematic review and modelling analysis showed that global prevalence of MetS in 2020 was about 7.6% (about 61.3 million) in children and adolescents, of which 1.4% of children aged under 13 years were living in northwestern Europe and Spain ranked third as the country with the highest prevalence of MetS in adolescents (9.9%)[45].

3. Obesity and cardiometabolic risk in childhood: pathophysiology

Adipose tissue is mainly comprised by adipose cells or adipocytes[46] and is divided in white and brown adipose tissue[19]. White adipose tissue has an important role in the storage of energy in the form of triglycerides[19] and in the production of biologically active molecules (adipokines) involved in metabolic homeostasis[47], including leptin,

interleukin 6, adiponectin and necrosis tumor alpha factor [48]. In terms of pathophysiology, obesity is characterized by a hypertrophy (increase of volume) in adipocytes[49] which produce an imbalance in adipokines[50], increase their levels[48] and lead to a proinflammatory state, insulin resistance[51], all of which increase the risk of metabolic disorders[50]. Furthermore, hypertrophy of white adipose tissue could lead to restriction of oxygen diffusion from capillaries to adipocytes, causing hypoxia, altering the expression of several genes, and leading to insulin resistance and disturbances in adrenergic signaling that increase inflammation and cellular damage[19]. Moreover, due to endocrine function [19] and the extensive capillarity network on white adipose tissue[52] and its connection with the rest of the body, dysregulation of adipokines may be related to effects on bone metabolism, reproduction, immunity, growth, among many other biological processes[53], which may explain the comorbidities of obesity.

4. Obesity and cardiometabolic risk in childhood: risk factors

The etiology of obesity is multifactorial[54–56], so that several factors interact in the called "obesogenic environment". This term was initially defined as "the sum of influences that the surroundings, opportunities, or conditions of life have on promoting obesity in individuals or populations" and include biological, behavioral, and environmental factors[55]. In overall, the main factors that have been

associated with obesity and its comorbidities are genetics[57], early life factors[58], lifestyle behaviors[59] and environment[60], among many others.

4.1. Genetics

At the end of the last century, several authors suggested the obesity as a hereditary disorder[61,62]. In 2007, fat mass and BMI were observed in association with the obesity-associated gene region[63]. Since then, BMI has been associated with about 97 specific sites of certain genes which may be overlapped with biological pathways that respond to changes in dietary habits[64]. Certain cardiometabolic risk factors have also been associated with several variants in genes[65] and epigenetic changes[66]. Studies in twins have shown that genetics may play an important role in BMI based on exposition to an obesogenic environment[67,68]. Recent evidence has shown that race/ethnicity may influence on several risk factors which are associated with higher likelihood of childhood obesity[69]. Moreover, the contribution of genetics to weight gain[70] as well as the impact of certain risk factors on adiposity[71] and cardiometabolic risk[72] could be different according to sex (established by sexual chromosomes). On the other hand, it has recently been reported that genetics may influence the success of obesity treatment based on lifestyle intervention[73].

4.2. Family and early life

The WHO[16] has recently described early life as "one of the greatest promises for breaking intergenerational cycle of obesity". In this sense, several family and early life factors, including the in-utero environment have been suggested in association with childhood obesity[74] and cardiometabolic risk[75]. In this sense, maternal weight status[76] (after or during pregnancy[77]), lifestyle while pregnant[78–80] (including diet, consumption of alcohol, physical activity, weight gain and smoking status), parenting styles[81], parental BMI[82], feeding practices[83], and lifestyle[84] have been related to adiposity and/or cardiometabolic risk in offspring. Moreover, exposure to gestational diabetes mellitus during pregnancy[85] has been associated with certain cardiometabolic risk factors such as systolic blood pressure, BMI z-score and glucose. Furthermore, a higher adherence to MedDiet in pregnant women has been reported in association with favorable outcomes in offspring such as decreased waist circumference[86], lower risk of accelerated BMI z-score trajectory and larger birth size[87]. Kanellopopoulou et al[88] suggested that family structure could act as a mediator between adherence to MedDiet and the likelihood of overweight or obesity in children. Recently, it has been suggested that size and weight at birth may favor the future onset of obesity[89,90] and increased cardiometabolic risk[90,91] in childhood and adolescence. A mechanism in energy balance and regulation of food intake has been suggested for breastfeeding,

which is rich in hormones such as adiponectin, ghrelin, resistin and obestatin that may play a role on adiposity[92–94] and levels of certain cardiometabolic risk factors[93]. Besides, breastfeeding duration could lead to different cardiometabolic outcomes in early life[77,95] and may be related to the development of certain dietary habits [96]. On the other hand, other factors related to early life such as and timing of introduction to solid foods[97], may increase the risk of adiposity and cardiometabolic disorders in children. Additionally, the delayed introduction of sugar and juice has been suggested in association with increased diet quality in childhood [98], which has the potential to impact on cardiometabolic profile.

4.3. Environment

Regarding environment, a wide range of factors have been suggested in relation with cardiometabolic health which may comprise family, community, school, politics, culture and economy[99], among many others. It is considered that obesity has a especial social predisposition so that, it has been frequently observed in association with socioeconomic status[100,101], which may be a mediator on the association between obesity and other risk factors such as lifestyle behaviors[102] or food insecurity[103,104]. Other environmental determinants, such as geographical school area[105], migration background[106], neighborhood features [107,108], childhood abuse[60] and repeated exposure to broad-spectrum antibiotics[109] have also been suggested in association with cardiometabolic risk.

4.4. Lifestyle

In children, several lifestyle behaviors have started to be assess in association with cardiometabolic risk since past years[110–113], which may be independent of some intrinsic factors[114] and include diet[111], other eating behaviors[115], physical activity[116], sedentary behaviors[117], sleep duration[118], among others. Interestingly, different patterns of lifestyle behaviors have been observed according to sex[102].

4.5. Diet quality, eating speed and eating frequency

4.5.1. First article

"Eating Speed, Eating Frequency, and Their Relationships with Diet Quality, Adiposity, and Metabolic Syndrome, or Its Components"

Tany E. Garcidueñas-Fimbres, Indira Paz-Graniel, Stephanie K. Nishi, Jordi Salas-Salvadó and Nancy Babio.

- Publication phase: published.
- Doctoral student's position: first author.
- Journal: Nutrients.
- Category: Nutrition & Dietetics.
- Range position: 17/89 – Q1.
- Impact factor: 5.717.
- Doi: 10.3390/nu13051687

Summary of results

In this review, 223 studies were identified but only 52 studies met the inclusion criteria and were included.

In children, observational and experimental studies reported mainly significant positive associations between fast-eating speed and adiposity and only a few reported inverse associations between slow eating and adiposity. A higher eating frequency was mostly observed in significant association with lower adiposity and cardiometabolic risk factors. Significant positive but also negative associations were observed between eating frequency and diet quality, including those for meal and snack frequency.

In adults, observational studies reported a significant association between fast-eating category and higher adiposity, levels of cardiometabolic risk factors and risk for developing MetS. Associations between eating frequency and cardiometabolic outcomes were inversely and also positively significant in observational studies and clinical trials but, only positive associations were observed for diet quality in cross-sectional studies.

Conclusion

Fast-eating speed could be associated with increased adiposity and MetS or its components. Furthermore, a greater eating frequency may be mainly associated with diet quality and lower risk of adiposity and MetS or its component

nutrients

MDPI

Review

Eating Speed, Eating Frequency, and Their Relationships with Diet Quality, Adiposity, and Metabolic Syndrome, or Its Components

Tany E. Garcidueñas-Fimbres [1,2,3,†], Indira Paz-Graniel [1,2,3,†], Stephanie K. Nishi [1,2,3], Jordi Salas-Salvadó [1,*] and Nancy Babio [1,2,3,*]

[1] Human Nutrition Unit, Department of Biochemistry and Biotechnology, Universitat Rovira i Virgili, 43201 Reus, Spain; tanyelizabeth.garciduenas@estudiants.urv.cat (T.E.G.-F.); indiradelsocorro.paz@urv.cat (I.P.-G.); stephanie.nishi@urv.cat (S.K.N.)
[2] Institut d'Investigació Sanitària Pere Virgili (IISPV), 43204 Reus, Spain
[3] CIBER Physiology of Obesity and Nutrition (CIBEROBN), Carlos III Health Institute, 28029 Madrid, Spain
* Correspondence: jordi.salas@urv.cat (J.S.-S.); nancy.babio@urv.cat (N.B.); Tel.: +34-977759313 (N.B.); Fax: +34-977759322 (N.B.)
† These authors contributed equally to this work.

Abstract: Excess body weight is a major global health concern, particularly due to its associated increased health risks. Several strategies have been proposed to prevent overweight and obesity onset. In the past decade, it has been suggested that eating speed/rate and eating frequency might be related to obesity. The main aim of this narrative review was to summarize existing evidence regarding the impact of eating speed/rate and eating frequency on adiposity, metabolic syndrome (MetS), or diet quality (DQ). For this purpose, a literature search of observational and interventional trials was conducted between June and September 2020 in PubMed and Web of Sciences databases, without any data filters and no limitations for publication date. Results suggest that children and adults with a faster eating speed/rate may be associated with a higher risk of developing adiposity, MetS or its components. Furthermore, a higher eating frequency could be associated with diet quality improvement, lower adiposity, and lower risk of developing MetS or its components. Further interventional trials are warranted to clarify the mechanism by which these eating behaviors might have a potential impact on health.

Keywords: eating speed; eating rate; eating frequency; adiposity; BMI; eating behaviors; metabolic syndrome; MetS

Citation: Garcidueñas-Fimbres, T.E.; Paz-Graniel, I.; Nishi, S.K.; Salas-Salvadó, J.; Babio, N. Eating Speed, Eating Frequency, and Their Relationships with Diet Quality, Adiposity, and Metabolic Syndrome, or Its Components. *Nutrients* **2021**, *13*, 1687. https://doi.org/10.3390/nu13051687

Academic Editors: Maria Luz Fernandez and Katarzyna Przybyłowicz

Received: 25 March 2021
Accepted: 13 May 2021
Published: 15 May 2021

1. Introduction

Weight disorders, such as overweight (OW) and obesity (OB), are prevalent globally, mainly because of unhealthy diets and sedentary behaviors [1]. In accordance with data from the World Health Organization, global obesity has nearly tripled since 1975 [1]. By 2016, more than 1.9 billion adults were overweight, of these over 650 million were obese and 340 million children and adolescents aged 5 to 19 years were overweight or obese. Meanwhile by 2019, over 38 million children under 5 years-old were overweight or obese [1]. Obesity is defined as a health problem that consists mainly of body fat accumulation, with a multifactorial etiology [2]. It is related to an immense variety of short and long-term consequences, which may affect quality of life [3]. Excess body weight (BW) has been associated with metabolic syndrome (abdominal obesity, elevated fasting plasma glucose, low level of high-density lipoprotein cholesterol (HDL-c), hypertriglyceridemia, and hypertension) [4], a complex interrelated network of metabolic risk factors [5], which increases the risk of diabetes and premature death [6].

Childhood obesity is a risk factor for overweight and obesity in adulthood [7]; therefore, its prevention and early treatment are major concerns for public health. Several

strategies have been proposed to prevent overweight and obesity onset, where the vast majority are based on avoiding sedentary behaviors and a western high energy density dietary pattern, rich in refined food, sugary beverage consumption, red and processed meat, and low in vegetables, fruits, whole grains, and legumes [8].

In the last decade, several authors have queried [9–11] if certain dietary behaviors, such as eating speed/rate, defined as the time required to eat an amount of food [12,13], and eating frequency, the number of eating occasions per day either reported as a meal or a snack [10,11,14–17], may be related to obesity, MetS, and dietary quality. It was also suggested that these eating behaviors may have a potential influence in the development of chronic diseases, such as diabetes and cardiovascular disease [18]. For example, in Asian populations, eating speed/rate has been associated with adiposity [19,20], but this has not been shown in Europeans [21]. In Europe, a study conducted in Spanish participants found a higher risk for hypertriglyceridemia in individuals with a fast-eating speed [21]. Moreover, significant associations have been observed between eating frequency and diet frequency, adiposity, or cardiovascular risk [22,23]. In children, the evidence is scarce, but it is suggested that these eating behaviors may play an important role in their health maintenance [24,25]. Along these lines, evidence is quite controversial because a frame of reference for adequate values and universal definitions for these behaviors have not been settled [17]. In view of the impact of eating frequency and eating speed/rate may have on health, the aim of this review was to summarize all the available evidence (with no limitation for publication date) of these eating behaviors in relation to body mass index (BMI), body weight, waist circumference (WC), diet quality, and other MetS components in children, adolescents, and adults.

2. Materials and Methods

2.1. Search Strategy and Selection Criteria

A literature search was performed in PubMed and Web of Science from June to September 2020 without any data filters, identifying observational and interventional trials examining possible associations between certain eating behaviors (e.g., eating speed/rate and eating frequency), diet quality, cardiometabolic biomarkers and adiposity measures in adults (>19 years) and children ($\leq$19 years).

For this narrative review, the following terms were used to define the exposure: eating speed, eating rate, eating frequency, meal frequency and snack frequency. For outcomes, keywords related to adiposity and cardiometabolic biomarkers were searched, such as glucose, cholesterol, blood pressure, adiposity, body weight, BMI, body mass index, WC, as well as diet quality. Furthermore, the search was complemented with a manual search of the reference lists of included articles.

2.2. Inclusion and Exclusion Criteria

To be included, articles had to meet the following inclusion criteria: full-text available, observational study, clinical study, clinical trial, journal article, randomized controlled trial, and involving humans, written in the English or Spanish language. A search was performed in the MEDLINE database (PubMed and Web of Science) until September 2020 and all available articles that met inclusion criteria were included. Exclusion criteria included letters to editors, abstracts only, case reports, registered protocols, and ongoing trials.

2.3. Study Selection

Two researchers conducted the article review and selection, with any disagreement being settled by consensus. A specific spreadsheet program (Excel, Microsoft Windows 10 Pro) was used for data collection and summarizing descriptive information and results from the included studies. Relevant data comprised of first author's surname, year of publication, population, study design, exposure, outcome variables and relevant results. The tables were drafted and classified by study design (cross-sectional, longitudinal, or

interventional) and age group (children or adult); their eligibility was widely discussed by consensus.

2.4. Exposures

Definitions for eating speed, eating rate, and eating frequency display great variability among studies. Below will describe the definitions used in the present review.

According to the literature, eating speed and eating rate are frequently used as synonyms, being mainly defined, as the time required to eat an amount of food. The most frequent assessment method used was based on self-reporting (subjective), but timers, videotapes, and direct observation were occasionally used to objectively assess eating speed or eating rate.

Eating frequency corresponds to the number of eating occasions per day [12], moreover some authors reported meal and/or snack frequency. Eating frequency is often assessed by self-report, but a few authors evaluated frequency and whether it was considered a meal or snack according to the energy percentage contribution (% energy) or based on the hour (clock time) [13–16].

2.5. Outcomes

In the present review, overweight and obesity in adults was defined as a BMI $\geq$ 25 kg/m^2 and a BMI $\geq$ 30 kg/m^2, respectively. Meanwhile in children, excess body weight was evaluated with BMI in kg/m^2 (according to IOTF criteria [26–29], British Growth reference criteria [14] or Central for Disease Control and Prevention criteria [12]) or percentiles (according to CDC criteria [30,31] or WHO criteria [32]), as well as BMI z-scores [4,10,12,14,17,24,25,27,31]. Abdominal obesity, in adults, was defined by WC cut-offs according to ethnicity. In Asian populations, cut-off points were established by having a waist circumference $\geq$85 cm in men, and $\geq$80 cm in women [33,34]. For European populations the cut-off point for abdominal obesity was considered as WC > 102 cm in men and >88 cm in women [33]. In children, only one study [32] evaluated abdominal obesity, which was defined as a waist-hip ratio >0.5 [35].

In adults, MetS was defined [33,36–39] when 3 or more of the following factors were present: (1) abdominal obesity: defined in Asian population as a waist circumference $\geq$85 in men, and $\geq$80 cm in women and in a European population as a WC > 102 cm in men and >88 cm in women; (2) high blood pressure: systolic blood pressure $\geq$130 mmHg or diastolic blood pressure $\geq$85 mmHg or taking an antihypertensive drug treatment; (3) high fasting plasma glucose: $\geq$5.6 mmol/L (100 mg/dL) or on a drug treatment for type 2 diabetes; (4) hypertriglyceridemia: $\geq$1.7 mmol/L (150 mg/dL) or on a drug treatment for high plasma triglycerides; (5) reduced HDL-c: < 1.04 mmol/L (40 mg/dL) for men or <1.3 mmol/L (50 mg/dL) for women.

In children, MetS was considered when there was central obesity (WC $\geq$90th percentile or using the adult cut-offs if lower) along with the presence of $\geq$2 additional factors: (1) high serum triglycerides: $\geq$1.7 mmol/L; (2) high fasting plasma glucose: $\geq$5.6 mmol/L; (3) reduced HDL-c: $\leq$1.03 mmol/L; (4) high blood pressure: systolic blood pressure $\geq$130 mmHg or diastolic blood pressure $\geq$85 mmHg [40]. In the case of Kelishadi et al. [32], elevated blood pressure cut-offs were based on the 90th percentile of systolic and diastolic blood pressure according to gender, age, and height [41].

Diet quality was assessed by distinct methods, such as the following indexes or scores: healthy eating index [42,43], Mediterranean diet score [44,45], mean adequacy ratio [46,47], healthy diet indicator [45,48], dietary guidelines index [49] or dietary quality score [50].

3. Results

3.1. Study Inclusion

The search identified 223 articles, 133 from PubMed and 90 from Web of Science. Additionally, five articles meeting inclusion criteria were identified from the manual search. Following an initial review of title and abstract, 176 articles were excluded: 33 duplicates,

121 did not present relevant outcomes, and 18 were reviews. Based on review of full manuscripts, an additional four articles were excluded by consensus due to irrelevant exposure or outcome. Therefore, 52 articles were included in this review (Figure 1).

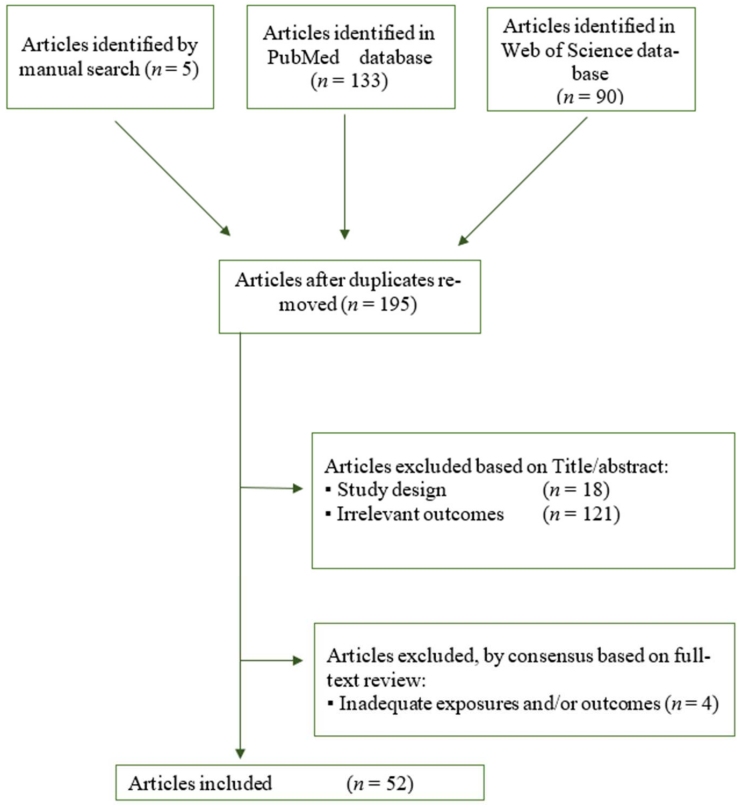

Figure 1. Flow diagram of the literature search and selection process.

3.2. Study Characteristic

Of the 52 studies, 18 were conducted in Asia, 14 in Europe, 18 in America, 1 in Oceania, and 1 in America and Europe. The year of publication spanned from 1964 to 2020. Nine studies (one study explored both eating speed/rate and eating frequency) included only children [4,12,14,24,26–28,30,31] one was based on children and adolescents (≤19 years-old) [51], two articles involved only adolescents [25,29], four articles recruited children, adolescents and young adults [10,15,17,32] and finally, 36 articles included only adults [2,5,9,11,13,16,18–23,52–75]. In total, 157,034 adults and 37,119 children and adolescents in the cross-sectional studies were included. Longitudinal studies incorporated 64,583 adults and 5610 children, and interventional trials were conducted in 357 adults and 24 children.

3.3. Cross-Sectional Studies

3.3.1. Eating Speed/Rate

Table 1 shows the main characteristics and results from cross-sectional studies examining the associations between eating speed/rate with adiposity measures, metabolic syndrome, and diet quality.

Table 1. Characteristics and main findings of cross-sectional studies that explored eating speed/rate.

Cross-Sectional Studies					
Children					
Author and Year	**Population**	**Exposure (s)**	**Outcome (s)**	**Adjusted Variables**	**Results**
Eloranta 2012 [26].	■ PANIC study. ■ Finnish. ■ *n* = 510 (247 girls and 263 boys). ■ Age: 6–8 years.	Slowness in eating: finishing eating in more than 30 min (self-reported).	OW, OB, WC, and HC.	Sex, age, total daily time of PA, total daily screen time, and parental income level.	■ Slowness in eating: OW/OB (OR = 0.61 [95% CI: 0.41, 0.92]), WC (β = −0.16, p < 0.01) and HC (β = −0.17, p < 0.01).
Fogel 2017 [24].	■ GUSTO cohort. ■ Asian. ■ *n* = 386 (184 girls and 202 boys). ■ Age: 4.5 years.	Eating rate (video recorded—gr/min).	WC.	None.	■ Eating rate: WC (r = 0.17, p < 0.01).
Okubo2017 [28].	■ Osaka Maternal and Child Health Study. ■ Japanese. ■ *n* = 492 mother-child pairs. ■ Age: 30–42 m. ■ 1 year follow-up.	Eating rate (self-reported).	BMI.	Child's sex and age (at fourth survey), maternal age and BMI at enrollment, education level, family income, pregnancy smoking status, maternal working status at 30 months postpartum, birth order, birth weight, duration of breast-feeding, time spent watching TV at 30 months of age, protein intake, fat intake, and dietary fiber at 30 months of age.	Fast eating rate vs. slow eating rate at 30 m.: ■ BMI at 30 m. (β = 0.70 [95% CI: 0.33, 1.08]).
Okubo 2018 [27].	■ SHOKUIKU Study ■ Japanese ■ *n* = 4451 (2136 girls and 2315 boys). ■ Age: 5–6 years.	Eating rate (self-reported).	OW and BMI z-score.	Sex, age, n° of siblings, PA and birthweight; residential block, parents' educational attainment, parents weight status, protein (% of energy), fat (% of energy), and dietary fiber intake (g/1000 kcal).	■ Fast eating rate vs. medium eating: OW (OR = 2.71 [95% CI: 2.10, 3.48]). ■ Fast eating rate: >BMI z-score (p < 0.01).

Table 1. *Cont.*

Adults					
Author and Year	**Population**	**Exposure (s)**	**Outcome (s)**	**Adjusted Variables**	**Results**
Lee 2013 [18].	■ South Korean. ■ *n* = 8775 (3956 women, 4819 men). ■ Age: 20–80 years.	Eating rate (self-reported).	High FPG, high BP, low HDL-c, high TG, BMI (>25 kg/m^2).	Age, alcohol, smoking, and exercise and BMI, total energy intake.	Fast eating rate vs. slow eating speed: ■ Women: BMI (OR = 3.35 [95% CI: 2.23, 5.3]). Other variables: NA.
Nagahama 2014 [55].	■ Japanese. ■ *n* = 56,865 (15, 045 women and 41,820 men). ■ Age: 17–99 years.	Self-reporting eating rate.	MetS, CO, high BP, high FPG, high TG, low HDL-c.	Age, smoking status, alcohol, regular physical activity and body mass index.	Fast eating rate vs. normal eating rate: ■ Men: CO (OR = 1.97 [95% CI: 1.88, 2.07]), low HDL-c (OR = 1.10 [95% CI: 1.03, 1.18]), high TG (OR = 1.07 [95% CI: 1.02, 1.12]) and MetS (OR = 1.10 [95% CI: 1.03, 1.17]). ■ Women: CO (OR = 1.44 [95% CI: 1.33, 1.56]). Other variables: NA.
Lee 2016 [20].	■ Japanese. ■ *n* = 4249 (2163 women and 2086 men). ■ Age: 20–80 years.	Eating quickly (self-reported).	OW.	Gender, age, living with spouse, occupation, education, visiting hospitals, habitual exercise, smoking status, and alcohol drinking.	■ Eating quickly vs. no eating quickly: OW (OR = 1.92 [95% CI: 1.62, 2.28]).
Hamada 2017 [53].	■ Japanese. ■ *n* = 84 (women college students). ■ Age: 19 years.	Eating speed: - Subjective (self-reported) - Objective (total number of chews, number of chews/bites, total meal duration, number of bites, chewing rate).	BW, BMI, WC, AC and HC.	None.	■ Total # chews and meal duration ($p < 0.05$): BW (r = 0.22, 0.24), BMI (r = 0.24, 0.27), WC (r = 0.26, 0.24), AC (r = 0.25, 0.27) and HC (r = 0.24, 0.22). ■ Total # bites ($p < 0.05$): BW (r = 0.25) and AC (r = 0.25). ■ Subjective fast eating speed vs. subjective slow eating speed ($p < 0.05$): > BW, BMI, WC, AC and HC.
van den Boer 2017 [13].	■ Dutch. ■ *n* = 1473 (732 women and 741 men). ■ Age: 20–70 years.	Eating speed (self-reported).	OW.	Age, smoking, level of education, emotional eating, restrained eating, external eating, energy intake, moderate to vigorous activity, and sedentary activity.	Fast eating speed vs. average eating speed in total population: OW (β = 0.90 [95% CI: 0.48, 1.32]). ■ Stratified analysis: OW in women (β = 1.13 [95% CI: 0.43, 1.84]).

Table 1. *Cont.*

Adults					
Author and Year	**Population**	**Exposure (s)**	**Outcome (s)**	**Adjusted Variables**	**Results**
Sonoda 2018 [52].	■ Japanese. ■ *n* = 863 men (From Japanese Maritime Self Defense Force).	Eating speed (self-reported).	BMI and WC.	Eating speed, n° of missing functional teeth, periodontal status, age, military ranks, alcohol, smoking, and exercise frequency.	■ Fast eating speed vs. slow/very slow eating speed: BMI≥ 25 kg/m^2 (OR = 5.04 [95% CI: 1.95, 13.07]), BMI≥ 30 kg/m^2 (OR = 4.80 [95% CI: 1.21, 19.09]), WC≥ 85 cm (OR = 6.59 [95% CI: 2.37, 18.48]) and WC≥ 90 cm (OR = 5.22 [95% CI: 1.81, 15.06]).
Tao 2018 [19].	■ Chinese. ■ *n* = 7972 (3508 women and 3436 men). ■ 18–65 years.	Eating speed (self-reported).	MetS, CO, elevated BP, elevated FPG, elevated TG, reduced HDL-c.	Age, education level, work stress, PA intensity, PA frequency, sleep duration, smoking, drinking, high salt intake, high intake of sugar, fat and/or meat, a mainly vegetable diet, frequency of eating breakfast, grain consumption, antihypertensive, antidiabetic, and/or hypolipidemic medication.	Fast eating speed vs. slow eating speed: ■ Total population: prevalence for MetS (OR = 2.27 [95% CI: 1.80, 2.86]), CO (OR = 1.68 [95% CI: 1.35, 2.09]), BP (OR = 1.82 [95% CI: 1.48, 2.24]), TG (OR = 1.51 [95% CI: 1.21, 1.88]) and HDL-c (OR = 1.33 [95% CI: 1.11, 1.60]). ■ Men: prevalence for MetS (OR = 2.21 [95% CI: 1.69, 2.91]), CO (OR = 1.53 [95% CI: 1.16, 2.02]), BP (OR = 1.50 [95% CI: 1.17, 1.92]), TG (OR = 1.69 [95% CI: 1.29, 2.23]) and HDL-c (OR = 1.35 [95% CI: 1.02, 1.79]). ■ Women: prevalence for MetS (OR = 2.27 [95% CI: 1.46, 3.53]), CO (OR = 1.98 [95% CI: 1.36, 2.88]), BP (OR = 3.02 [95% CI: 2.00, 4.56]) and FPG (OR = 1.56 [95% CI: 1.05, 2.33]).

Table 1. *Cont.*

Adults					
Author and Year	**Population**	**Exposure (s)**	**Outcome (s)**	**Adjusted Variables**	**Results**
Paz-Graniel 2019 [21].	▪ PREDIMED-Reus study. ▪ Spanish. ▪ *n* = 792 (451 women and 341 men). ▪ Age: 55–80 years.	Eating speed (self-reported).	OB, MetS, CO, hypertriglyceridemia, low HDL-c, high BP, high FPG.	Age, sex, educational level, smoking status, use of dental prosthesis, total energy intake (kcal/day), alcohol consumption (g/day), physical activity (MET/min/day), and adherence to Mediterranean diet.	▪ Fast eating speed: HR+ 59% for hypertriglyceridemia (HR = 1.59 [95% CI: 1.08, 2.02]). ▪ MetS and other variables: NA.
Wuren 2019 [54].	▪ Japan Multi-Institutional Collaborative Cohort study. ▪ Japanese. ▪ *n* = 5888 (2495 women and 3393 men). ▪ Age: 70–79 years.	Eating rate (self-reported).	BMI (> 25 kg/m^2) and WC (> 80 cm in females and > 90 cm in males).	Age, current smoker, alcohol, PA, total energy intake, medication for hypertension, diabetes and/or dyslipidemia, sleep duration, psychological stress, education level, family structure, fast food, restaurants or food service use, packed lunch, dinner, snacking, and breakfast time.	Fast eating rate vs. normal eating rate: ▪ BMI (men: (OR = 1.48 [95% CI: 1.25, 1.76]; women: OR = 1.78 [95% CI: 1.39, 2.26]). ▪ WC (men: OR = 1.45 [95% CI: 1.21, 1.74]; women: OR = 1.34 [95% CI: 1.11, 1.61]).

Abbreviations: AC, abdominal circumference; β, beta coefficient; BMI, body mass index; BP, blood pressure; BW, body weight; CO, central obesity; FPG, fasting plasma glucose; GUSTO, Growing UP in Singapore Towards Healthy Outcomes; HC, hip circumference; HDL-c, high density lipoprotein cholesterol; HR, hazard ratio; M., months; MET, Metabolic Equivalent of Task; MetS, metabolic syndrome; NA, no associations; OB, obesity; OR, odds ratio; OW, overweight; PA, physical activity; r, PANIC, Physical Activity and Nutrition in Children; Pearson's correlation; PREDIMED, Prevencion con Dieta Mediterránea; TG, triglycerides; vs., versus; WC, waist circumference.

Children

Regarding adipose measurements, fast eating speed/rate was significantly associated with greater risk of overweight (odds ratio (OR) = 2.71; 95% CI: 2.10, 3.48) and overweight/obesity (β = 0.70; 95% CI: 0.33, 1.08) [27,28], waist circumference [24] and higher BMI z-score [27]. Whereas, slowness in eating showed significantly inverse associations with overweight (OR = 0.61; 95%CI: 0.41, 0.92) and waist circumference [26]. No studies exploring dietary quality or metabolic risk biomarkers were found that met the inclusion criteria of this review.

Adults

From the total number of included articles exploring the association between eating speed and adiposity measures, four of them also examined MetS biomarkers, but none explored an association with dietary quality. Seven articles examined BMI, six looked at WC, and one assessed body weight. Hamada et al. [53] studied the speed of eating, subjectively (self-reported) and objectively (total number of chews, number of chews per bite, total meal duration, number of bites, chewing rate). They observed significant inverse correlations between total number of chews and total meal duration with body weight, BMI, waist circumference and abdominal obesity [53]. Moreover, several authors reported significant associations between self-reported fast eating speed/rate and increased risk for abdominal obesity prevalence [19,53,55], BMI [13,18,20,52,54], and larger WC [52,54]. No significant associations between eating speed and adiposity were reported in a study of a Spanish population conducted by Paz-Graniel et al. [21].

Regarding components of MetS, four studies explored associations between eating speed/rate and fasting plasma glucose, HDL-c, triglycerides, and blood pressure, and two studies assessed the risk of MetS prevalence. Paz-Graniel et al. [21] observed a 59% higher risk of hypertriglyceridemia in the fastest eating speed group compared to the slowest (HR = 1.59; 95% CI: 1.16–2.17). Similar results were reported in two additional studies [19,55], where fast-eating speed was associated with 7–69% greater risk of higher triglycerides. Fast eating speed/rate was also significantly associated with higher prevalence of low HDL-c [19,55] fasting plasma glucose [19], blood pressure [19], and MetS [19,55]. Nonetheless, not all cross-sectional studies in adults found significant associations between eating speed/rate and prevalence of high fasting plasma glucose [18,21,55] low HDL-c [18,21], high blood pressure [18,21,55], or MetS [21].

3.3.2. Eating Frequency

Table 2 shows the description and relevant results from cross-sectional studies examining the associations between eating frequency with adiposity measures, MetS, and diet quality.

Children

The association between eating frequency and adiposity measures was explored in seven cross-sectional studies, four of these examined associations with BMI and waist circumference, three explored associations with BMI z-score, and two with total body weight. Significant inverse associations were reported between eating frequency and BMI [14,26,29,32], BMI z-score [14,25], body weight (β = −0.78) [14], WC [14,25,26], and abdominal obesity (OR = 0.73; 95% CI: 0.63, 0.85) [32]. In one study [14] an increase in eating frequency was significantly associated with higher BMI z-score in children with central obesity. In British and American adolescents, for every additional daily eating occasion or meal frequency, a significant increase in BMI z-score was observed [10,17]. No additional associations between eating frequency and anthropometric measures were reported in children [25,29,32].

Table 2. Characteristics and main findings of cross-sectional studies that explored eating frequency.

Cross-Sectional Studies					
Children					
Author and Year	**Population**	**Exposure (s)**	**Outcome (s)**	**Adjusted Variables**	**Results**
Eloranta 2012 [26].	▪ PANIC study. ▪ Finnish. ▪ n = 510 (247 girls and 263 boys). ▪ Age: 6–8 years.	Eating frequency: ▪ 3 main meals/day: breakfast, lunch and dinner. ▪ Snacks (all eating and drink occasions besides main meals).	OW/OB, WC, HC.	Sex, age, total daily time of physical activity, total daily screen time, and parental income level.	Eating 3 main meals vs. not eating 3 main meals: ▪ OW/OB (OR = 0.37 [95% CI: 0.18, 0.75]). ▪ WC ($\beta = -0.16$, $p < 0.01$) and HC ($\beta = -0.1$, $p < 0.01$). Other variables: NA.
Jennings 2012 [14].	▪ SPEEDY study. ▪ British. ▪ n = 1700 (952 girls and 748 boys). ▪ Age: 9–10 years.	Eating frequency: number of time periods of food or drinks consumption (6–9 h, 9–12 h, 12–14 h, 14–17 h, 17–20 h, 20–22 h, 22 h–6 h): ▪ Breakfast (6–9 h). ▪ Mid-day meal (12–14 h). ▪ Evening meal (17–20 h). ▪ Snack (any other time).	BMI, BMI z-score, BW and WC.	Gender, parental education, under-reporting, energy intake, and physical activity.	▪ Healthy weight children ($p \leq 0.03$): BW ($\beta = -0.78$), BMI ($\beta = -0.17$), BMI z-score ($\beta = -0.10$) and WC ($\beta = -0.38$). ▪ Centrally obesity children: BMI z-score ($\beta = 0.09$, $p < 0.05$). ▪ Other variables: NA.
Jääskeläinen 2013 [29].	▪ Northern Finland Birth Cohort 1986. ▪ Finnish. ▪ n = 6247 (3181 girls and 3066 boys). ▪ Age: 16 years.	Meal frequency: ▪ Regular meal pattern: 5 meals a day including breakfast. ▪ Semi-regular meal pattern: $\leq$4 meals a day including breakfast. ▪ Breakfast skippers: $\leq$ 4 meals a day, not including breakfast.	OW/OB, WC, hyperglycemia, hypertriglyc-eridemia, low HDL-c, HT.	Early life factors: birth weight for gestational age, maternal weight gain in the first 20 weeks of gestation, maternal pre-pregnancy BMI, pregnancy smoking, maternal glucose metabolism, and parity. Later childhood factors: tobacco, sleep duration, PA, sedentary time, Tanner stage, parental education level, and body mass index.	Regular meal pattern vs. semi-regular meal pattern: 1. Model adjusted for early life factors: ▪ Boys: hypertriglyceridemia (OR = 0.48 [95% CI: 0.26, 0.89]) and OW/OB (OR = 0.47 [95% CI: 0.34, 0.65]). ▪ Girls: OW/OB (OR = 0.57 [95% CI: 0.41, 0.79]). 2. Model adjusted for later childhood factors: ▪ Boys: OW/OB (OR = 0.41 [95% CI: 0.29, 0.58]). ▪ Girls: OW/OB (OR = 0.63 [95% CI: 0.45, 0.89]). Other variables: NA.
Murakami 2014 [10].	▪ British. ▪ n = 1636 (803 girls and 833 boys). ▪ Age: 4–18 years.	Eating frequency: all eating occasions for food/drinks, except for those providing < 210 kJ of energy.	BMI z-score, HDL- c, TG, SBP and DBP.	Age, sex, social class, physical activity levels, intakes of protein, fat, total sugar and dietary fiber and BMI z-score (in the analysis of blood lipid profile, and blood pressure).	▪ Adolescents $\geq$ 11 years: BMI z-score ($\beta = 0.11$, $p < 0.01$). ▪ Other variables: NA.

Table 2. *Cont.*

Cross-Sectional Studies					
Children					
Author and Year	**Population**	**Exposure (s)**	**Outcome (s)**	**Adjusted Variables**	**Results**
Evans 2015 [51].	▪ Daily D study. ▪ American. ▪ *n* = 176 (89 girls and 87 boys). ▪ Age: 9–15 years.	Eating, meal and snack frequency.	DQ (HEI-2005).	School, maternal education, free or reduced-price school lunch eligibility, and physical activity.	In children (9–11 years.): ▪ EF (β = 2.60, $p < 0.01$) and SF (β = 2.31, $p = 0.02$). In adolescents (12–15 years.): ▪ MF (β = 5.40, $p = 0.01$) and SF (β = −2.73, $p < 0.01$).
House 2015 [25].	▪ SOLAR cohort. ▪ American (Hispanic). ▪ *n* = 191 (83 girls and 108 boys). ▪ Age: 11–15 years.	Eating frequency: ▪ Infrequent: <3 per day. ▪ Frequent: ≥3 meals/day.	BMI z-score, WC, BW, FPG, HDL-c, TG.	Tanner stage, sex, mean energy, total fat, total fat and height, total lean, and height and insulin sensibility.	▪ Frequent eaters vs. Infrequent eaters ($p \leq 0.01$): <BMI z-score, WC and TG. ▪ Other variables: NA.
Kelishadi 2016 [32].	▪ CASPIAN-IV study. ▪ Iranian. ▪ *n* = 13,486 (6635 girls and 6851 boys). ▪ Age: 6–18 years.	Eating frequency (≤3, 4, 5 or ≥6): ▪ Breakfast/lunch/dinner. ▪ Number of snacks.	Elevated BP, elevated DBP, elevated SBP, OW, OB, and AO.	Age, gender, and living area, Screen time, physical activity, socioeconomic status, and sleeping hours, and BMI (only in blood pressure).	▪ ≥6 EF vs. ≤ 3 EF: OB (OR = 0.54 [95% CI: 0.44, 0.65]) and AO (OR = 0.73 [95% CI: 0.63, 0.85]). ▪ Other variables: NA.
Murakami 2016 [17].	▪ British. ▪ *n* = 1636 (803 girls and 833 boys). ▪ Age: 4–18 years.	1. Eating frequency: times/day. 2. Meal and snack frequencies: ▪ Based on energy percentage contribution (meal with ≥15% and snack with <15% of total energy). ▪ Based on time (meals: 6–10 h, 12–15 h and 18–21 h; snack: any other time).	DQ (MDS) and BMI z-score.	Age, sex, social class, physical activity and plausible energy reporters (in adolescents).	In children (4–10 years.): ▪ DQ ($p < 0.01$): EF (β = −0.30), SF-energy% (β = −0.20) SF-time (β = −0.31), MF-time (β = −0.40). In adolescents: ▪ DQ ($p \leq 0.03$): EF (β = −0.10), SF-energy-% (β = −0.09), SF-time (β = −0.18) ▪ BMI z-score: MF-clock time (β = 0.13, $p = 0.02$).
Murakami 2016 [15].	▪ American. ▪ *n* = 10,462 (5188 girls and 5274 boys). ▪ Age: 6–19 years	Eating frequency: times/day. Meal and snack frequencies: ▪ Self report (meals: breakfast, brunch, lunch, supper, and dinner; others: snacks) ▪ Based on time (meals: 6–9 h, 12–14 h and 17–20 h; snack: other). ▪ Based on energy percentage contribution (meal: ≥15%, snack: <15% of total energy).	DQ (HEI-2010).	Sex, age, race/ethnicity, family poverty income ratio, education of household head, household size, PA, watching television and computer use, weight status, dietary reporting status, and survey cycle.	Children of 6–11 years. ($p < 0.03$): ▪ EF (β = 1.21), MF-energy% (β = 1.45), MF-self report (β = 3.59) and MF-time (β = 1.72), SF-energy% (β = 0.70) and SF-self report (β = 0.60). Adolescents ($p < 0.01$): ▪ EF (β = 1.52), MF-energy% (β = 1.74), MF-self report (β = 3.56) and MF-time (β = 1.99) and SF-energy% (β = 1.00).

Table 2. *Cont.*

Cross-Sectional Studies					
Adults					
Author and Year	**Population**	**Exposure (s)**	**Outcome (s)**	**Adjusted Variables**	**Results**
Fábry 1964 [75].	▪ Czech. ▪ *n* = 379 men. ▪ Age: 60–64 years.	Meal frequency: ≤ 3, 3–4 (±snacks) or ≥ 5 per day	OW.	None.	▪ ≤3 MF vs. ≥5 MF: > OW ($p < 0.05$).
Drummond 1998 [61].	▪ Scottish. ▪ *n* = 79 (37 women and 42 men). ▪ Age: 20–55 years.	Eating frequency: ▪ Eating occasion (food and beverage). ▪ Meal (breakfast, lunch or dinner). ▪ Snack.	BW and BMI.	None.	▪ EF in men: BW (r = −0.34, $p = 0.03$). ▪ Other variables: NA.
Zizza 2012 [66].	▪ American. ▪ *n* = 11,209 (5789 women and 5420 men. ▪ Age: ≥ 20 years.	Snack frequency: 0, 1, 2, 3 o ≥ 4.	DQ (HEI-2005).	Sex, race/ethnicity, education, smoking, PA, consumption of ≥ meals/day, chronic diseases, age, BMI, and meals energy contribution.	▪ Higher SF: > DQ ($p < 0.01$).
Kim 2014 [22].	▪ South Korean. ▪ *n* = 4625 (2294 women and 2331 men). ▪ Age: ≥ 19 years.	▪ Eating frequency: all eating occasions (<2, 3, 4 or ≥5). ▪ Meal frequency: 1, 2 or 3. ▪ Snack frequency: 0, 1, 2 or ≥ 3.	HT, AO (WC ≥85 cm in women and ≥90 cm in men) and DQ (MAR).	Age, sex, smoking, smoking amount, alcohol consumption frequency, PA frequency, IPAQS, total daily calorie/sodium/potassium/calcium intake, sleep sufficiency, stress level, MAR, BMI, WC, meal, and snack frequency.	≥5 EF vs. 3 EF: ▪ HT in AO (OR = 0.5 [95% CI: 0.31, 0.82]). ≥3 SF vs. 0 EF: ▪ HT in DQ < 50% (OR = 0.5 [95% CI: 0.23, 0.89]).
Aljuraiban 2015 [62].	▪ INTERMAP study. ▪ American and British. ▪ *n* = 2385 (1153 women and 1232 men). ▪ Age: 40–59 years.	Eating frequency/day: < 4, 4 to < 5, 5 to < 6 and 6.	BMI.	Age, gender, educational level, hours of moderate and heavy physical activity, smoking, special diet, dietary supplement use, and population sample.	▪ BMI (β = −1.1 [95% CI: −1.6, −0.7]).
O'Connor 2015 [64].	▪ Fenland study. ▪ British. ▪ *n* = 10,092 (5446 women and 4646 men).	Snack frequency (self-reported).	BMI and WC.	Age, alcohol, smoking, age at completing full-time education, test site, main meal, light meal, drink-only snack, plasma vitamin C, energy intake, screen time, and PA energy expenditure.	SF in women: BMI (β = 0.29 [95% CI: 0.13, 0.44]) and WC (β = 0.73 [95% CI: 0.4, 1.1]). Every additional unit in SF: ▪ Women with BMI ≥ 25 kg/m^2: WC (β = 0.80 [95% CI: 0.34, 1.26]). ▪ Men with BMI < 25 kg/m^2: WC (β = −0.52 [95% CI: −0.90, −0.14]).

Table 2. *Cont.*

Cross-Sectional Studies					
Barnes 2015 [60].	▪ American. ▪ n = 233 (157 women and 76 men). ▪ Age: 18–60 years.	Snack frequency: Beverage and/or meal in any eating occasion that is not breakfast, lunch, or dinner.	BMI and DQ (HEI-2010).	Age, sex, race/ethnicity, education level, income, job type, marital/partner status, physical activity, and total daily energy intake.	▪ No significant associations.
Holm 2015 [58].	▪ European. ▪ n = 7531 (3612 women and 3919 men). ▪ Age: 15–80 years.	Meal frequency: 0–3, 4, ≥5.	DQ (DQS).	Age, gender, household composition, educational level, occupational status and socioeconomic level.	▪ EF ≥ 5 vs. EF 0–3 ($p \leq 0.02$): Denmark (β = 0.71), Finland (β = 0.37), Norway (β = 0.48) and Sweden (β = 0.68).
Zhu 2016 [65].	▪ American. ▪ n = 7791 (3774 women and 4017 men). ▪ Age: ≥ 20 years.	Eating frequency (self-reported).	DQ (HEI-2010), BMI, WC.	Age, race/ethnicity, ratio of family income: poverty, PA level, smoking, and energy intake.	Both genders: ▪ DQ in women (β = 1.63, $p < 0.01$) and men (β = 1.7, $p < 0.01$). ▪ WC in women (β = −0.8, $p = 0.01$) and men (β = −0.6, $p = 0.03$). Women: ▪ BMI (β = −0.4, $p < 0.01$).
Murakami 2016 [16].	▪ British. ▪ n = 1487 (809 women and 678 men). ▪ Age: 19–64 years.	Meal and snack frequency: ▪Based on energy percentage contribution: meal: ≥15% of total energy intake; snack: < 15%. ▪ Based on time: meal: from 06.00–10.00 h, 12.00–15.00 h or 18.00–21.00 h; snack: other occasion.	DQ (MDS and HDI), BMI and WC.	Age, social class, EI: EER (energy intake misreporting), smoking status, PA, protein intake (% of energy), fat intake (% of energy), total sugar intake (% of energy), alcohol intake (% of energy), and dietary fiber intake (g/10 MJ, continuous).	MDS or HDI: Men ▪ Positive ($p \leq 0.02$): MF-energy% (MDSβ = 0.27, HDIβ = 0.26). ▪ Negative ($p < 0.01$): SF-energy% (MDSβ = −0.12, HDIβ = −0.09), SF-time (MDSβ = −0.19, HDIβ = −0.17). Women ▪ Positive ($p < 0.05$): MF-time (MDSβ = 0.14, HDIβ = 0.12), MF-energy% (MDSβ = 0.27). ▪ Negative ($p \leq 0.02$): SF-time (MDSβ = −0.36, HDIβ = −0.15), SF-energy% (MDSβ = −0.11). BMI or WC: Men ▪ Positive ($p < 0.05$): MF-time (BMIβ = 0.33, WCβ = 0.85), SF-time (BMIβ = 0.36, WCβ = 0.87) and SF-energy% (BMIβ = 0.32, WCβ = 0.77).Women ▪ Positive ($p \leq 0.02$): SF-time (BMIβ = 0.88, WCβ = 2.09) and SF-energy% (BMIβ = 0.4, WCβ = 0.69).

Table 2. *Cont.*

Cross-Sectional Studies					
Murakami 2016 [11].	▪ American. ▪ *n* = 19,427 (9826 women and 9601 men). ▪ ≥20 years.	Eating frequency: all eating occasions (kcal >50). Meal and snack frequencies: ▪ Self-reported (meals: breakfast, brunch, lunch, and dinner; others: snacks) ▪ Based on time (meals: 6–9 h, 12–14 h and 17–20 h; snack: other). ▪ Based on energy percentage contribution (meal: ≥ 15%, snack: < 15% of total energy).	DQ (HEI-2010).	Age group, race and ethnicity, years of education, family poverty income ratio, smoking, any recreational PA, weight status, dietary reporting status, and survey cycle.	Every additional eating occasions was positively associated to DQ in all measures ($p < 0.01$): Men: ▪ EF (β = 1.77). ▪ MF-%energy (β = 4.09), MF-self-report (β = 4.22) and MF-clock time (β = 2.14). ▪ SF-%energy (β = 1.52), SF-self-report (β = 1.26) and SF- clock time (β = 1.28). Women: ▪ EF (β = 2.22). ▪ MF-%energy (β = 3.62), MF-self-report (β = 5.35) and MF-clock time (β = 2.70). ▪ SF-%energy (β = 1.97), SF-self-report (β = 1.52) and SF- clock time (β = 1.57).
Leech 2016 [57].	▪ Australian. ▪ *n* = 4323 (2270 women and 2053 men). ▪ Age: ≥19 years.	Eating frequency: ▪ Eating occasion: food/beverages with ≥ 210 kJ (1–3, 4–5 or ≥6). ▪ Meals: breakfast, brunch, lunch, dinner, or supper (1–2 or ≥ 3). ▪ Snacks: 0–1, 2–3 or ≥ 4.	DQ (DGI-2013).	Age, education, income, country of birth, PA, total sedentary time, smoking, alcohol, currently dieting, eating more or less than usual, and ratio of reported total energy intake.	▪ Eating occasion: men (β = 1.38 [95% CI: 0.71, 2.05]) and women (β = 1.12 [95% CI: 0.34, 1.90]). ▪ MF: men (β = 5.60 [95% CI: 3.89, 7.34]) and for women (β = 4.11 [95% CI: 2.23, 5.93]).
House 2018 [63].	▪ American (Hispanic). ▪ *n* = 92 (47 women and 45 men). ▪ Age: 18–19 years.	Eating frequency: infrequent (<3 meals/day) or frequent (>4 meals/day).	BMI, BMI z-score, BW, WC.	Age, sex and percent time spent in moderate to vigorous physical activity.	Infrequent eaters vs. frequent eaters: ▪ Total population: > BMI ($p = 0.02$) and BMI z-score ($p = 0.03$). In stratified analyses by sex: ▪ Women: > BMI ($p = 0.04$). Other variables: NA.

Table 2. *Cont.*

Cross-Sectional Studies					
Kim 2018 [23].	■ South Korean. ■ n = 6951 (3487 women and 3464 men). ■ Age: 19–93 years.	■ Eating frequency: all eating occasions (<3, 4 or ≥5). ■ Meal frequency: 1, 2 or 3. ■ Snack frequency: 0, 1, 2 or 3.	BMI, WC, DQ (MAR).	Age group, sex, smoking, alcohol drinking frequency, PA, resistance PA frequency, household income, education level, stress level, EI, depressed mood, meal frequency, and snack frequency.	In ≥ 5 EF vs. < 3 EF: ■ <BMI ($p < 0.01$) and WC ($p < 0.01$). ■ <BMI ($p < 0.01$) and WC ($p < 0.01$) in highest DQ. 3 MF vs. 2 MF:■ <BMI ($p < 0.04$) and WC ($p < 0.01$). ■ <WC ($p < 0.01$) in highest DQ. 0 SF vs. 2 SF: ■ <BMI ($p < 0.01$) and WC ($p < 0.01$). ■ <BMI ($p < 0.01$) in highest DQ.
Alamri 2020 [59].	■ Saudi. ■ n = 435 women. ■ Age: 20–25 years.	Snack frequency: Other eating occasions besides breakfast, lunch, or dinner.	WC ≤ 88 cm, WC > 88 cm, BMI 18.5 to < 25 and BMI ≥ 25.	None.	■ > SF in the evening: > frequent in WC> 88 cm ($p = 0.04$) and BMI≥ 25 ($p = 0.04$). ■ Other variables: NA.

Abbreviations: AO, abdominal obesity (waist-height > 0.5); β, beta coefficient; BMI, body mass index; BP, blood pressure; BW, body weight; CASPIAN-IV, childhood and adolescence surveillance and prevention of adult non-communicable disease; DBP, diastolic blood pressure; DGI, dietary guidelines index; DQ, diet quality; DQS, dietary quality score; EF, eating frequency; ELFA, early life factors adjustment; HC, hip circumference; HDI, health diet indicator; HDL-c, high density lipoprotein cholesterol; HT, hypertension; INTERMAP, international study of macro-and micro-nutrients; IPAQS, international physical activity questionnaire score; LCFA, later childhood factors adjustment; MAR, mean adequacy ratio; MDS, Mediterranean diet score; MF, meal frequency; NA, no association; OB, obesity; OR, odds ratio; OW, overweight; PA, physical activity; r, PANIC, physical activity and nutrition in children; Pearson's correlation; SBP, systolic blood pressure; SF, snack frequency; SOLAR, study of Latino adolescents at risk for diabetes; SPEEDY study; sport, physical activity and eating behavior: environmental determinants in young people; TG, triglycerides; vs., versus; WC, waist circumference.

MetS components as outcomes were examined in four cross-sectional studies. American and Finnish boys with higher eating frequency had significantly lower serum triglycerides concentrations (OR = 0.48; 95% CI: 0.26, 0.89) [29]. No additional associations with MetS components were found [10,25,29,32].

Evidence relating to diet quality in children and adolescents showed significant positive associations among eating frequency, meal frequency, or snack frequency and diet quality [15,51]. Other authors found significant negative associations with eating frequency [17], meal frequency [17], and snack frequency [17,51].

Adults

In total, 11 cross-sectional studies examined associations between eating frequency and adiposity in adults. A greater eating frequency was significantly associated with lower body weight [61], lower BMI [23,62,63,65] and lower waist circumference compared to a lower eating frequency [23,65]. Research conducted in British and Saudi participants [16,59,64] reported significant positive associations between snack frequency [16,59] or meal frequency [16] and the prevalence of overweight or obesity. In a study conducted by Fábry et al. [75], lower meal frequency was significant associated with higher overweight prevalence.

Individuals with high meal frequency [16] or snack frequency [16,64] have been reported to present with larger WC. In stratified analyses by weight status, men with BMI $\leq$ 25 kg/m^2 were reported to have a negative association between snack frequency and WC (β = $-$0.52, 95% CI: $-$0.90, $-$0.14) [64]. This was not observed in women, however, in women with a BMI $\geq$ 25 kg/m^2 a higher snack frequency was positively associated with WC (β = 0.80, 95% CI: 0.34, 1.26). In this study, women with waist circumference >88 cm showed a significantly greater snack frequency during the evening [64]. In South Korean participants, higher total eating frequency, but not the frequency of eating snacks, was significantly associated with lower BMI in participants with higher diet quality [23]. House et al. [63] reported that a significantly higher BMI z-score was observed in participants eating three or less meals per day compared to those eating four daily meals. Other authors [60,61,63] have reported no significant associations between eating frequency and BMI, body weight or WC. Kim et al. [22] reported that South Korean participants with a high frequency of eating and snacking and abdominal obesity had a 50% lower risk of having hypertension (OR = 0.5; 95% CI: 0.31, 0.82). Participants with high snack frequency and low diet quality also showed 50% lower risk of hypertension (OR = 0.5; 95% CI: 0.23, 0.89) compared to those with no snack consumption.

Seven cross-sectional studies assessed associations between meal or snaking frequency and diet quality. Significant positive associations were observed between eating frequency, meal frequency or snack frequency and diet quality in six studies [11,16,57,58,65,66]. A significant but negative association between diet quality and distinct assessment methods for snack frequency have also been reported [16]. In one study, no associations between these exposures and diet quality were found [60].

3.4. Longitudinal Studies

3.4.1. Eating Speed/Rate

Table 3 shows longitudinal studies exploring the associations between eating speed/rate and adiposity outcomes.

Table 3. Characteristics and main findings of longitudinal studies that explored eating speed/rate.

Longitudinal Studies					
Children					
Author and Year	**Population**	**Exposure (s)**	**Outcome (s)**	**Adjusted Variables**	**Results**
Okubo 2017 [28].	▪ Osaka Maternal and Child Health Study. ▪ Japanese. ▪ *n* = 492 mother-child pairs. ▪ Age: 30–42 m. ▪ 1 year follow-up.	Eating rate (self-reported).	BMI.	Child's sex and age (at fourth survey), maternal age and BMI at enrollment, education level, family income, pregnancy smoking status, maternal working status at 30 months postpartum, birth order, birth weight, duration of breast-feeding, time spent watching TV at 30 months of age, protein intake, fat intake, and dietary fiber at 30 months of age.	Fast eating rate vs. slow eating rate at 30 m.: ▪ BMI at 42 m. (β = 0.67 [95% CI: 0.24, 1.10]).
Adults					
Author and Year	**Population**	**Exposure (s)**	**Outcome (s)**	**Adjusted Variables**	**Results**
Tanihara 2011 [9].	▪ Japanese. ▪ *n* = 529 men. ▪ Age: 20–59 years. ▪ 8 year follow-up.	Eating speed (self-reported).	OW.	Age, drinking, smoking, regular exercise.	▪ Fast vs. medium or slow eating speed: OW (OR = 1.80 [95% CI: 1.25, 2.59]).
Yamane 2014 [2].	▪ Japanese. ▪ n = 1314 (638 women and 676 men). ▪ Pre-universities. ▪ 3 year follow-up.	Eating quickly.	OW.	Gender, eating quickly, frequently consuming fatty foods.	▪ Eating quickly vs. no eating quickly: OW (OR = 4.40 [95% CI: 2.22, 8.75]).
Zhu 2015 [5].	▪ Japanese ▪ *n* = 8941 (5517 women and 3424 men). ▪ Age: 40–75 years. ▪ 3 year follow-up.	Eating speed (self-reported).	MetS, WC, HDL-c, TG, BP and FPG.	Age and sex, drinking alcohol, dietary behavior, physical activity, sleeping, and Medication history.	▪ Fast eating speed vs. not fast eating speed: HR for MetS (HR = 1.30 [95% CI: 1.05, 1.60]), WC (HR = 1.35 [95% CI: 1.10, 1.66]) and lower HDL-c (HR = 1.37 [95% CI: 1.12, 1.67]). ▪ Other variables: NA.
Leong 2016 [56].	▪ New Zealander. ▪ *n* = 1014 women. ▪ Age: 40–50 years. ▪ 3 year follow-up.	Eating speed (self-reported).	OW.	Baseline BMI, age, socioeconomic status, thyroid condition, ethnicity, change in physical activity, change in smoking status and change in menopause status.	▪ No significant associations for OW.

Abbreviations: β, beta coefficient; BMI, body mass index; BP, blood pressure; FPG, fasting plasma glucose; HDL-c, high density cholesterol; HR, hazard ratio; M, months of age; MetS, metabolic syndrome; NA, no associations; OB, obesity; OR, odds ratio; OW, overweight; TG, triglycerides; vs., versus; WC, waist circumference.

Children

Only one longitudinal study met inclusion criteria. After 1 year of follow-up, Okubo et al. [28] reported a positive association between rate of eating at 30 months of age and overweight or obesity at 42 months of age (β = 0.67; 95% CI: 0.24–1.10).

Adults

All of the longitudinal studies included explored adiposity measures but only one examined MetS components. Tanihara et al. [9] and Yamane et al. [2] observed that a fast eating speed/rate was associated with a significantly increased risk of being overweight, however in another study conducted in New Zealand [56] no significant associations were reported. Fast eating speed/rate was also associated with a 35% increased risk of developing increased WC (hazard ratio (HR) = 1.35; 95% CI: 1.10–1.66) [5], a 37% higher risk for developing low HDL-c (HR = 1.37; 95% CI: 1.12–1.67) and were 30% more likely to experience MetS incidence (HR = 1.30; 95% CI: 1.05–1.60) in a Japanese population [5].

3.4.2. Eating Frequency

Table 4 shows the characteristics of the longitudinal studies included examining the associations between eating frequency and adiposity, MetS or diet quality.

Children

Three longitudinal studies explored the association between eating frequency and anthropometric measures. In two studies, each with a 10-year follow-up, eating frequency was shown to be significantly inversely associated with WC [30], BMI z-score [31] and BMI [30]. Ritchie et al. [30], reported that greater meal frequency was associated with higher BMI in American female adolescents. Additional associations for BMI z-score and overweight were not observed [4,31].

Adults

Two longitudinal articles exploring the association of eating frequency with adiposity met inclusion criteria. Kahleova et al. [67] conducted a 7-year follow-up study, reporting a positive association between eating frequency and changes in BMI (β = 0.04; 95% CI: 0.02–0.06). Whereas, Larsen et al. [74] observed that higher baseline meal frequency was associated with a decrease in BMI (β = −0.14; 95% CI: −0.27–0.00) and waist circumference (β = −0.49; 95% CI: −0.99–0.00) during a 6-year follow-up [74].

3.5. Interventional Trials

3.5.1. Eating Speed/Rate

Table 5 summarizes the details of the intervention trials that met inclusion criteria assessing the effect of eating speed/rate on adiposity measures, metabolic syndrome, and diet quality.

Children

Only one intervention trial met inclusion criteria. Faith et al. [12] conducted an 8-week parallel study in 24 American children aged 4 to 8 years old. The participants in the intervention group attended interactive sessions focused on decelerate eating speed and were encouraged to eat slower through the use of timers and the performance of interactive activities with their families during eating occasions. The eating speed decreased in the intervention group and was associated with significantly lower BMI and BMI z-score.

Adults

No intervention trials met inclusion criteria assessing eating speed/rate in adults.

3.5.2. Eating Frequency

Table 6 shows the characteristics and relevant results from intervention articles investigating the effect of eating frequency on adiposity measures, MetS, and diet quality.

Table 4. Characteristics and main findings of longitudinal studies that explored eating frequency.

Longitudinal Studies					
Children					
Author and Year	**Population**	**Exposure (s)**	**Outcome (s)**	**Adjusted Variables**	**Results**
Franko 2008 [31].	■ American (black and white race). ■ *n* = 2375 girls. ■ Age: 9–10 years. ■ 10 year follow-up.	Meal frequency: number of days consumed ≥ 3 meals (breakfast, snack, lunch or other).	BMI-for-age z-score and OW.	Visit, study site, parental education, socioeconomic status, race, energy intake and indicators of physical activity.	■ MF: BMI-for-age z score (β = −0.047, $p < 0.01$).
Ritchie 2012 [30].	■ NGHS study. ■ American (black and white race). ■ *n* = 2372 girls. ■ Age: 9–10 years. 10 year follow-up.	Eating frequency: ■ Eating episode: 1–3, 3.1–4, 4.1–6 or >6 per day. ■ Meal: 1–2.5 or > 2.5 meals per day. ■ Snack: 0–1, 1.1–2, 2.1–3 or > 3 per day.	BMI and WC.	BMI or WC, race, parental education, physical activity, Television/video viewing, and total energy intake, dieting for weight loss.	■ >6 total eating episodes/day: <BMI ($p = 0.01$) and WC ($p = 0.04$). ■ >2.5 MF: >BMI ($p = 0.04$).
Taylor 2017 [4].	■ New Zealander. ■ *n* = 371 (175 girls and 196 boys). ■ Age: 1–3.5 years. ■ 3.5 year follow-up	Eating frequency (all eating occasions).	BMI z-score.	POI intervention group, household factors, maternal parity/education, infant sex, birth weight, pre-pregnancy BMI, pregnancy smoking, and exclusive breastfeeding.	No significant associations.
Adults					
Author and Year	**Population**	**Exposure (s)**	**Outcome (s)**	**Adjusted Variables**	**Results**
Kahleova 2017 [67].	■ AHS-2. ■ North American. ■ *n* = 50,660. ■ Age: ≥ 30 years. ■ 7 ± 1 year follow-up.	Meal frequency and timing: ■ Breakfast: from 5–11 h. ■ Lunch: from 12–16 h. ■ Dinner: from 17–23 h.	BMI.	Age, sex, ethnicity, marital status, education, personal income, dietary pattern, exercise, sleep, television watching, energy intake, and high blood pressure medicine.	■ ≥6 MF vs. 3 MF: BMI (β = 0.04 [95% CI: 0.02, 0.06]).
Larsen 2019 [74].	■ MONICA study. ■ Danish. ■ *n* = 2124 (1044 women and 1080 men). ■ Middle-aged. ■ 6 year follow-up.	Total eating, meal and snack frequency (self-reported).	BMI and WC.	Baseline measure of outcome, smoking, alcohol, PA, education, age, gender, menopausal status for women, and height (in WC analysis only).	Baseline MF: ■ 6-y. change: BMI (β = −0.14 [95% CI: −0.27, 0.00]) and WC (β = −0.49 [95% CI: −0.99, 0.00]).

Abbreviations: ADHS-2, Adventist Health Study 2; β, beta coefficient; BMI, body mass index; EF, eating frequency; MF, meal frequency; MONICA, Danish Monitoring Trends and determinants in Cardiovascular Disease; NA, no associations; NGHS, National Heart, Lung, and Blood Institute Growth and Health Study; OR, odds ratio; OW, overweight; PA, physical activity; POI, Prevention of Overweight in Infancy; vs., versus; WC, waist circumference.

Table 5. Characteristics and main findings of interventional trials that explored eating speed/rate.

Interventional Trials						
Children						
Author and Year	**Study Design**	**Population**	**Intervention Description**	**Comparing Group**	**Adjusted Variables**	**Results**
Faith 2019 [12].	Parallel: 8 weeks.	▪ American. ▪ *n* = 24 girls and boys. ▪ Age: 4–8 years.	RePace: ▪ 5 interactive sessions of 1 h for parents and children, over 8 weeks. ▪ Small timers were given at the clinic, programmed to vibrate at 30-s intervals during the mealtimes and snacks. ▪ Chat Jar during mealtimes to encourage a slower eating. DUC (at the end of the 8 weeks): ▪ 30 min of informative educational sessions (healthy eating recommendations, importance of family meals, slowly eating speed).	RePace or DUC.	Child age, sex and baseline BMI and BMI z-score.	▪ Intervention: BMI ($\beta = -0.57$, $p = 0.02$) and BMI z-score ($\beta = -0.23$, $p = 0.03$).

Abbreviations: β, beta coefficient; BMI, body mass index; DUC, delayed usual care; RePace, reduced eating pace.

Table 6. Characteristics and main findings of interventional trials that explored eating frequency.

Interventional Trials						
Adults						
Author and Year	**Study Design**	**Population**	**Intervention Description**	**Comparing Group**	**Adjusted Variables**	**Results**
Carlson 2007 [70].	Crossover: 18 weeks.	■ American. ■ *n* = 15 (10 women and 5 men). ■ Age: 40–50 years.	■ Controlled diet: 3 meals/day: breakfast, lunch and dinner or 1 meal/day: during 4 Hours in the early evening (16–20 h). ■ 11-weeks off-diet.	3 MF or 1 MF/day.	Period-specific baseline values.	■ 3 MF vs. 1 MF: < FPG (p < 0.01).
Stote 2007 [72].	Crossover: two 8 weeks periods.	■ American. ■ *n* = 15 (10 women and 5 men). ■ Age: 40–50 years.	1 meal/day: during a 4 h period in the early evening.	3 MF/day (breakfast, lunch and dinner).	First observation within a period.	3 MF vs. 1 MF: ■ < SBP (p = 0.02) and DBP (p = 0.04). ■ > BW (p = 0.01). 3 MF vs. 1 MF/day: ■ < HLD-c (p = 0.01). ■ FPG and TG: NA.
Kanaley 2014 [68].	Crossover: 3 days with a 12 h period each one.	■ American. ■ *n* = 14 (11 women and 3 men). ■ Age: 20–59 years.	■ Low frequency (3MF), high frequency (6MF) or high frequency + high protein (6MFHP). ■ Wash out: 1 month between each study day.	Low frequency, high frequency or high frequency + protein regimen.	None.	■ 6 MFHP vs. 3 MF or 6 MF: < FPG tAUC (p < 0.01). ■ 3 MF vs. 6 MF: NA for FPG.
Alencar 2015 [69].	Crossover: 6-weeks.	■ American. ■ *n* = 11 women. ■ Age: 35–60 years.	2 MF pattern (every 5–6 h), 6 MF pattern: (every 2–3 h) or washout phase (4 MF).	2 MF pattern, 6 MF pattern or washout phase.	None.	■ 2 MF vs. 6 MF: > HDL-c (p < 0.05).
Megson 2017 [71].	Parallel: 3 months.	■ American. ■ *n* = 211 (176 women and 35 men). ■ Age: 18–70 years.	■ Group SURI program: website + a pedometer + periodic newsletters; community exercise programs + prizes and recognition. ■ Group SURI + IBWL: SURI program + internet Behavioral weight loss program. ■ Group SURI + IBWL + Group: included Weekly group meetings.	SURI program, SURI + IBWL and SURI + IBWL + Group.	Treatment arm.	■ EF: NA for BW loss.
Yildiran 2019 [73].	Parallel: 3 months.	■ Turkish. ■ *n* = 47 women. ■ Age: 20–49 years.	3 meals/day (3 main meals) or 6 meals/day (3 main meals + 3 snacks).	3 MF or 6 MF/day.	None.	■ Not significant effects in BMI, WC, BW, FPG, TG, HDL-c between groups.

Abbreviations: BMI, body mass index; BW, body weight; DBP, diastolic blood pressure; EF, eating frequency; FPG, fasting plasma glucose; HDL-c, high density lipoprotein cholesterol; HMF, high meal frequency; IBWL, Internet behavioral weight loss program; LMF, low meal frequency; MF, meal frequency; NA, no association; NE, no effect; PPG, postprandial peak of glucose; SBP, systolic blood pressure; SURI, Shape Up Rhode Island; tAUC, total area under the curve; TG, triglycerides; vs., versus; WC, waist circumference.

Children

No intervention trials article met inclusion criteria assessing eating frequency in children.

Adults

Three intervention trials investigated the effect of eating frequency on measures of adiposity. Stote et al. [72] conducted a crossover study in Americans, where the intervention consisted of either one or three daily meals for two 8-week periods. Compared with one daily meal, the group consuming the three daily meals pattern had a significantly higher body weight. In a Turkish study, different dietary patterns showed no effects for WC or BMI [73]. Similarly, an American trial studying meal frequency also did not observe a significant effect on body weight, WC or BMI [71]. Diet quality was not examined in any intervention trial that met inclusion criteria for adults.

The impact of eating frequency on MetS components was examined in seven intervention trials. Stote et al. [72] reported that greater meal frequency resulted in a significantly lower blood pressure and HDL-c. An American study [69], comparing consumption of two or six daily meals for a total of 6 weeks reported that low-eating frequency induced higher HDL-c levels. In an American population, compared with one daily meal, three meals per day led to a significantly lower fasting plasma glucose in a study that lasted 18 weeks [70]. Kanaley et al. [68] conducted a study in Americans in which the main interventions were a dietary pattern consisting of three meals, six meals or six high protein meals per day. Compared to the dietary patterns involving three meals or six meals, eating six high protein daily meals led to lower levels of fasting plasma glucose [68]. Other studies [72,73] reported no significant effects of different eating frequency patterns on MetS components.

4. Discussion

The findings of this review suggest that a faster eating speed/rate could be associated with an increased risk of adiposity and MetS or its components. Furthermore, a greater eating frequency may be mainly associated with diet quality and lower risk of adiposity and MetS or its components.

Regarding eating speed/rate in children, some cross-sectional studies concluded that eating speed was positively associated with overweight [26,27] and positively correlated to WC [24]. Similar results were also reported in a longitudinal [28] and in an interventional trial [12]. Associations between eating frequency and adiposity (BMI, BMI z-score, waist circumference and body weight) in children were mainly inversely associated in cross-sectional [25,26,29] and longitudinal studies [30,31]. However, some authors reported positive associations between eating frequency and BMI z-score in adolescents [10,17] and in children with central obesity [14]. In adults self-reported faster eating speed was frequently associated with higher risk of overweight or obesity and larger WC [13,20] in cross-sectional studies but only overweight was associated with faster eating speed/rate in longitudinal studies [2,9]. However, an observational study reported positive partial correlations between objective eating speed with BMI, body weight, and WC [53]. Regarding eating frequency, inverse associations were reported in cross-sectional and longitudinal studies with adiposity indicators [74,75], except for a few observational and interventional studies that otherwise reported positive associations [16,64,72]. Differences in the findings among studies could be partly explained by gender-specific lifestyle factors such as body composition, physical activity, hormone function, and dietary habits [76]. Moreover, cardiovascular risk factors may coexist simultaneously, increasing the risk of developing additional related metabolic alterations as a result of synergic effects [19]. In childhood, it has been proposed by some authors that higher parenthood control [77,78] and consequently feeding restriction, as well as an improvement in energy compensation, which declines with age [14] may contribute to disparities in adiposity, BMI z-score, or body weight for age until 5 years old [79].

With regard to diet quality, few cross-sectional studies reported that higher eating frequency, snack frequency and meal frequency improved diet quality in American children

and adolescents [15,51]. On the contrary, inverse associations between snack frequency and diet quality were reported mainly in teenagers [17,51]. Similar to in adulthood, more autonomy in food choices prevail in adolescence, and this may play an important role in the quality of the diet, especially in regard to snacking. [51]. In adults, higher diet quality was mainly positively associated with the number of eating occasions [16,57,58,65]. However, one author reported inverse results specifically for snack frequency [16]. It should be noted that snack frequency was usually defined as the number of eating occasions outside of main meals [16] and that differed from meals based on the amount of energy contributed. Nowadays a harmonized, globally accepted criterion for diet quality has not been defined, perhaps due to differences in culture and traditions. However, several organizations have published dietary guidelines that describe specific indicators, groups of food or even healthy dietary patterns, which may be used to fit *a priori* diet quality indexes as were described in the articles included in this review.

With reference to MetS and its components, observational studies conducted in adults showed that eating speed/rate was significantly associated with MetS onset [5,19,55], high fasting plasma glucose, triglycerides, blood pressure [18,19,21], and low HDL-c [18,55]. In contrast, distinct assessment methods of eating frequency were inversely associated with lower risk of hypertension in adults with abdominal obesity or with lower diet quality [22]. Furthermore, in interventional trials, a greater eating frequency was associated with lower fasting plasma glucose [68,70].

The potential mechanism by which eating behaviors (eating speed/rate or eating frequency) are linked to adiposity and cardiometabolic risk factors remain unclear. Eating is a complex physiological act influenced by multiple endogenous and exogenous factors. Previous studies highlight mastication [27], bite frequency [80,81], oral sensory exposure to meals and food texture as potential determinants for feeding regulation, chiefly by inducing gastrointestinal hormonal secretion [82,83]. In this process, numerous gastrointestinal hormones are involved but mainly ghrelin, peptide YY, leptin and insulin [82], are noted to play a key role in energy intake, [84] adiposity and metabolism [85]. Leptin and insulin are classified as long-acting adiposity signals due to their role in lipid and glucose metabolism [86], with an important influence on adiposity regulation [82]. While ghrelin determines meal initiation and peptide YY induces a decrease in appetite [87]. In fact, an increase in peptide YY has been reported with slowing eating speed [83]. Additionally, it has been suggested that longer oro-sensory exposure may promote greater satiation and therefore, a protective effect on overfeeding [24]. Moreover, eating frequency has been related to satiety [88] and thermic effect of food, which has the potential to impact metabolic rate [63]. Additionally, a lower eating frequency has been associated with reduced insulin sensitivity caused by a higher insulin response to meals in irregular eaters [22]. This is related to overactivation of the sympathetic nervous system, excess of angiotensinogen secretion and renal sodium retention with evident effects in blood pressure.

Despite the potential effects of eating behaviors (eating speed and eating frequency), it is already known that the onset of overweight, obesity, and MetS is the result of a multifactorial etiology [89], such as gene–environment factors [3] (including weight gained during pregnancy [29] or parental BMI [28]), type of population [32], sleep duration [29], ethnicity [90], parental income [26] and physical activity [30]. Furthermore, we cannot disregard the influence of differences in methodology (follow-up periods, intervention group, age, socioeconomic status, total energy intake, cultural differences in dietary habits, etc.) among studies that might explained the discrepancies observed in the present findings.

In the present review we sought to synthetize the available evidence regarding eating behaviors in relation to adiposity, MetS components and diet quality. However, this review is not without limitations. First, due to its design, cause–effect associations in cross-sectional and longitudinal studies are not possible to discern and we cannot disregard that relevant articles may exist that were not included. Secondly, drawing firm conclusions is difficult due to the great variability and heterogeneity between articles, the lack of consensus about eating speed/rate and eating frequency definitions, as well as differences in overweight

or obesity definitions used for children. Third, eating speed/rate was explored, in the vast majority of studies, subjectively through self-reported questionnaires, which may not reflect true eating speed/rate [53]. Moreover, BMI was used frequently to assess adiposity despite its several limitations [91]. Similarly, it is important to highlight that one study used an out-of-date definition of overweight [75]. A special consideration about the criteria used to define metabolic syndrome is that is has undergone several variations since 1999. However, all of the articles included in the present review used the harmonized criteria established by Alberti et al., in 2009 except for one study which used the ATPIII criteria. Likewise, it is necessary to highlight the limited number of studies conducted in children, and the scarcity of longitudinal and interventional trials exploring associations between eating speed/rate or meal frequency and diet quality.

5. Conclusions

Limited evidence suggests an association between faster eating speed/rate, adiposity and increased risk of developing MetS. While a higher eating frequency is associated with lower adiposity, better diet quality and lower MetS risk. However, more long-term and interventional trials are warranted in the future to clarify these associations and the mechanisms by which they affect adiposity and cardiometabolic health. If these associations were proven, strategies focused on eating behaviors in early life, such as eating speed/rate and eating frequency may be recommended for the prevention of excess body weight, cardiovascular risk, and metabolic disease development.

Author Contributions: N.B. and J.S.-S. conceived the idea for the manuscript and study design. T.E.G.-F., I.P.-G., S.K.N., N.B. and J.S.-S. contributed to data interpretation and led the writing of the Section 4 of the manuscript. S.K.N. contributed to improve the English grammar in the manuscript. All authors reviewed the manuscript for important intellectual content and approved the final version to be published. All authors have read and agreed to the published version of the manuscript.

Funding: This research was funded by "Consejo Nacional de Ciencia y Tecnología" (Government of Mexico) with grant number 769789 (T.E.G.-F.). I.P.-G. received a grant from the Spanish Ministry of Education, Culture, and Sports (FPU 17/01925). S.K.N. was supported by a Canadian Institutes of Health Research (CIHR) fellowship (grant MFE-171207). J.S.-S., senior author of this study, is partially supported by ICREA under the ICREA Academia program. Authors gratefully acknowledge the financial support of Universitat Rovira i Virgili (2021OPEN).

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Conflicts of Interest: The authors declare no conflict of interest.

References

1. World Health Organisation (WHO). Obesity and Overweight. Available online: https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight (accessed on 4 November 2020).
2. Yamane, M.; Ekuni, D.; Mizutani, S.; Kataoka, K.; Sakumoto-Kataoka, M.; Kawabata, Y.; Omori, C.; Azuma, T.; Tomofuji, T.; Iwasaki, Y.; et al. Relationships between eating quickly and weight gain in Japanese University students: A longitudinal study. *Obesity* **2014**, *22*, 2262–2266. [CrossRef]
3. Llewellyn, C.; Wardle, J. Behavioral susceptibility to obesity: Gene-environment interplay in the development of weight. *Physiol. Behav.* **2015**, *152*, 494–501. [CrossRef]
4. Taylor, R.W.; Iosua, E.; Heath, A.L.M.; Gray, A.R.; Taylor, B.J.; Lawrence, J.A.; Hanna, M.; Cameron, S.L.; Sayers, R.; Galland, B. Eating frequency in relation to BMI in very young children: A longitudinal analysis. *Public Health Nutr.* **2017**, *20*, 1372–1379. [CrossRef]
5. Zhu, B.; Haruyama, Y.; Muto, T.; Yamazaki, T. Association between eating speed and metabolic syndrome in a three-year population-based cohort study. *J. Epidemiol.* **2015**, *25*, 332–336. [CrossRef] [PubMed]
6. Mok, A.; Haldar, S.; Lee, J.C.Y.; Leow, M.K.S.; Henry, C.J. Postprandial changes in cardiometabolic disease risk in young Chinese men following isocaloric high or low protein diets, stratified by either high or low meal frequency—A randomized controlled crossover trial. *Nutr. J.* **2016**, *15*. [CrossRef] [PubMed]
7. Jahnke, D.L.; Warschburger, P.A. Familial transmission of eating behaviors in preschool-aged children. *Obesity* **2008**, *16*, 1821–1825. [CrossRef] [PubMed]

8. Munsters, M.J.M.; Saris, W.H.M. Body weight regulation and obesity: Dietary strategies to improve the metabolic profile. *Annu. Rev. Food Sci. Technol.* **2014**, *5*, 39–51. [CrossRef]
9. Tanihara, S.; Imatoh, T.; Miyazaki, M.; Babazono, A.; Momose, Y.; Baba, M.; Uryu, Y.; Une, H. Retrospective longitudinal study on the relationship between 8-year weight change and current eating speed. *Appetite* **2011**, *57*, 179–183. [CrossRef]
10. Murakami, K.; Livingstone, M.B.E. Associations of eating frequency with adiposity measures, blood lipid profiles and blood pressure in British children and adolescents. *Br. J. Nutr.* **2014**, *111*, 2176–2183. [CrossRef]
11. Murakami, K.; Livingstone, M.B.E. Associations between Meal and Snack Frequency and Diet Quality in US Adults: National Health and Nutrition Examination Survey 2003–2012. *J. Acad. Nutr. Diet.* **2016**, *116*, 1101–1113. [CrossRef] [PubMed]
12. Faith, M.S.; Diewald, L.K.; Crabbe, S.; Burgess, B.; Berkowitz, R.I. Reduced Eating Pace (RePace) Behavioral Intervention for Children Prone to or with Obesity: Does the Turtle Win the Race? *Obesity* **2019**, *27*, 121–129. [CrossRef] [PubMed]
13. Van den Boer, J.H.W.; Kranendonk, J.; van de Wiel, A.; Feskens, E.J.M.; Geelen, A.; Mars, M. Self-reported eating rate is associated with weight status in a Dutch population: A validation study and a cross-sectional study. *Int. J. Behav. Nutr. Phys. Act.* **2017**, *14*. [CrossRef]
14. Jennings, A.; Cassidy, A.; Van Sluijs, E.M.F.; Griffin, S.J.; Welch, A.A. Associations between eating frequency, adiposity, diet, and activity in 9–10 year old healthy-weight and centrally obese children. *Obesity* **2012**, *20*, 1462–1468. [CrossRef]
15. Murakami, K.; Livingstone, M.B.E. Meal and snack frequency in relation to diet quality in US children and adolescents: The National Health and Nutrition Examination Survey 2003–2012. *Public Health Nutr.* **2016**, *19*, 1635–1644. [CrossRef]
16. Murakami, K.; Livingstone, M.B.E. Associations between meal and snack frequency and diet quality and adiposity measures in British adults: Findings from the National Diet and Nutrition Survey. *Public Health Nutr.* **2016**, *19*, 1624–1634. [CrossRef]
17. Murakami, K.; Livingstone, M.B.E. Decreasing the number of small eating occasions (<15% of total energy intake) regardless of the time of day may be important to improve diet quality but not adiposity: A cross-sectional study in British children and adolescents. *Br. J. Nutr.* **2016**, *115*, 332–341. [CrossRef] [PubMed]
18. Lee, K.S.; Kim, D.H.; Jang, J.S.; Nam, G.E.; Shin, Y.N.; Bok, A.R.; Kim, M.J.; Cho, K.H. Eating rate is associated with cardiometabolic risk factors in Korean adults. *Nutr. Metab. Cardiovasc. Dis.* **2013**, *23*, 635–641. [CrossRef]
19. Tao, L.; Yang, K.; Huang, F.; Liu, X.; Li, X.; Luo, Y.; Wu, L.; Guo, X. Association between self-reported eating speed and metabolic syndrome in a Beijing adult population: A cross-sectional study. *BMC Public Health* **2018**, *18*. [CrossRef]
20. Lee, J.S.; Mishra, G.; Hayashi, K.; Watanabe, E.; Mori, K.; Kawakubo, K. Combined eating behaviors and overweight: Eating quickly, late evening meals, and skipping breakfast. *Eat. Behav.* **2016**, *21*, 84–88. [CrossRef] [PubMed]
21. Paz-Graniel, I.; Babio, N.; Mendez, I.; Salas-Salvadó, J. Association between eating speed and classical cardiovascular risk factors: A cross-sectional study. *Nutrients* **2019**, *11*, 83. [CrossRef]
22. Kim, S.; Park, G.H.; Yang, J.H.; Chun, S.H.; Yoon, H.J.; Park, M.S. Eating frequency is inversely associated with blood pressure and hypertension in Korean adults: Analysis of the Third Korean National Health and Nutrition Examination Survey. *Eur. J. Clin. Nutr.* **2014**, *68*, 481–489. [CrossRef]
23. Kim, S.; Yang, J.H.; Park, G.H. Eating frequency is inversely associated with BMI, waist circumference and the proportion of body fat in Korean adults when diet quality is high, but not when it is low: Analysis of the Fourth Korea National Health and Nutrition Examination Survey (KNHANE IV). *Br. J. Nutr.* **2018**, *119*, 918–927. [CrossRef]
24. Fogel, A.; Goh, A.T.; Fries, L.R.; Sadananthan, S.A.; Velan, S.S.; Michael, N.; Tint, M.-T.; Fortier, M.V.; Chan, M.J.; Toh, J.Y.; et al. Faster eating rates are associated with higher energy intakes during an ad libitum meal, higher BMI and greater adiposity among 4·5-year-old children: Results from the Growing Up in Singapore Towards Healthy Outcomes (GUSTO) cohort. *Br. J. Nutr.* **2017**, *117*, 1042–1051. [CrossRef]
25. House, B.T.; Shearrer, G.E.; Miller, S.J.; Pasch, K.E.; Goran, M.I.; Davis, J.N. Increased eating frequency linked to decreased obesity and improved metabolic outcomes. *Int. J. Obes.* **2015**, *39*, 136–141. [CrossRef] [PubMed]
26. Eloranta, A.M.; Lindi, V.; Schwab, U.; Tompuri, T.; Kiiskinen, S.; Lakka, H.M.; Laitinen, T.; Lakka, T.A. Dietary factors associated with overweight and body adiposity in Finnish children aged 6–8 years: The PANIC Study. *Int. J. Obes.* **2012**, *36*, 950–955. [CrossRef]
27. Okubo, H.; Murakami, K.; Masayasu, S.; Sasaki, S. The Relationship of Eating Rate and Degree of Chewing to Body Weight Status among Preschool Children in Japan: A Nationwide Cross-Sectional Study. *Nutrients* **2018**, *11*, 64. [CrossRef] [PubMed]
28. Okubo, H.; Miyake, Y.; Sasaki, S.; Tanaka, K.; Hirota, Y. Rate of eating in early life is positively associated with current and later body mass index among young Japanese children: The Osaka Maternal and Child Health Study. *Nutr. Res.* **2017**, *37*, 20–28. [CrossRef] [PubMed]
29. Jääskeläinen, A.; Schwab, U.; Kolehmainen, M.; Pirkola, J.; Järvelin, M.R.; Laitinen, J. Associations of meal frequency and breakfast with obesity and metabolic syndrome traits in adolescents of Northern Finland Birth Cohort 1986. *Nutr. Metab. Cardiovasc. Dis.* **2013**, *23*, 1002–1009. [CrossRef]
30. Ritchie, L.D. Less frequent eating predicts greater BMI and waist circumference in female adolescents. *Am. J. Clin. Nutr.* **2012**, *95*, 290–296. [CrossRef]
31. Franko, D.L.; Striegel-Moore, R.H.; Thompson, D.; Affenito, S.G.; Schreiber, G.B.; Daniels, S.R.; Crawford, P.B. The relationship between meal frequency and body mass index in black and white adolescent girls: More is less. *Int. J. Obes.* **2008**, *32*, 23–29. [CrossRef]

32. Kelishadi, R.; Qorbani, M.; Motlagh, M.E.; Heshmat, R.; Ardalan, G.; Bahreynian, M. Association of eating frequency with anthropometric indices and blood pressure in children and adolescents: The CASPIAN-IV Study. *J. Pediatr. (Rio. J.).* **2016**, *92*, 156–167. [CrossRef]
33. Alberti, K.G.M.M.; Eckel, R.H.; Grundy, S.M.; Zimmet, P.Z.; Cleeman, J.I.; Donato, K.A.; Fruchart, J.C.; James, W.P.T.; Loria, C.M.; Smith, S.C. Harmonizing the metabolic syndrome: A joint interim statement of the international diabetes federation task force on epidemiology and prevention; National heart, lung, and blood institute; American heart association; World heart federation; International atherosclerosis society; And international association for the study of obesity. *Circulation* **2009**, *120*, 1640–1645.
34. Lee, S.Y.; Park, H.S.; Kim, D.J.; Han, J.H.; Kim, S.M.; Cho, G.J.; Kim, D.Y.; Kwon, H.S.; Kim, S.R.; Lee, C.B.; et al. Appropriate waist circumference cutoff points for central obesity in Korean adults. *Diabetes Res. Clin. Pract.* **2007**, *75*, 72–80. [CrossRef]
35. Li, C.; Ford, E.S.; Mokdad, A.H.; Cook, S. Recent trends in waist circumference and waist-height ratio among US children and adolescents. *Pediatrics* **2006**, *118*. [CrossRef] [PubMed]
36. Alberti, K.G.M.M.; Zimmet, P.; Shaw, J. The metabolic syndrome—a new worldwide definition. *Lancet* **2005**, *366*, 1059–1062. [CrossRef]
37. Grundy, S.M.; Cleeman, J.I.; Bairey Merz, C.N.; Brewer, H.B.; Clark, L.T.; Hunninghake, D.B.; Pasternak, R.C.; Smith, S.C.; Stone, N.J. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. *Circulation* **2004**, *110*, 227–239. [CrossRef]
38. Cleeman, J.I.; Grundy, S.M.; Becker, D.; Clark, L. Expert panel on detection evaluation and treatment of high blood cholesterol in adults Executive summary of the third report (NCEP) -adult treatment panel III. *J. Am. Med. Assoc.* **2001**, *285*, 2486–2497.
39. Chobanian, A.V.; Bakris, G.L.; Black, H.R.; Cushman, W.C.; Green, L.A.; Izzo, J.L.; Jones, D.W.; Materson, B.J.; Oparil, S.; Wright, J.T.; et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension* **2003**, *42*, 1206–1252. [CrossRef]
40. Zimmet, P.; Alberti, G.K.M.M.; Kaufman, F.; Tajima, N.; Silink, M.; Arslanian, S.; Wong, G.; Bennett, P.; Shaw, J.; Caprio, S. The metabolic syndrome in children and adolescents—An IDF consensus report. *Pediatr. Diabetes* **2007**, *8*, 299–306. [CrossRef] [PubMed]
41. Fallah, Z.; Qorbani, M.; Motlagh, M.E.; Heshmat, R.; Ardalan, G.; Kelishadi, R. Prevalence of prehypertension and hypertension in a nationally representative sample of iranian children and adolescents: The CASPIAN-IV study. *Int. J. Prev. Med.* **2014**, *5* (Suppl. S1), S57. [PubMed]
42. Guenther, P.M.; Kirkpatrick, S.I.; Reedy, J.; Krebs-Smith, S.M.; Buckman, D.W.; Dodd, K.W.; Casavale, K.O.; Carroll, R.J. The healthy eating Index-2010 is a valid and reliable measure of diet quality according to the 2010 dietary guidelines for Americans. *J. Nutr.* **2014**, *144*, 399–407. [CrossRef] [PubMed]
43. Guenther, P.M.; Casavale, K.O.; Reedy, J.; Kirkpatrick, S.I.; Hiza, H.A.B.; Kuczynski, K.J.; Kahle, L.L.; Krebs-Smith, S.M. Update of the Healthy Eating Index: HEI-2010. *J. Acad. Nutr. Diet.* **2013**, *113*, 569–580. [CrossRef] [PubMed]
44. Trichopoulou, A.; Orfanos, P.; Norat, T.; Bueno-de-Mesquita, B.; Ocké, M.C.; Peeters, P.H.M.; van der Schouw, Y.T.; Boeing, H.; Hoffmann, K.; Boffetta, P.; et al. Modified Mediterranean diet and survival: EPIC-elderly prospective cohort study. *Br. Med. J.* **2005**, *330*, 991–995. [CrossRef] [PubMed]
45. Struijk, E.A.; Beulens, J.W.; May, A.M.; Fransen, H.P.; Boer, J.M.A.; De Wit, G.A.; Onland-Moret, N.G.; Van Der Schouw, Y.T.; Hoekstra, J.; Bueno-De-Mesquita, H.B.; et al. Dietary patterns in relation to disease burden expressed in Disability-Adjusted Life Years. *Am. J. Clin. Nutr.* **2014**, *100*, 1158–1165. [CrossRef]
46. Madden, J.P.; Goodman, S.J.; Guthrie, H.A. Validity of the 24-hr. recall. Analysis of data obtained from elderly subjects. *J. Am. Diet. Assoc.* **1976**, *68*, 143–147.
47. Kant, A.K. Indexes of overall diet quality: A review. *J. Am. Diet. Assoc.* **1996**, *96*, 785–791. [CrossRef]
48. Huijbregts, P.; Feskens, E.; Räsänen, L.; Fidanza, F.; Nissinen, A.; Menotti, A.; Kromhout, D. Dietary pattern and 20 year mortality in elderly men in Finland, Italy, and the Netherlands: Longitudinal cohort study. *Br. Med. J.* **1997**, *315*, 13–17. [CrossRef]
49. Thorpe, M.G.; Milte, C.M.; Crawford, D.; McNaughton, S.A. A revised Australian dietary guideline index and its association with key sociodemographic factors, health behaviors and body mass index in peri-retirement aged adults. *Nutrients* **2016**, *8*, 160. [CrossRef] [PubMed]
50. Toft, U.; Kristoffersen, L.H.; Lau, C.; Borch-Johnsen, K.; Jørgensen, T. The Dietary Quality Score: Validation and association with cardiovascular risk factors: The Inter99 study. *Eur. J. Clin. Nutr.* **2007**, *61*, 270–278. [CrossRef]
51. Evans, E.W.; Jacques, P.F.; Dallal, G.E.; Sacheck, J.; Must, A. The role of eating frequency on total energy intake and diet quality in a low-income, racially diverse sample of schoolchildren. *Public Health Nutr.* **2015**, *18*, 474–481. [CrossRef]
52. Sonoda, C.; Fukuda, H.; Kitamura, M.; Hayashida, H.; Kawashita, Y.; Furugen, R.; Koyama, Z.; Saito, T. Associations among obesity, eating speed, and oral health. *Obes. Facts* **2018**, *11*, 165–175. [CrossRef]
53. Hamada, Y.; Miyaji, A.; Hayashi, Y.; Matsumoto, N.; Nishiwaki, M.; Hayashi, N. Objective and subjective eating speeds are related to body composition and shape in female college students. *J. Nutr. Sci. Vitaminol.* **2017**, *63*, 174–179. [CrossRef] [PubMed]
54. Wuren, M.A.; Endoh, K.; Kuriki, K. Eating rate as risk for body mass index and waist circumference obesity with appropriate confounding factors: A cross-sectional analysis of the Shizuoka-Sakuragaoka J-MICC Study. *Asia Pac. J. Clin. Nutr.* **2019**, *28*, 79–91. [CrossRef]
55. Nagahama, S.; Kurotani, K.; Pham, N.M.; Nanri, A.; Kuwahara, K.; Dan, M.; Nishiwaki, Y.; Mizoue, T. Self-reported eating rate and metabolic syndrome in Japanese people: Cross-sectional study. *BMJ Open* **2014**, *4*, e005241. [CrossRef]

56. Leong, S.L.; Gray, A.; Horwath, C.C. Speed of eating and 3-year BMI change: A nationwide prospective study of mid-age women. *Public Health Nutr.* **2016**, *19*, 463–469. [CrossRef] [PubMed]
57. Leech, R.M.; Livingstone, K.M.; Worsley, A.; Timperio, A.; McNaughton, S.A. Meal frequency but not snack frequency is associated with micronutrient intakes and overall diet quality in Australian men and women. *J. Nutr.* **2016**, *146*, 2027–2034. [CrossRef] [PubMed]
58. Holm, L.; Lund, T.B.; Niva, M. Eating practices and diet quality: A population study of four Nordic countries. *Eur. J. Clin. Nutr.* **2015**, *69*, 791–798. [CrossRef]
59. Alamri, E.S. Snacking patterns in relation to waist circumference and body mass index in female Saudi undergraduate students Medical Science. *Med. Sci.* **2020**, *24*, 1128–1135.
60. Barnes, T.L.; French, S.A.; Harnack, L.J.; Mitchell, N.R.; Wolfson, J. Snacking Behaviors, Diet Quality, and Body Mass Index in a Community Sample of Working Adults. *J. Acad. Nutr. Diet.* **2015**, *115*, 1117–1123. [CrossRef] [PubMed]
61. Drummond, S.E.; Crombie, N.E.; Cursiter, M.C.; Kirk, T.R. Evidence that eating frequency is inversely related to body weight status in male, but not female, non-obese adults reporting valid dietary intakes. *Int. J. Obes.* **1998**, *22*, 105–112. [CrossRef] [PubMed]
62. Aljuraiban, G.S.; Chan, Q.; Oude Griep, L.M.; Brown, I.J.; Daviglus, M.L.; Stamler, J.; Van Horn, L.; Elliott, P.; Frost, G.S. The Impact of Eating Frequency and Time of Intake on Nutrient Quality and Body Mass Index: The INTERMAP Study, a Population-Based Study. *J. Acad. Nutr. Diet.* **2015**, *115*, 528–536.e1. [CrossRef]
63. House, B.T.; Shearrer, G.E.; Boisseau, J.B.; Bray, M.S.; Davis, J.N. Decreased eating frequency linked to increased visceral adipose tissue, body fat, and BMI in Hispanic college freshmen. *BMC Nutr.* **2018**, *4*, 10. [CrossRef]
64. O'Connor, L.; Brage, S.; Griffin, S.J.; Wareham, N.J.; Forouhi, N.G. The cross-sectional association between snacking behaviour and measures of adiposity: The Fenland Study, UK. *Br. J. Nutr.* **2015**, *114*, 1286–1293. [CrossRef]
65. Zhu, Y.; Hollis, J.H. Associations between eating frequency and energy intake, energy density, diet quality and body weight status in adults from the USA. *Br. J. Nutr.* **2016**, *115*, 2138–2144. [CrossRef] [PubMed]
66. Zizza, C.A.; Xu, B. Snacking is associated with overall diet quality among adults. *J. Acad. Nutr. Diet.* **2012**, *112*, 291–296. [CrossRef]
67. Kahleova, H.; Lloren, J.I.; Mashchak, A.; Hill, M.; Fraser, G.E. Meal frequency and timing are associatedwith changes in body mass index in Adventist Health Study 2. *J. Nutr.* **2017**, *147*, 1722–1728. [CrossRef]
68. Kanaley, J.A.; Heden, T.D.; Liu, Y.; Fairchild, T.J. Alteration of postprandial glucose and insulin concentrations with meal frequency and composition. *Br. J. Nutr.* **2014**, *112*, 1484–1493. [CrossRef] [PubMed]
69. Alencar, M.K.; Beam, J.R.; McCormick, J.J.; White, A.C.; Salgado, R.M.; Kravitz, L.R.; Mermier, C.M.; Gibson, A.L.; Conn, C.A.; Kolkmeyer, D.; et al. Increased meal frequency attenuates fat-free mass losses and some markers of health status with a portion-controlled weight loss diet. *Nutr. Res.* **2015**, *35*, 375–383. [CrossRef]
70. Carlson, O.; Martin, B.; Stote, K.S.; Golden, E.; Maudsley, S.; Najjar, S.S.; Ferrucci, L.; Ingram, D.K.; Longo, D.L.; Rumpler, W.V.; et al. Impact of reduced meal frequency without caloric restriction on glucose regulation in healthy, normal-weight middle-aged men and women. *Metabolism* **2007**, *56*, 1729–1734. [CrossRef] [PubMed]
71. Megson, M.; Wing, R.; Leahey, T.M. Effects of breakfast eating and eating frequency on body mass index and weight loss outcomes in adults enrolled in an obesity treatment program. *J. Behav. Med.* **2017**, *40*, 595–601. [CrossRef]
72. Stote, K.S.; Baer, D.J.; Spears, K.; Paul, D.R.; Harris, G.K.; Rumpler, W.V.; Strycula, P.; Najjar, S.S.; Ferrucci, L.; Ingram, D.K.; et al. A controlled trial of reduced meal frequency without caloric restriction in healthy, normal-weight, middle-aged adults. *Am. J. Clin. Nutr.* **2007**, *85*, 981–988. [CrossRef]
73. Yildiran, H.; Mercanligil, S.M. Does increasing meal frequency improve weight loss and some biochemical parameters in overweight/obese females? *Nutr. Hosp.* **2019**, *36*, 66–72. [CrossRef]
74. Larsen, S.C.; Heitmann, B.L. More Frequent Intake of Regular Meals and Less Frequent Snacking Are Weakly Associated with Lower Long-Term Gains in Body Mass Index and Fat Mass in Middle-Aged Men and Women. *J. Nutr.* **2019**, *149*, 824–830. [CrossRef] [PubMed]
75. Fábry, P.; Hejl, Z.; Fodor, J.; Braun, T.; Zvolánková, K. The Frequency of Meals Its Relation to Overweight, Hypercholesterolæmia, and Decreased Glucose-Tolerance. *Lancet* **1964**, *284*, 614–615. [CrossRef]
76. Chang, S.H.; Chang, Y.Y.; Wu, L.Y. Gender differences in lifestyle and risk factors of metabolic syndrome: Do women have better health habits than men? *J. Clin. Nurs.* **2019**, *28*, 2225–2234. [CrossRef]
77. Johnson, C.M.; Henderson, M.S.; Tripicchio, G.; Rozin, P.; Heo, M.; Pietrobelli, A.; Berkowitz, R.I.; Keller, K.L.; Faith, M.S. Observed parent–child feeding dynamics in relation to child body mass index and adiposity. *Pediatr. Obes.* **2018**, *13*, 222–231. [CrossRef] [PubMed]
78. Birch, L.L.; Fisher, J.O. Development of eating behaviors among children and adolescents. *Pediatrics* **1998**, *101*, 539–549. [PubMed]
79. De Onis, M. WHO Child Growth Standards based on length/height, weight and age. *Acta Paediatr. Int. J. Paediatr.* **2006**, *95*, 76–85. [CrossRef]
80. Hermsen, S.; Mars, M.; Higgs, S.; Frost, J.H.; Hermans, R.C.J. Effects of eating with an augmented fork with vibrotactile feedback on eating rate and body weight: A randomized controlled trial. *Int. J. Behav. Nutr. Phys. Act.* **2019**, *16*. [CrossRef]
81. Salazar Vázquez, B.Y.; Salazar Vázquez, M.A.; López Gutiérrez, G.; Acosta Rosales, K.; Cabrales, P.; Vadillo-Ortega, F.; Intaglietta, M.; Pérez Tamayo, R.; Schmid-Schönbein, G.W. Control of overweight and obesity in childhood through education in meal time habits. The 'good manners for a healthy future' programme. *Pediatr. Obes.* **2016**, *11*, 484–490. [CrossRef] [PubMed]

82. Galhardo, J.; Hunt, L.P.; Lightman, S.L.; Sabin, M.A.; Bergh, C.; Sodersten, P.; Shield, J.H. Normalizing eating behavior reduces body weight and improves gastrointestinal hormonal secretion in obese adolescents. *J. Clin. Endocrinol. Metab.* **2012**, *97*, 193–201. [CrossRef]
83. Li, J.; Zhang, N.; Hu, L.; Li, Z.; Li, R.; Li, C.; Wang, S. Improvement in chewing activity reduces energy intake in one meal and modulates plasma gut hormone concentrations in obese and lean young Chinese men. *Am. J. Clin. Nutr.* **2011**, *94*, 709–716. [CrossRef] [PubMed]
84. Krop, E.M.; Hetherington, M.M.; Nekitsing, C.; Miquel, S.; Postelnicu, L.; Sarkar, A. Influence of oral processing on appetite and food intake—A systematic review and meta-analysis. *Appetite* **2018**, *125*, 253–269. [CrossRef] [PubMed]
85. Argyrakopoulou, G.; Simati, S.; Dimitriadis, G.; Kokkinos, A. How important is eating rate in the physiological response to food intake, control of body weight, and glycemia? *Nutrients* **2020**, *12*, 1734. [CrossRef] [PubMed]
86. Szewczyk-Golec, K.; Woźniak, A.; Reiter, R.J. Inter-relationships of the chronobiotic, melatonin, with leptin and adiponectin: Implications for obesity. *J. Pineal Res.* **2015**, *59*, 277–291. [CrossRef]
87. Shah, M.; Crisp, K.; Adams-Huet, B.; Dart, L.; Bouza, B.; Franklin, B.; Phillips, M. The effect of eating speed at breakfast on appetite hormone responses and daily food consumption. *J. Investig. Med.* **2015**, *63*, 22–28. [CrossRef]
88. House, B.T.; Cook, L.T.; Gyllenhammer, L.E.; Schraw, J.M.; Goran, M.I.; Spruijt-Metz, D.; Weigensberg, M.J.; Davis, J.N. Meal skipping linked to increased visceral adipose tissue and triglycerides in overweight minority youth. *Obesity* **2014**, *22*, 77–84. [CrossRef]
89. Campión, J.; Milagro, F.I.; Martínez, J.A. Individuality and epigenetics in obesity. *Obes. Rev.* **2009**, *10*, 383–392. [CrossRef]
90. Yaghootkar, H.; Whitcher, B.; Bell, J.D.; Thomas, E.L. Ethnic differences in adiposity and diabetes risk—insights from genetic studies. *J. Intern. Med.* **2020**, *288*, 271–283. [CrossRef]
91. Simmonds, M.; Llewellyn, A.; Owen, C.G.; Woolacott, N. Predicting adult obesity from childhood obesity: A systematic review and meta-analysis. *Obes. Rev.* **2016**, *17*, 95–107. [CrossRef] [PubMed]

4.6. Physical activity

In children, this lifestyle behavior has been widely assessed in relation to obesity since past years[119–122]. Nevertheless, associations with cardiometabolic risk factors have started to be especially inquired until recently[116,123,124]. Recommendations for physical activity in childhood varies according to age however, is has been recognized that younger children required higher levels of physical activity in order to decrease the risk of disease [125,126]. Therefore, compliance with recommendations of moderate-to-vigorous physical activity has been reported in association with lower adiposity[121]. Physical activity has also been associated with improvements of some comorbidities of obesity such as systemic inflammation[122] and insulin resistance[127], for which a reduction in BMI was observed in parallel[127]. Besides, favorable effects on several cardiometabolic risk factors were reported by different types of exercise[116,128,129], including aerobic physical activity[123], resistance physical activity[128] and moderate-to-high intensity interval training[130]. However, differences on these effects have been observed according to duration, training time, the setting and the work/rest ratio[130]. Furthermore, physical activity may act as a mediator in the association between some lifestyle behaviors, such as diet, and levels of cardiometabolic risk factors[131]. In addition, concomitant associations between diet, sleep duration and sedentary behaviors have been reported in relation to the

cardiometabolic profile according to physical activity levels[132], which might contribute to some of the health outcomes associated with physical activity.

4.7. Sedentary behaviors

At present, digital media devices (e.g., television, computer, newer mobile devices, etc.) are ubiquitous and an important part of our daily lives, so their impact on childhood health have become a Public Health concern[133]. The current screentime recommendation is limited to 2 hours per day[134] and it has been suggested that non-compliance could contribute to certain health disorders in children. Regarding its consequences on cardiometabolic profile, higher screentime has been associated with adiposity[133,135] and other cardiometabolic disorders[117,136] in children. Some differences in these outcomes have been observed according to sex[137,138], weight status[137], mealtime digital media use[136,139,140] or type of digital media device used[137]. At the same time, digital media use during mealtimes has been observed in association with variations on cardiometabolic outcomes according to age and the specific mealtime[136]. With respect to obesity-related health disorders, a higher risk to develop some of them such as insulin resistance have been observed in association with the increased number of digital media devices in children's bedroom[127]. At the same time, digital media use[133,141] and eating whilst watching TV[139] has been reported in association with decreased intake of plant-based foods[139] but

increased consumption of food in overall[141], especially unhealthy food[133,142] such as high-fat and/or high-sugar foods[139], including sugar-sweetened beverages, pizza, fried foods, sweets, and snacks[139]. Some authors have suggested that these associations could be potentially related to digital media content[143] and/or food advertising promotions[141,144]. Additionally, there is evidence of a potential indirect association between screentime and physical activity that may contribute to health outcomes [117].

4.8. Sleep duration

Sleep is a vital part of human's life and is especially essential for children´s health[145,146]. Therefore, certain sleep characteristics such as duration, timing and variability have been continually assessed, objectively and subjectively, in association with health[147]. According to the National Sleep Foundation[148], sleep duration requirements are consistent with the life stage and tend to decrease over the years. Also, there is a consensus on sleep quality recommendations that can be summarized in shorter sleep latencies, fewer awakenings, reduce wake after sleep onset, regardless of age[149]. Compliance with recommendations has been reported in association with extrinsic and intrinsic factors such as bedtime[150,151], child age, temperament, digital media use, parenting style and socioeconomic status[151], among others. Sleep duration has been widely assessed so that longer sleep duration has been associated with lower adiposity[145,152] and shorter sleep duration with higher

adiposity [153–156]. Stronger associations between short sleep duration and increased risk of obesity have been reported in children aged 3-13 year-old[157]. With regard to other cardiometabolic risk factors, shorter sleep duration in early childhood was associated with higher HDL-c concentrations [158] , higher blood pressure[159,160] (which could be independent of BMI[159]) as well as insulin resistance[153,160] and consequently decreased insulin sensitivity[153]. However, at present, evidence on the associations between sleep duration and systematic inflammation or cardiometabolic risk is inconsistent[160]. On the other hand, the sleeping pattern has also been observed in association with other lifestyle behaviors so that short sleepers may be more sedentary[153], have greater dietary energy intake[161] and lower diet quality[153,161] , while long sleepers could spend more time physically active[153,162].

4.9. Lifestyle behaviors scores

Traditionally, individual lifestyle behaviors have been assessed to measure the impact of lifestyle on cardiometabolic health. However, in recent years, some evidence in children has emerged on the concomitant assessment of certain lifestyle behaviors, including physical activity, diet, sleep duration and sedentary behaviors[111,163–165].

Bawaked et al.[111] created the Child healthy lifestyle score (CHLS) comprised of either favorable (extracurricular physical activity,

sleep duration and plant-based foods consumption) or unfavorable (screentime and consumption of ultra-processed food) lifestyle behaviors which were divided into tertiles and given a score according to their beneficial or detrimental potential effects so higher scores corresponded to greater adherence to healthy lifestyles[111]. On the other hand, in a population of European children from 8 countries, other composite adherence score was estimated[164] according to the compliance with key targets of some lifestyle behaviors such as water-sweetened beverages, vegetables/fruits consumption, screentime, physical activity, sleep duration and stress-related behaviors, in which adhering to four or more of these key targets was regarded as a healthier lifestyle[164]. The Preschoolers Diet-Lifestyle Index[163] was created to assess its association with obesity in preschool children by scoring dietary intake (fruits, vegetables, red meat, meat products, white meat, fish, seafood, legumes, dairy products, sweets, grains and unsaturated fats), moderate-to-vigorous physical activity and screentime, for which the total score was summed and divided into tertiles. Greater adherence to healthy lifestyle behaviors in all these scores[111,163,164] was observed in association with lower adiposity, at baseline[111,163,164] and/or in long-term[111]. The combined detrimental impact of lifestyle behaviors has also been assessed in children[165], in short and long-term. In this scoring system [165], the lifestyle factors assessed were certain children's factors (weight status, physical

inactivity, outdoors time, breakfast consumption, digital media use) but also parental factors (weight status, physical inactivity and smoking habit). According to the sum of all negative lifestyle factors, a higher number was associated to higher risk for cardiometabolic disorders[165]. Similarly, to the Preschoolers Diet-Lifestyle Index[163], the concomitant associations between some lifestyle behaviors and certain obesity comorbidities, such as insulin resistance, were explored by another lifestyle-diet quality index[166], for which the likelihood of showing insulin resistance was lower in those schoolchildren with higher adherence to healthy lifestyle profiles[166].

5. Obesity and cardiometabolic risk in childhood: prevention

There is no single prevention measure that could halt the growing obesity epidemic[167]. To address obesity requires to considerate its etiology context[74], since it is multi-causal, and a whole-of-government approach[168,169]. It has been suggested that prevention of childhood obesity and its comorbidities should comprise care for women before conception and in pregnancy as well as children's health, which must involve a wide range of lifestyle interventions[170–173] such as diet, physical activity and many other factors[16,174]. Greater benefits in the prevention of obesity and cardiometabolic disorders in childhood has been observed by the supervision of a multidisciplinary team that includes dietitians and physicians [175], as well as the participation of parents[176,177] and family[178,179].

II. RATIONALE AND JUSTIFICATION

Rationale and Justification

Across the world, obesity is a widely extended Public Health major concern. It represents one of the most prevalent non-communicable diseases which is linked to a considerable number of comorbidities, including cardiometabolic disorders, and could lead to disability and death. The early onset of obesity has been associated with higher risk of related complications in the upcoming life stages. In Europe, near to 1 in 3 schoolchildren have overweight or obesity and its tendency is to increase. This is mainly due to its etiology which comprises an intricated interrelated network of several factors that coexist with the loss of normal hormone function and other internal body processes creating an obesogenic vicious cycle that results in a great challenge to address. At the same time, further potential obesity risk factors have been suggested however, the evidence in children is very limited.

The role of diet quality in obesity has been recognized for years. Evidence highlights the importance of a healthy diet, mainly abundant in plant-based and minimally processed foods, in the prevention and treatment of obesity at all ages. However, the impact of certain specific dietary patterns such as MedDiet, an extensively consumed dietary pattern in Mediterranean countries, on obesity and other cardiometabolic disorders has been mainly assessed in adults and their benefits are well-recognized in this life stage however, these have yet to be acknowledged in children.

Rationale and Justification

On the other hand, in recent years there has emerged a growing interest in additional diet-associated eating behaviors, such as eating speed, as potential risk factors for obesity and cardiometabolic disorders. Nevertheless, evidence is still scarce, especially in children, and is controversial mainly due to the lack of a universal definition and a frame of reference for adequate values.

In the last years, lifestyle has been determined as a relevant risk factor for adiposity and its associated cardiometabolic risk. Lifestyle behaviors have usually been assessed individually albeit in recent years, they have begun to be assessed concomitantly, mainly those traditional lifestyle behaviors whose detrimental effects on health are well-recognized (e.g., physical activity, consumption of plant-based foods, sleep duration, sedentary behaviors, among others). To the best of our knowledge, no previous published studies have assessed the impact on cardiometabolic health by the co-occurrence of traditional lifestyle behaviors and a few others such as eating speed and adherence to the MedDiet, in children.

In this sense, given that the foundation of future health is stablished in childhood and especially in early life, a greater contribution to scientific evidence in all these fields is key to help addressing the global obesity pandemic and alleviate its effects on present and future generations.

III. HYPOTHESIS AND OBJETIVES

Hypothesis

In a population of healthy Spanish preschool children, fast eating speed is associated with increased adiposity and higher levels of cardiometabolic risk factors, as well as lower diet quality. In addition, greater diet quality, slow eating and compliance with recommendations for breastfeeding, physical activity, sleep duration and sedentary behaviors are associated with a lower prevalence risk of overweight or obesity and decreased levels of certain cardiometabolic risk factors which could lead to decreased risk of disease in childhood and adulthood.

Objectives

a. General objective

To assess the association between diet quality, eating speed, eating frequency and other lifestyle behaviors and cardiometabolic risk factors (adiposity, blood pressure, fasting plasma glucose and lipids profile) in a population of healthy Spanish children aged 3-6 years.

b. Specific objectives

i. To summarized evidence on the associations of eating speed and eating frequency with adiposity, cardiometabolic risk and diet quality in children.

ii. To assess the associations between eating speed, diet quality and cardiometabolic risk factors in CORALS study cohort.

Hypothesis and Objectives

iii. To analyze the associations between a composite score comprised of 6 healthy lifestyle behaviors as well as its individual components (breastfeeding, sleep duration, physical activity, screentime, adherence to MedDiet and eating speed) with cardiometabolic disorders in CORALS study cohort.

IV. METHODOLOGY

The present doctorate thesis has been performed in the context of one review and two original articles. This section will describe the methodology of the original articles, which have been conducted in the framework of CORALS study.

1. Childhood Obesity Risk Assessment Longitudinal Study (CORALS)

1.1. Study Design and Objectives

CORALS is a prospective ongoing multicenter study conducted in preschool children from 7 Spanish cities (Barcelona, Córdoba, Navarra, Reus, Santiago de Compostela, Valencia and Zaragoza) with a 10-year follow-up. The main aim is to identify the risk factors for childhood obesity. Secondary objectives include the assessment of the associations between obesity as well as its comorbidities and: a) perinatal risk, socioeconomic, cultural and environmental factors; b) lifestyle patterns (diet, drinks consumption, physical activity, sedentary behaviors, sleeping pattern) and c) genetic polymorphisms, metabolomic and metagenomic profiles (assessed by omics techniques). The Ethics Committee of each recruitment center approved the study protocol (Reference numbers: 051/2019; 4155/2019; 2019/18; 9/19; 09/2019; 19/27; 2019/131), which was conducted following the standards of the Declaration of Helsinki. A written informed consent was signed by parents or caregivers of all participants recruited.

1.2. Study population

Eligible participants were children aged 3-6 years whose tutors had agreed to participate. From March 2019 to July 2023, a total of 1508 participants met inclusion criteria and were included in CORALS study. The inclusion criteria included to sign the consent form; attend the inclusion visit and fulfill the questionnaire for leisure-time physical activity and the 3-days food record. The exclusion criteria were to belong to a family with difficulty participating; comprehension or language difficulties or to have unstable residence.

1.3. Data assessments

1.3.1. Early life and maternal factors

Early life factors and family factors were assessed by questionnaires including beginning/ending of breastfeeding (months), weight at birth (kg) and certain maternal characteristics such as weight gain during pregnancy (kg), weight (kg), height (m), educational level (primary or lower, secondary, academic-graduate or no reported) and socio-professional category (homemaker, student, retired, unemployed or employee). The duration of breastfeeding was estimated and then classified according to WHO recommendations (<6 months or ≥6 months)[180]. Maternal weight status was estimated according to BMI and re-categorized in underweight/normal weight (<25 kg/m^2) and overweight/obesity (≥25 kg/m^2) [181].

1.3.2. Diet and nutrients

Dietary assessment was performed by semi-quantitative 125-item food frequency questionnaires which aim was to evaluate the average daily nutrient intake over the last year. For each item, all possible answers were transformed into grams or milliliters per day using the standard portion size of each item. Spanish dietary databases[182–184] were used to estimate total energy (kcal/day) and nutrient intake. Dietary assessment included protein intake per body weight (grams of daily protein intake/body weight) as well as percentage for daily intake of carbohydrates, proteins, and fatty acids. The frequency of consumption (g/day) was assessed for dairy products, vegetables, fruits, nuts, protein foods, cereals and legumes, pastries, candies, and beverages.

1.3.3. Diet quality

An 18-item MedDiet ad-hoc questionnaire adapted to children was provided to parents or caregivers. This questionnaire is derived from a MedDiet score validated in the study population of the PREDIMED-Plus clinical trial[185].

The 18-item MedDiet questionnaire assessed the consumption of:

a) Foods included in the MedDiet such as olive oil, fruits, vegetables, cereals, nuts, seeds, cheese, yogurt, legumes, fish, seafood, poultry, and rabbit.

b) Foods not included in the MedDiet such as butter, cream, carbonated/sugary beverages, junk food, dairy desserts and pre-cooked/ready to eat food.
c) Certain typical foods consumed by children (e.g., gummy worms, sweets, petit suisse, etc.) that are not included in the MedDiet, for which, in some cases, the amount of servings was adapted.

Fulfilling each item sum 1 point to total score. Therefore, the score range was from 0 to 18 points in which 0 points meant null adherence and 18 points meant the highest adherence to MedDiet.

1.3.4. Eating Speed

Parents or caregivers completed the question 'How long does it usually take for your child to eat in each meal?' for breakfast, lunch, and dinner. Eating speed was estimated by summing the total minutes reported in the 3 main meals. Then, tertiles were created in order to classify eating speed in categories (slow, moderate and fast).

1.3.5. Physical activity

The physical activity was assessed by a questionnaire based on the Outdoor Playtime Checklist and the Outdoor Playtime Recall Questions[186]. The total length per week (hours) was estimated by summing the time of participation reported in physical education classes and extracurricular sports and physical activities.

The Active Lifestyle score was assessed by a 13-item ad hoc questionnaire based on leisure physical activities and sedentary behaviors so that a higher score represents a more active lifestyle.

1.3.6. Sleep

The duration of nighttime sleep and naps on weekdays and weekend days/holidays was assessed separately by the questions: 'how long does your child sleep at night?' and 'how long does your child nap?'. Daily sleep duration (hours/day) was estimated by summing the total duration of nighttime sleep and naps and then was divided by 7 (which corresponds to the total number of days of a week).

1.3.7. Screentime

Digital media use (television, computer, cell phone, game console) was assessed separately for weekdays and weekends days using the following questions: 'how long does your child watch television?' and 'how long does your child play in the computer/cell phone/game console?'.

1.3.8. Adiposity

Anthropometric measures were assessed by registered staff, according to standard procedures and with light clothing but no shoes. A precision scale (Tanita MC780SMA; Tanita Europe, B.V.) was used to estimated body weight (kg) and body fat mass (kg; by bioelectrical impedance). The height (cm) was measured by a portable stadiometer (seca 213, Escala 20-205 cm; SECA). A

measuring tape (seca 201) was used to measure waist circumference, midway between the lowest rib and the iliac crest. Weight status was estimated [187] according to BMI (kg/m^2) and re-categorized in underweight/normal weight and overweight/obesity. Fat mass index (FMI) was calculated as body fat mass (kg)/height^2, according to VanItilleet al. [188]

1.3.9. Blood pressure

The blood pressure was measured in triplicate at 5-minutes intervals with an automatic oscillometer (Omron M3 Intellisense HEM-75051-EV; IOMRON Healthcare Europe B.V.) and a child-adapted cuff in the nondominant arm. Systolic and diastolic blood pressure were reported, separately, as the mean of all determinations in each case.

1.3.10. Lipids and fasting plasma glucose

The collection of blood samples was performed in primary care centers by Pediatrics expert nurses in fasting conditions (8 hours). Serum and plasma samples were used to determine fasting plasma glucose, total cholesterol, high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), and triglycerides. Non-high-density lipoprotein cholesterol (non-HDL-c) was estimated according to the equation suggested by Frost et al[189].

1.4. Statistical Analyses

The detailed statistical analyses have been reported in each original article and therefore, a summary will be described in this

subsection. In the two original articles, cross-sectional analyses were conducted using the CORALS database updated to December 2021. Exposure variables were categorized into tertiles so that differences across tertiles in baseline general characteristics of the populations studied were reported as mean ± standard deviation (SD) or median [interquartile range (IQR)] for continuous variables and as percentage (numbers) for categorical variables according to ANOVA, Kruskal Wallis or chi-square tests, respectively. Logistic [odds ratio (OR); 95% confidence interval] and multiple linear [β; 95% confidence interval (CI)] regression models were fitted to assess the associations between exposure and outcomes variables. Statistical significance was set at a two-tailed p-value < 0.05 with a confidence interval of 95% and all the analyses were conducted using Stata 14 software program (StataCorp).

2. Second article

"Associations between eating speed, diet quality, adiposity, and cardiometabolic risk factors"

2.1. Selection of participants

From a total of 1371 preschool children recruited since March 2019 and until June 2021, 956 participants were included in the final analyses. Participants attended the first visit, met eligibility and inclusion criteria, did not have missing data or implausible reported energy intake and had available data in the duration of main meals

(breakfast, lunch and dinner). Children with current diagnosis of chronic diseases such as diabetes mellitus type 2, hypertension, or familiar hypercholesterolemia were excluded.

2.2. Specific data assessments

For the purposes of the second article, eating speed was re-categorized so that the higher tertile corresponded to fast-eating speed category. Also, additional analyses were conducted using the Child Eating Behavior Questionnaire (CEBQ) scale "slowness in eating" which was validated recently[190]. The questions included in this scale are: 'my child finishes his/her meal quickly'; 'my child eats slowly'; 'my child takes more than 30 minutes to finish a meal' and 'my child eats more and more slowly during the course of a meal'. The CEBQ scale "slowness in eating" was divided into tertiles and then re-categorized so that the 3rd tertile represented the fast-eating speed category.

2.2.1. Statistical Analyses

For the purposes of the second article, the following additional statistical analyses were conducted:

a) The reference category of the exposure variable corresponded to the slow eating.

3. Third article

"Associations between eating speed, diet quality, adiposity and cardiometabolic risk factors"

Methodology

3.1. Selection of participants

For the purposes of this article 938 participants were included in the final analyses from the total of 1371 participants recruited until June 2021. Participants attended the first visit in CORALS, met eligibility/inclusion criteria, had available and plausible values of reported energy intake, available data in meals duration, physical activity, breastfeeding, sleeping duration, the 18-item questionnaire of adherence to the MedDiet and sedentary behaviors and no current diagnosis for chronic diseases.

3.2. Specific data assessments

For the purposes of the third article, the following additional data assessments were performed:

a) Weight at birth was categorized in low (<2.5 kg), normal (2.5-3.9 kg), and high weight at birth (>4 kg), according to the international cutoffs[191].
b) Participants were classified according to whether they were exclusively breastfed during the first 6 months of life [180].
c) The highest tertile of eating speed represented slow eating speed.
d) The total time per week of sports and physical activities was categorized according to international recommendations[192]: ≥120 min/week (compliance) and <120 min/week (non-compliance).

e) Sleep duration was categorized according to the National Sleep Foundation's recommendations[148] in inadequate or could be adequate (3-5 year-old: <10 or >13 h/day; 6 year-old: <9 or >11 h/day) and adequate (3-5 year-old: 10-13h/day; 6 year-old: 9-11h/day).

f) Daily screentime was estimated by summing the time reported in each question and derived in a quantitative variable, for which categories were created as follows: <2 h/day (compliance with recommendation) and ≥2 h/day (non-compliance with recommendation)[134].

3.2.1. Composite score comprised of 6 healthy lifestyle behaviors

This composite score was created according to compliance with recommendations for breastfeeding, sleep duration, sports and physical activities, screentime, adherence to the MedDiet and eating speed. For breastfeeding, sleep duration, sports and physical activities and screentime, non-compliance meant 0 points and compliance with recommendations was scored with 1 point. Adherence to the MedDiet and eating speed were classified into tertiles in which a different scoring system was used (T1=0 points; T2= 0.5 points; T3= 1 point) and 3rd tertiles corresponded to slow eating speed and highest adherence to MedDiet, respectively. Total score (0-6 points) was estimated by summing all the points. Participants were classified into adherence categories according to

tertiles in which the 3rd tertile corresponded to the highest adherence to the 6 healthy lifestyle behaviors.

3.2.2. Statistical Analyses

For the purposes of the third article, Bonferroni's tests for multiple comparison were used in those one-factor ANOVA analyses that were statistically significant. Further logistic or multiple linear regression models were fitted in order to assess the individual associations between lifestyle behaviors and cardiometabolic risk factors. For all models, reference categories corresponded to the 1st tertile.

V. RESULTS

Second article

"Associations Between Eating Speed, Diet Quality, Adiposity, and Cardiometabolic Risk Factors"

Tany E. Garcidueñas-Fimbres, Indira Paz-Graniel, Carlos Gómez-Martínez, Jose Manuel Jurado-Castro, Rosaura Leis, Joaquin Escribano, Luis A. Moreno, Santiago Navas-Carretero, Olga Portoles, Karla A. Pérez-Vega, Mercedes Gil-Campos, Alicia López-Rubio, Cristina Rey-Reñones, Pilar De Miguel-Etayo, J. Alfredo Martínez, Katherine Flores-Rojas, Rocío Vázquez-Cobela, Verónica Luque, Maria Luisa Miguel-Berges, Belén Pastor-Villaescusa, Francisco Jesus Llorente-Cantarero, Jordi Salas-Salvadó and Nancy Babio on behalf of the CORALS study investigators.

- Publication phase: published.
- Doctoral student's position: first author.
- Journal: Journal of Pediatrics.
- Category: Pediatrics.
- Range position: 9/130 – D1.
- Impact factor: 6.314.
- Doi: 10.1016/j.jpeds.2022.08.024

Summary of results

Fast-eating category was associated with higher prevalence risk of overweight or obesity (OR, 2.9; 95% CI, 1.8-4.4; P< 0.01); larger waist

circumference (β, 2.6 cm; 95% CI, 1.5-3.8; P< 0.01), and greater FMI (β, 0.3 kg/m^2; 95% CI,0.1-0.5; P< 0.01), systolic blood pressure (β, 2.8 mmHg; 95% CI, 0.6-4.9; P< 0.05), and fasting plasma glucose levels (β, 2.7 mg/dL, 95% CI,1.2-4.2; P< 0.01) but lower adherence to the Mediterranean diet (β, 0.5 points; 95% CI, 0.9 to 0.1; P< 0.05).

Conclusion

Eating fast is associated with higher levels of adiposity and certain cardiometabolic risk factors, as well as lower adherence to the Mediterranean diet in Spanish children. However, further longitudinal studies and clinical trials are needed to confirm these associations.

Associations Between Eating Speed, Diet Quality, Adiposity, and Cardiometabolic Risk Factors

Tany E. Garcidueñas-Fimbres, MD, MSc[1,2,3], Indira Paz-Graniel, PhD[1,2,3], Carlos Gómez-Martínez, MSc[1,2,3], Jose Manuel Jurado-Castro, PhD[1,4], Rosaura Leis, MD, PhD[1,5,6,7], Joaquin Escribano, MD, PhD[3,8], Luis A. Moreno, MD, PhD[1,9,10], Santiago Navas-Carretero, PhD[1,11,12], Olga Portoles, PhD[1,13], Karla A. Pérez-Vega, MSc[1,14], Mercedes Gil-Campos, MD, PhD[1,4], Alicia López-Rubio, MSc[5,6,7], Cristina Rey-Reñones, PhD[15,16,17], Pilar De Miguel-Etayo, PhD[1,9,10], J. Alfredo Martínez, MD, PhD[11], Katherine Flores-Rojas, PhD[4], Rocío Vázquez-Cobela, PhD[1,5,6,7], Verónica Luque, PhD[3,8], Maria Luisa Miguel-Berges, PhD[1,9,10], Belén Pastor-Villaescusa, PhD[4], Francisco Jesus Llorente-Cantarero, PhD[1,18], Jordi Salas-Salvadó, MD, PhD[1,2,3,*], and Nancy Babio, PhD[1,2,3,*], on behalf of the Childhood Obesity Risk Assessment Longitudinal Study (CORALS) study investigators

Objective To assess the associations between eating speed, adiposity, cardiometabolic risk factors, and diet quality in a cohort of Spanish preschool-children.

Study design A cross-sectional study in 1371 preschool age children (49% girls; mean age, 4.8 ± 1.0 years) from the Childhood Obesity Risk Assessment Longitudinal Study (CORALS) cohort was conducted. After exclusions, 956 participants were included in the analyses. The eating speed was estimated by summing the total minutes used in each of the 3 main meals and then categorized into slow, moderate, or fast. Multiple linear and logistic regression models were fitted to assess the β-coefficient, or OR and 95% CI, between eating speed and body mass index, waist circumference, fat mass index (FMI), blood pressure, fasting plasma glucose, and lipid profile.

Results Compared with participants in the slow-eating category, those in the fast-eating category had a higher prevalence risk of overweight/obesity (OR, 2.9; 95% CI, 1.8-4.4; $P < .01$); larger waist circumference (β, 2.6 cm; 95% CI, 1.5-3.8 cm); and greater FMI (β, 0.3 kg/m^2; 95% CI, 0.1-0.5 kg/m^2), systolic blood pressure (β, 2.8 mmHg; 95% CI, 0.6-4.9 mmHg), and fasting plasma glucose levels (β, 2.7 mg/dL, 95% CI, 1.2-4.2 mg/dL) but lower adherence to the Mediterranean diet (β, −0.5 points; 95% CI, −0.9 to −0.1 points).

Conclusions Eating fast is associated with higher adiposity, certain cardiometabolic risk factors, and lower adherence to a Mediterranean diet. Further long-term and interventional studies are warranted to confirm these associations. *(J Pediatr 2023;252:31-9)*.

The vast majority of strategies to prevent or treat overweight and obesity in children are based on increasing physical activity, decreasing sedentary behaviors, and promoting adherence to a healthy diet.[1] However, some

BMI	Body mass index
CEBQ	Child Eating Behaviour Questionnaire
CESNID	Centre d'Enseyament Superior de Nutrició i Dietètica
CORALS	Childhood Obesity Risk Assessment Longitudinal Study
FMI	Fat mass index
HDL-c	High-density lipoprotein cholesterol
LDL-c	Low-density lipoprotein cholesterol

From the [1]Consorcio CIBER, Fisiopatología de la Obesidad y Nutrición, Instituto de Salud Carlos III, Madrid, Spain; [2]Universitat Rovira i Virgili, Departament de Bioquímica i Biotecnologia, Unitat de Nutrició Humana, Reus, Spain; [3]Institut d'Investigació Sanitària Pere Virgili, Reus, Spain; [4]Reina Sofia University Hospital, Maimónides Biomedical Research Institute of Cordoba, University of Córdoba, Córdoba, Spain; [5]Unit of Pediatric Gastroenterology, Hepatology and Nutrition, Pediatric Service, Complejo Hospitalario Universitario de Santiago de Compostela, Santiago de Compostela, Spain; [6]Pediatric Nutrition Research Group, Health Research Institute of Santiago de Compostela, Santiago de Compostela, Spain; [7]Unit of Investigation in Human Nutrition, Growth and Development of Galicia, University of Santiago de Compostela, Santiago de Compostela, Spain; [8]Pediatrics, Nutrition, and Development Research Unit, Universitat Rovira i Virgili, Reus, Spain; [9]Growth, Exercise, Nutrition and Development Research Group, Instituto Agroalimentario de Aragón, Instituto de Investigación Sanitaria Aragón, University of Zaragoza, Zaragoza, Spain; [10]Department of Physiatry and Nursing, University of Zaragoza, Zaragoza, Spain; [11]Center for Nutrition Research, University of Navarra, Pamplona, Spain; [12]IdisNA Institute for Health Research, Pamplona, Spain; [13]Department of Preventive Medicine and Public Health, University of Valencia, Valencia, Spain; [14]Cardiovascular Risk and Nutrition Research Group, Hospital del Mar Medical Research Institute, Barcelona, Spain; [15]Departament de Ciències Mèdiques Bàsiques, Facultat de Medicina, Universitat Rovira i Virgili, Reus, Spain; [16]Unitat de Suport a la Recerca Camp de Tarragona, Fundació Institut Universitari per a la Recerca a l'Atenció Primària de Salut Jordi Gol i Gurina, Reus, Spain; [17]Centre d'Atenció Primària St Pere, Direcció d'Atenció Primària Camp de Tarragona, Institut Català de la Salut, Reus, Spain; and [18]Department of Specific Didactics, Faculty of Education, University of Córdoba, Córdoba, Spain

*Co Senior authors and contributed equally.

A list of additional members of the CORALS study investigators is available at www.jpeds.com (**Appendix**).

Supported by the Consejo Nacional de Ciencia y Tecnología (769789, to T.G.-F.); the Spanish Ministry of Education, Culture, and Sports (FPU 17/01925, to I.P.-G.); and the Universitat Rovira i Virgili (2020PMF-PIPF-37, to C.G.-M.). V.L. received a Serra Hunter Fellowship from Generalitat de Catalunya. J.S.-S. is partially supported by the Catalan Institute for Research and Advanced Studies (ICREA) under the ICREA Academia program. Funds for the establishment of the CORALS cohort in the first year of the study (2019) were provided by an agreement between the Danone Institute and the Spanish Biomedical Research Center on Physiopathology of Obesity and Nutrition. The authors declare no conflicts of interest.

https://doi.org/10.1016/j.jpeds.2022.08.024

recent studies have reported that greater adiposity and other cardiometabolic risk factors also can be influenced by certain behaviors, such as eating speed and eating frequency, among others, in adults, adolescents, and children.[2] Eating quickly has been related to higher dietary energy intake, body mass index (BMI),[2] obesity prevalence,[3,4] and certain metabolic disorders.[2,5] In contrast, chewing slowly and increasing the number of chewing cycles during a meal have been negatively associated with adiposity.[6] It has been proposed that a slower eating speed might enhance the development of a satiety signal, which could limit the total food intake.[7]

Even though a universal definition for "speed of eating" has not been settled, a few authors define it as the time that a person takes to eat one meal.[8] Other authors refer to "eating rate" (as the estimation in grams per minute of food consumed)[9] or "energy intake rate" (energy intake by unit of time; kcalories per minute),[10] which may be considered more objective measures. Eating rate has been associated with food textures,[11] tastes,[10] and an increased overall energy.[12] Some authors have suggested that a fast eating rate is associated with high-fat content meals,[10] which usually have a softer texture and intense taste.[13] In addition, the consumption of soft-textured foods may be associated with a lower number of chewing cycles.[14] Meanwhile, a low energy intake rate has been associated with solid textures and low water content,[10] whereas a greater energy intake rate has been related to fat, sweet, and sour taste intensity,[10] as well as to increased consumption of ultra-processed foods that may contribute to poor dietary quality.[15] A high energy intake rate also has been associated with a faster eating rate.[16]

To our knowledge, associations between eating speed and diet quality have not been explored in children, and their associations with cardiometabolic risk factors have been rarely assessed. The present cross-sectional study aimed to assess the associations between eating speed, diet quality, adiposity, and cardiometabolic risk factors in a Spanish cohort of children aged 3-6 years.

Methods

This cross-sectional study is based on the baseline data of the Childhood Obesity Risk Assessment Longitudinal Study (CORALS), a prospective ongoing multicenter study conducted in preschool children that has a 10-year expected follow-up (https://corals.es/). The main aim of the CORALS project is to identify potential risk factors for childhood obesity. Briefly, eligible participants are children aged 3-6 years attending the selected schools across 7 Spanish cities (Barcelona, Córdoba, Pamplona, Reus, Santiago de Compostela, Valencia, and Zaragoza) whose tutors had agreed to participate. To be enrolled in the study, parents or caregivers had to sign a consent form, attend the inclusion visit, and complete several questionnaires (eg, leisure time physical activity, 3-day food record, social-demographic data). The exclusion criteria include belonging to a family with difficulty participating, comprehension or language difficulties, and unstable residence.

The Ethics Committee of each recruitment center approved the study protocol (reference nos. 051/2019, 4155/2019, 2019/18, 9/19, 09/2019, 19/27, and 2019/131), which was conducted following the standards of the Declaration of Helsinki.

Children aged 3-6 years recruited between March 2019 and June 2021 were selected (n = 1371). Participants who attended the baseline visit and completed the provided questionnaires, as well as those who had available data in the duration of breakfast, lunch, and dinner, were included in the present analyses. Children with current diagnosis of chronic diseases, such as diabetes mellitus type 2, hypertension, and familiar hypercholesterolemia, were excluded from these analyses.

Eating speed was assessed through the question "How long does it usually take for your child to eat in each meal?" Speed was calculated by summing the total time (in minutes) reported by parents in the 3 main meals (breakfast, lunch, and dinner). Then categories for slow, moderate, and fast-eating speed were determined by tertiles. These categories were recategorized so that the highest tertile represents the fast-eating category. Additional analyses were performed using as exposure variable the "slowness in eating" scale from the validated Child Eating Behaviour Questionnaire (CEBQ),[17] which has been recently validated in Spanish children of the same age.[18] The possible responses included on the CEBQ "slowness in eating" scale are "my child finishes his/her meal quickly," "my child eats slowly," "my child takes more than 30 minutes to finish a meal," and "my child eats more and more slowly during the course of a meal." This scale was categorized by tertiles that were recategorized, with the third tertile as the fast-eating category.

Body weight (in kilograms) and body fat mass (in kilograms) were assessed with a precision scale (Tanita MC780SMA; Tanita Europe, B.V.). Body fat mass was assessed by octopolar multifrequency bioelectrical impedance. Height (in centimeters) was assessed using a portable stadiometer (seca 213, Escala 20-205 cm; SECA). These assessments were performed with the child in light clothing, without shoes, and according to standard procedures. BMI was calculated as weight (in kilograms) divided by height (in meters squared) and classified as underweight/normal weight or overweight/obesity using the cutoff points defined by Cole et al.[19] Waist circumference was determined with a measuring tape (seca 201), midway between the lowest rib and the iliac crest. The fat mass index (FMI) was estimated as body fat mass (in kilograms)/squared height (in meters).[20]

Blood pressure was determined in the nondominant arm and measured 3 times (with 5-minute intervals between measurements) using an automatic oscillometer (Omron M3 Intellisense HEM-75051-EV; IOMRON Healthcare Europe B.V.) with a child-adapted cuff. Blood pressure was recorded as the mean of the 3 measurements for both systolic and diastolic blood pressures.

Blood samples were collected after a minimum of 8 hours of fasting. Serum samples were used to determine levels of fasting plasma glucose, total cholesterol, high-density

lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), and triglycerides by standard procedures. Non-HDL-c was calculated with the equation suggested by Frost et al.[21]

Diet quality was assessed by an ad hoc questionnaire of 18 items adapted to children from a 14-item Mediterranean diet questionnaire validated in an elderly population.[22] This questionnaire evaluates the consumption of typical foods in the Mediterranean diet (eg, olive oil, fruits, vegetables, cereals, nuts, seeds, cheese, yogurt, legumes, fish, seafood, poultry, rabbit), as well as additional foods that are not included in this dietary pattern (ie, butter, cream, carbonated/sugary beverages, junk food, dairy desserts, and precooked/ready-to-eat foods). The number of servings has been adapted for several items, and certain typical foods consumed by children (eg, gummy worms, sweets, petit suisse) are also included in this questionnaire (**Table I**; available at www.jpeds.com). Compliance with each of the 18 items was scored as 1 point; thus, the total score ranged from 0 to 18, with 0 representing no adherence and 18 representing the greatest adherence.

A set of self-administered questionnaires completed by parents or caregivers at home was used to collect information on early life factors, maternal data (eg, age, BMI, educational level, socio-professional status), and lifestyle patterns, among others. Weight at birth (kilograms), mother's weight gain during pregnancy (kilograms), and breastfeeding (months of age) were recorded.

A physical activity questionnaire based on the Outdoor Playtime Checklist and the Outdoor Playtime Recall Questions[23] was provided. An additional ad hoc questionnaire with 13 components based on leisure physical activity that includes sedentary behaviors was used to determine the active lifestyle score (**Table II**; available at www.jpeds.com).

Sleep pattern was explored with the questions "how long does your child sleep at night during weekdays and on weekend days or holidays?" and "how long does your child nap during weekdays and on weekend days or holidays?" The total sleep duration (hours per day) was calculated summing night sleep hours and nap time on weekdays and weekend days or holidays divided by 7 (ie, the total days of the week).

Trained dietitians assessed the dietary intake using a semi-quantitative 125-item food frequency questionnaire adapted from a food frequency questionnaire validated in adults.[24] The 9 possible answers ranged from "never" to "more than 6 times per day" and were transformed into grams or milliliters per day using the standard portion size of each item. The CESNID (Centre d'Enseyament Superior de Nutrició i Dietètica) database was used to calculate total energy and nutrient intake.[25] Total energy intake was estimated according to Goldberg cutoffs adapted to children[26]; participants with missing data or implausible reported energy intake were excluded from the analyses.

The present analyses were conducted using the CORALS database updated to December 2021. To compare general characteristics and dietary variables among categories of eating speed, one-factor ANOVA, the Kruskal–Wallis test, or χ^2 test was used. The data were presented as mean ± SD or median (IQR) for quantitative variables and as percentage (number) for qualitative variables. The Kolmogorov–Smirnov test was used to assess the normal distribution of variables. Missing data <10% on covariates was imputed to the mean.[27] Multiple linear regression models were fitted to assess associations between eating speed (exposure) and different outcomes—waist circumference, FMI, systolic blood pressure, diastolic blood pressure, fasting plasma glucose, total cholesterol, HDL-c, LDL-c, non-HDL-c, triglycerides, and Mediterranean diet score—which were reported as β coefficients and 95% CIs. Logistic regression models were fitted to explore the association between eating speed and weight status (outcome), expressed as OR and 95% CIs. All models were adjusted by the following confounders: center, sex, age, maternal BMI, and mother's educational level, total energy intake, breastfeeding, total minutes of physical activity a week, and total hours of sleep per day. In our analyses, the slower category of eating speed served as the reference. Interaction analyses were performed by sex, maternal factors (BMI and educational level), weight at birth, and sedentary behaviors.

Sensitivity analyses were performed for FMI excluding those participants aged 3-4 years owing to a lack of validation of bioimpedance equations (Tanita MC780SMA) in children aged <5 years. Correlation analyses were performed between the variable calculated from the CEBQ "slowness in eating" scale and the eating speed variable estimated in minutes per day as well as between the 18-item and the 14-item questionnaires of adherence to the Mediterranean diet. All analyses were conducted using Stata 14 (StataCorp), and statistical significance was set at a 2-tailed P value <.05.

Results

Among the total of 1371 participants who attended the first visit, 49 were excluded for not meeting the inclusion criteria. Subsequently, 193 children had missing data for the time spent at breakfast, lunch, and/or dinner, precluding estimation of eating speed. In addition, 143 participants were excluded because of missing data or implausible reported energy intake. Finally, 30 participants with a current diagnosis of chronic diseases also were excluded from the analyses. Therefore, a total sample of 956 children (49% girls) were included in the analyses (**Figure**). For 88% of the study sample, the questionnaires were completed by the mother.

The main characteristics of the sample according to category of eating speed at baseline are shown in **Table III**. The mean age was 4.8 ± 1 years. Compared with participants in the slow-eating category, children with fast eating speed were more likely to have overweight or obesity, larger waist circumference, greater FMI, and higher systolic blood pressure and fasting plasma glucose concentration ($P < .05$). Mothers whose children were categorized as fast-eaters had higher BMI and weight status, as well as lower educational level ($P < .05$), compared with slow eaters. No other significant between-group differences were observed.

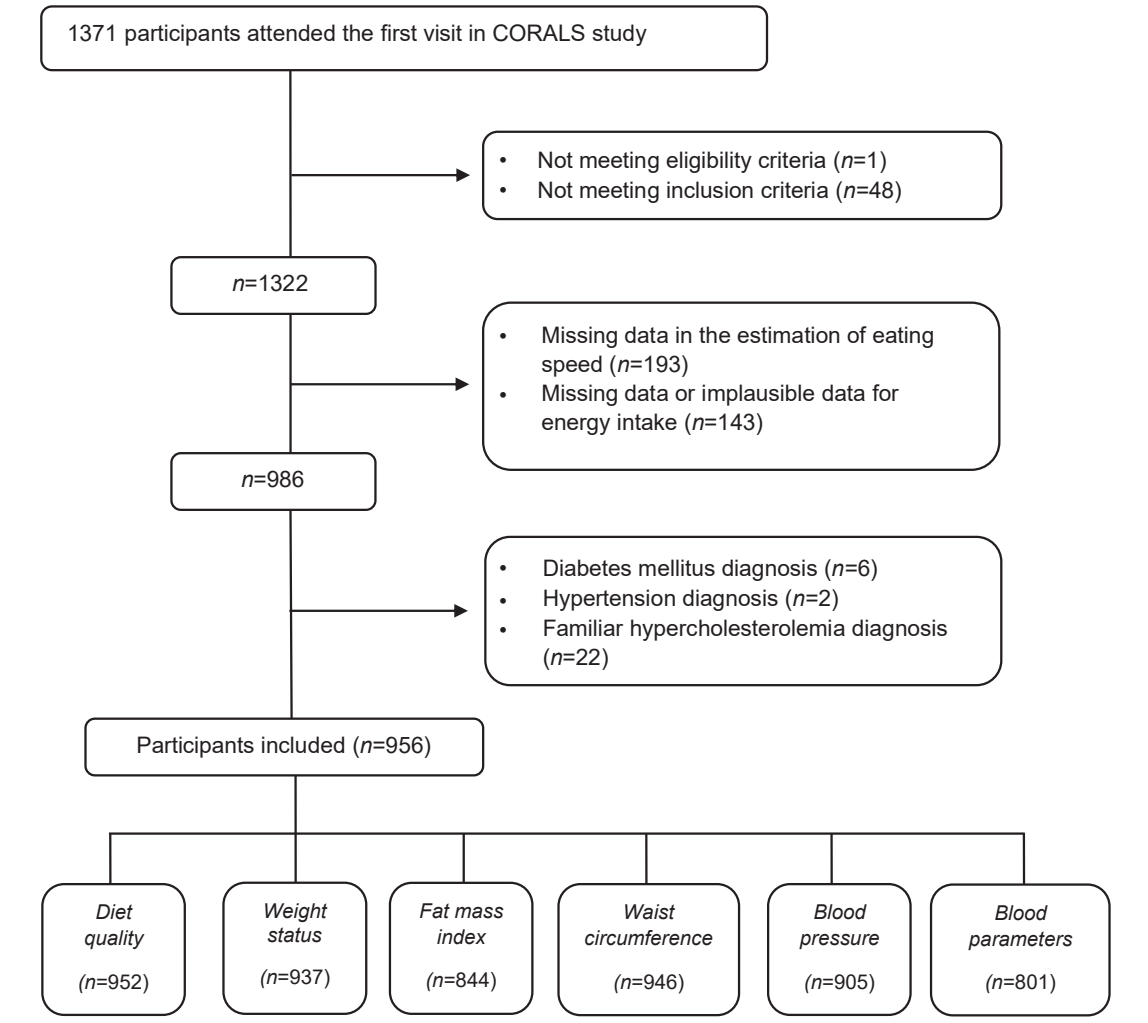

Figure. Flow diagram for the CORALS cohort.

Table IV lists the dietary characteristics according to eating speed categories. The percentage of total daily energy intake from carbohydrates differed significantly across the eating speed categories, higher in the fast-eating category. Participants in the fast-eating category also were more likely to have lower intakes of total fatty acids, monounsaturated fatty acids, and protein per body weight, as well as lower nut consumption and adherence to the Mediterranean diet.

The associations between eating speed category and outcome are shown in **Table V**. Compared with children in the slow-eating category, in unadjusted models and after adjusting for potential confounders, participants in the fast-eating category showed a higher risk for overweight/obesity (OR, 2.9; 95% CI, 1.8-4.4; $P < .01$). Compared with slower eaters, fast eaters had higher values for waist circumference, FMI, systolic blood pressure, and fasting plasma glucose concentration, as well as lower diet quality (β, −0.5; 95% CI, −0.9 to −0.1; $P < .05$). When we excluded those children aged <5 years from the association with FMI, the results remained significant in the full adjusted model (β, 0.5; 95% CI, 0.2-0.8; $P < .01$). Compared with the slow-eating category, an inverse association was found between the fast-eating category and total cholesterol in the full adjusted model. No significant associations were observed between eating speed and the other cardiometabolic risk factors.

Data on eating speed measured by the CEBQ "slowness in eating" scale also showed positive associations between the fast-eating category and overweight/obesity (OR, 4.2; 95% CI, 2.7-6.4; $P < .01$), waist circumference (β, 3.1; 95% CI, 2.1-4.2; $P < .01$), and FMI (β, 0.7; 95% CI, 0.5-0.9; $P < .01$).

Table III. General characteristics of the study population according to eating speed categories

Characteristics	Eating speed categories			*P* value*
	Slow (>85 min), N = 291	Moderate (66-85 min), N = 325	Fast (<65 min), N = 340	
Eating speed, total min/day/3 main meals, mean ± SD	108.3 ± 18.3	76.2 ± 4.9	53.8 ± 9.2	**<.001**
Age, y, mean ± SD	4.8 ± 1.1	4.8 ± 1.0	4.9 ± 1.0	.100
Girls, % (n)	50.5 (147)	48.9 (159)	48.2 (164)	.845
Adiposity				
Weight status, % (n)				**<.001**
Underweight or normal weight	87.5 (251)	79.2 (251)	70.3 (234)	
Overweight or obesity	12.5 (36)	20.8 (66)	29.7 (99)	
Waist circumference, cm, mean ± SD	50.3 ± 7.5	52.4 ± 6.0	53.4 ± 8.3	**<.001**
FMI, kg/m^2, mean ± SD	3.7 ± 1.2	3.8 ± 1.4	4.1 ± 1.3	**.003**
Cardiovascular risk factors				
Systolic blood pressure, mmHg, mean ± SD	102.0 ± 12.9	103.1 ± 13.2	105.2 ± 12.9	**.009**
Diastolic blood pressure, mmHg, mean ± SD	64.5 ± 12.6	63.9 ± 12.5	66.1 ± 12.0	.068
Fasting glucose, mg/dL, mean ± SD	75.8 ± 8.8	77.8 ± 8.7	78.8 ± 9.9	**<.001**
Total cholesterol, mg/dL, median (IQR)	167 (149-186.5)	166 (149-184)	162 (145-179)	.055
HDL-c, mg/dL, median (IQR)	56 (48.5-66)	58 (50-67)	56.4 (49-64.7)	.495
LDL-c, mg/dL, median (IQR)	96 (83-112)	95 (83-112.6)	92 (78-105.1)	.083
Non-HDL-c, mg/dL, median (IQR)	107 (94-123.5)	107 (93-126)	104 (90-119)	.215
Triglycerides, mg/dL, median (IQR)	53 (44.5-67.5)	53 (42-65)	52 (43-66)	.556
Early life factors				
Weight at birth, kg, mean ± SD	3.3 ± 0.5	3.3 ± 0.5	3.2 ± 0.6	.554
Breastfeeding duration, % (n)				.872
<6 mo	40.3 (94)	42.7 (108)	41.6 (106)	
≥6 mo	59.7 (139)	57.3 (145)	58.4 (149)	
Mother's weight gain during pregnancy, kg, mean ± SD	12.5 ± 4.7	12.7 ± 4.6	12.3 ± 4.5	.589
Lifestyle factors				
Sports and physical activities, min/wk	197.2 ± 117.7	179.0 ± 105.1	178.7 ± 115.2	.069
Total sleep duration per day, h	10.4 ± 0.9	10.4 ± 1.1	10.4 ± 1.3	.667
Active lifestyle score, 0-13 points	9.2 ± 1.6	9.1 ± 1.6	9.1 ± 1.6	.726
Maternal factors				
Age, y, mean ± SD	40.7 ± 4.5	41.4 ± 7.4	40.6 ± 5.8	.162
BMI, kg/m^2, mean ± SD	24.3 ± 4.6	25.1 ± 4.8	25.1 ± 5.4	**.049**
Weight status, % (n)				**.022**
Underweight or normal weight	67.7 (197)	56.9 (185)	61.2 (208)	
Overweight or obesity	32.3 (94)	43.1 (140)	38.8 (132)	
Educational level, % (n)				**.021**
Primary or lower	7.2 (21)	9.5 (31)	12.1 (41)	
Secondary	40.2 (117)	35.1 (114)	43.8 (149)	
Academic–graduated or underreported	52.6 (153)	55.4 (180)	44.1 (150)	
Socio-professional category, % (n)				.583
Homemaker/student/retired/unemployed	26.5 (77)	30.2 (98)	27.8 (93)	
Employee	73.5 (214)	69.9 (227)	72.2 (242)	

*P values calculated using the χ^2 test for categorical variables and ANOVA for continuous variables. A P value <.05 was considered significant (bold type).

No significant associations were observed for other cardiometabolic risk factors, but the direction of the associations was the same.

In correlation analyses, both eating speed variables (minutes/day and the CEBQ "slowness in eating" scale) were positively correlated ($r = 0.43$; $P < .001$), as well as the 2 questionnaires assessing adherence to the Mediterranean diet (the 18-item and 14-item questionnaires) ($r = 0.39$; $P < .01$).

Interaction analyses between eating speed category, sex, mother's BMI and educational level, weight at birth, and sedentary behaviors were not significant.

Discussion

Our results on eating speed and adiposity are in line with previous observational studies reporting that self-reported fast eating speed is associated with higher weight status and larger waist circumference in adults[28,29] and Asian children.[12,30] In addition, a longitudinal study conducted in Japanese girls reported a positive association between eating speed and body fat mass percentage, but FMI was not assessed.[31] Moreover, an 8-week interventional trial in American children that aimed to decelerate eating speed through nutritional education, timers, and interactive activities with families resulted in a decrease in eating speed in association with lower BMI.[32]

The association between eating speed and blood pressure also has been explored in Asian adults, in which fast eating was associated with higher blood pressure.[33] Similarly in children, Yamagishi et al observed higher systolic blood pressure in boys with continuous fast-eating speed; the association was not significant in girls, however.[34] In line with our results regarding eating speed and fasting plasma glucose concentration, Nohara et al reported men, but not women, who

Table IV. Dietary characteristics according to eating speed category

Characteristics	Eating speed category			*P* value*
	Slow (>85 min), N = 291	Moderate (66-85 min), N = 325	Fast (<65 min), N = 340	
Dietary intake contribution				
Total energy intake, kcal/day, mean ± SD	1698 ± 340	1688 ± 351	1714 ± 370	.632
Carbohydrates, %, mean ± SD	42.0 ± 5.5	42.9 ± 5.4	43.7 ± 5.5	**<.001**
Proteins, %, mean ± SD	15.1 ± 2.1	15.0 ± 2.2	14.9 ± 2.0	.505
Total fatty acids, %, mean ± SD	42.9 ± 5.9	42.1 ± 5.7	41.4 ± 5.9	**.007**
Saturated fatty acids, %, mean ± SD	14.1 ± 1.9	14.0 ± 2.0	13.8 ± 2.1	.184
Monounsaturated fatty acids, %, mean ± SD	19.6 ± 4.7	18.9 ± 4.4	18.4 ± 4.4	**.006**
Polyunsaturated fatty acids, %, mean ± SD	6.2 ± 1.4	6.1 ± 1.7	6.2 ± 1.8	.941
Protein intake per body weight, g/kg/d, mean ± SD	3.5 ± 0.9	3.3 ± 0.8	3.2 ± 1.0	**<.001**
Fiber, g/1000 kcal, mean ± SD	8.5 ± 2.3	8.5 ± 2.4	8.5 ± 2.3	.985
Source goal, % (n)				.674
<14 g/1000 kcal	95.9 (279)	96.9 (315)	97.1 (330)	
≥14 g/1000 kcal	4.1 (12)	3.1 (10)	2.9 (10)	
Sodium, mg/dL, mean ± SD	2308 ± 736	2320 ± 719	2307 ± 684	.967
Diet quality				
Mediterranean Diet Score, 0-18 points	11.1 ± 2.9	10.7 ± 2.8	10.5 ± 2.5	**.015**
Dairy products, mean ± SD				
Milk, g/day	324.9 ± 239.1	312.6 ± 221.7	320.9 ± 234.1	.795
Yogurt, g/day	105.0 ± 79.2	111.2 ± 85.7	117.6 ± 110.9	.240
Cheese, g/day	12.5 ± 10.8	12.3 ± 12.9	12.6 ± 12.3	.959
Derivative dairy products, g/day	80.2 ± 82.9	85.5 ± 93.9	90.4 ± 99.5	.389
Protein foods, mean ± SD				
White meat, g/day	24.7 ± 9.8	23.9 ± 9.0	23.9 ± 9.2	.492
Unprocessed red meat, g/day	18.8 ± 11.9	18.9 ± 13.2	19.0 ± 13.3	.974
Processed and derivatives meat products, g/day	26.3 ± 16.6	25.7 ± 14.4	24.5 ± 14.7	.319
Egg, g/day	23.5 ± 8.6	23.2 ± 8.5	23.2 ± 10.2	.889
Fish and seafood, g/day	34.9 ± 17.0	33.9 ± 17.4	32.5 ± 17.9	.222
Vegetables and fruits, mean ± SD				
Vegetables, g/day	76.8 ± 52.8	77.4 ± 51.1	75.8 ± 52.7	.921
Tubers, g/day	40.3 ± 18.6	39.9 ± 19.2	41.4 ± 20.3	.576
Fruits, g/day	188.6 ± 113.2	190.0 ± 124.1	181.4 ± 122.9	.611
Nuts, g/day, mean ± SD	4.2 ± 5.7	3.3 ± 4.4	3.5 ± 4.6	**.045**
Cereals and legumes, mean ± SD				
Legumes, g/day	14.0 ± 6.4	14.0 ± 6.2	14.3 ± 8.5	.815
Refined bread, cereals, and pasta, g/day	70.3 ± 32.2	72.7 ± 34.9	74.5 ± 38.4	.333
Brown bread, cereals, pasta and rice, g/day	5.5 ± 13.5	3.7 ± 10.7	4.5 ± 11.2	.163
Miscellaneous, mean ± SD				
Oil and fats, g/day	27.7 ± 16.6	26.4 ± 15.7	25.4 ± 15.3	.212
Pastries, g/day	38.3 ± 29.4	40.6 ± 28.6	41.2 ± 30.5	.454
Sugars and candies, g/day	12.9 ± 10.5	13.2 ± 10.6	13.6 ± 11.0	.696
Beverages, mean ± SD				
Water, g/day	852.6 ± 390.9	859.0 ± 371.8	348.9 ± 363.2	.941
Sugary beverages, g/day	110.5 ± 126.9	104.4 ± 130.8	124.8 ± 130.5	.111
Tea and infusions, g/day	5.3 ± 28.3	6.0 ± 24.7	8.8 ± 32.9	.276

**P* values calculated by the χ^2 test for categorical variables and ANOVA for continuous variables. *P* values <.05 were considered significant (bold type).

reported fast-eating speed had higher fasting glucose concentrations and blood pressure values.[35] Another study of middle-aged Japanese adults reported an independent and positive association between fast eating and insulin resistance; however, the association with blood glucose level was not assessed.[36] To date, no published studies have explored the association between eating speed and fasting plasma glucose in children. In contrast with a recent review that reported significant associations between eating speed and triglyceride and lower HDL-c levels in adults,[2] our results did not show any significant associations in a sample of children. Inconclusive results in these studies may be related to variations in populations (eg, ethnicity, age, culture), as well as additional differences in methodology.

In the present study, we observed an inverse association between eating speed and the adherence to Mediterranean diet. This result could be explained by the main characteristics of the Mediterranean diet, such as its high content of plant-based foods (which provide high amounts of dietary fiber) and fish/seafood and low content of red meat/processed meat products.[37] Even though there were no significant differences in the majority of food groups in our analyses, the overall beneficial effect of the Mediterranean diet is greater than the effects of its individual components. Furthermore, in a meta-analysis[38] and a randomized-crossover study,[39] positive effects were observed between the consumption of fiber or protein and appetite suppression as well as satiety, which might have an impact on eating speed.[7] Additionally, associations between faster eating,[16] sweet-tasting foods,[10] and ultra-processed foods[40] have been reported. Ultra-processed foods are characterized by their high energy, salt, sugar, and fat content,[41] which

Table V. Crude and multivariate β-coefficients (95% CI) or OR (95% CI) of adiposity, diet quality, and cardiometabolic risk factors according to eating speed categories in the total population

	Eating speed category		
Variables	Slow (>85 min)	Moderate (66-85 min)	Fast (<65 min)
Adiposity			
Weight status, n	287	317	333
Crude model	1 (ref)	1.8 (1.2-2.9)†	2.9 (1.9-4.5)†
Model 1	1 (ref)	1.8 (1.1-2.8)*	2.8 (1.8-4.4)†
Model 2	1 (ref)	1.8 (1.1-2.8)*	2.9 (1.8-4.4)†
Waist circumference, n	285	323	338
Crude model	0 (ref)	1.9 (0.7-3.0)†	2.9 (1.8-4.0)†
Model 1	0 (ref)	1.7 (0.6-2.7)†	2.5 (1.4-3.6)†
Model 2	0 (ref)	1.7 (0.6-2.8)†	2.5 (1.4-3.8)†
FMI, n	258	283	303
Crude model	0 (ref)	0.2 (−0.1 to 0.4)	0.4 (0.2-0.6)†
Model 1	0 (ref)	0.1 (−0.1 to 0.3)	0.3 (0.1-0.5)†
Model 2	0 (ref)	0.1 (−0.1 to 0.3)	0.3 (0.1-0.5)†
Cardiovascular risk factors			
Systolic blood pressure, n	274	304	325
Crude model	0 (ref)	1.0 (−1.1 to 3.2)	3.2 (1.1-5.3)†
Model	0 (ref)	0.8 (−1.3 to 2.9)	2.8 (0.7-4.9)†
Model 2	0 (ref)	0.8 (−1.3 to 3.0)	2.8 (0.6-4.9)*
Diastolic blood pressure, n	275	305	325
Crude model	0 (ref)	−0.6 (−2.6 to 1.4)	1.6 (−0.4 to 3.6)
Model 1	0 (ref)	−0.7 (−2.7 to 1.3)	1.6 (−0.4 to 3.6)
Model 2	0 (ref)	−0.8 (−2.8 to 1.2)	1.5 (−0.5 to 3.5)
Fasting glucose, n	241	270	290
Crude model	0 (ref)	2.0 (0.4-3.6) *	3.0 (1.5-4.6)†
Model 1	0 (ref)	2.2 (0.7-3.7)†	2.8 (1.3-4.3)†
Model 2	0 (ref)	2.1 (0.6-3.6)†	2.7 (1.2-4.2)†
Total cholesterol, n	240	271	287
Crude model	0 (ref)	−0.5 (−5.4 to 4.4)	−4.5 (−9.4 to 0.3)
Model 1	0 (ref)	−0.2 (−5.1 to 4.6)	−4.5 (−9.4 to 0.3)
Model 2	0 (ref)	−0.6 (−5.4 to 4.3)	−5.0 (−9.8 to −0.2)*
HDL-c, n	240	270	286
Crude model	0 (ref)	0.0 (−2.3 to 2.4)	−1.0 (−3.3 to 1.3)
Model 1	0 (ref)	0.2 (−2.2 to 2.5)	−0.9 (−3.3 to 1.4)
Model 2	0 (ref)	0.2 (−2.1 to 2.6)	−0.9 (−3.2 to 1.5)
LDL-c, n	225	255	273
Crude model	0 (ref)	0.3 (−4.1 to 4.7)	−3.2 (−7.4 to 1.0)
Model 1	0 (ref)	0.3 (−4.1 to 4.6)	−3.2 (−7.5 to 1.0)
Model 2	0 (ref)	0.1 (−4.2 to 4.5)	−3.7 (−7.9 to 0.6)
Non-HDL-c, n	240	270	286
Crude model	0 (ref)	−1.0 (−5.4 to 3.4)	−3.5 (−7.8 to 0.9)
Model 1	0 (ref)	−0.8 (−5.2, to 3.6)	−3.5 (−7.9 to 0.9)
Model 2	0 (ref)	−1.2 (−5.6 to 3.2)	−4.1 (−8.4 to 0.3)
Triglycerides, n	240	271	286
Crude model	0 (ref)	−1.6 (−5.3 to 2.1)	−2.3 (−6.0 to 1.3)
Model 1	0 (ref)	−2.3 (−5.9 to 1.2)	−3.4 (−7.0 to 0.1)
Model 2	0 (ref)	−2.4 (−6.0 to 1.2)	−3.5 (−7.1 to 0.1)
Diet quality			
Mediterranean Diet Score, points	289	324	339
Crude model	0 (ref)	−0.3 (−0.8 to 0.1)	−0.6 (−1.1 to −0.2)†
Model 1	0 (ref)	−0.3 (−0.7 to 0.1)	−0.5 (−0.9 to −0.1)*
Model 2	0 (ref)	−0.3 (−0.7 to 0.1)	−0.5 (−0.9 to −0.1)*

Multiple linear regressions models were fitted and expressed in β coefficients (95% CI). For weight status, logistic regressions models were fitted and expressed in OR (95% CI). The weight status is a dichotomous outcome (underweight or normal weight -1- and overweight or obesity -2-).
Model 1 adjusted by center, sex, age, maternal BMI, and mother's educational level.
Model 2 adjusted additionally by total energy intake, breastfeeding, total minutes of physical activity per week, and total hours of sleep per day.
*Associations with $P < .05$.
†Associations with $P < .01$.

may contribute to food reinforcement and a higher overall energy intake,[41] perhaps because of their high palatability.[40] On the other hand, an unexpected result in our analyses was the association between fast eating speed and lower total cholesterol. In this sense, a potential association between lower fat intake and a decrease in total cholesterol and LDL-c has been reported,[42] which could partially explain this result, as our analyses showed a lower percentage of total fatty acids in those participants in the fast-eating category.

This study has some limitations that merit mention. First, because it is a cross-sectional study, we cannot draw cause-and-effect conclusions or discard bidirectional associations. Second, our study sample included Spanish preschool children, and thus our findings cannot be extrapolated to other populations. Third, we cannot dismiss the possibility that associations might be related to residual confounding or that undetected cardiometabolic disorders in our study population may exist because of age. Fourth, equations for bioelectrical impedance analysis have not been designed for children aged <5 years; however results from the analyses excluding children aged 3-4 years remained similar to those in the total population. Fifth, although eating speed was self-reported, it was estimated according to the time reported by main caregivers, not based on self-perception. Moreover, analyses from the CEBQ "slowness in eating" scale showed similar results as those observed in the main analyses for adiposity. Sixth, the 18-item Mediterranean diet questionnaire has not been validated in children; nevertheless, it was positively correlated to the validated 14-item Mediterranean diet questionnaire, and results should be interpreted cautiously.

In conclusion, eating fast is associated with higher levels of adiposity and certain cardiometabolic risk factors, as well as lower adherence to the Mediterranean diet in Spanish children. However, further long-term studies and clinical trials are needed to confirm these associations. ■

We thank all the CORALS participants and their parents or caregivers as well as the health centers and primary schools for their collaboration, the CORALS personnel for their outstanding support, and the staff of all associated primary care centers for their exceptional work. We particularly acknowledge the Institut d'Investigacion Sanitaria Pere Virgili (IISPV) Biobank (PT20/00197), which is integrated in the Instituto de Salud Carlos III (ISCIII) Platform for Biobanks and Biomodel.

Submitted for publication Apr 19, 2022; last revision received Aug 15, 2022; accepted Aug 18, 2022.

Reprint requests: Nancy Babio, PhD, Unitat de Nutrició Humana, Departament de Bioquímica i Biotecnología, Universitat Rovira i Virgili, Reus, Spain. E-mail: jordi.salas@urv.cat; nancy.babio@urv.cat

Data Statement

Data sharing statement available at www.jpeds.com.

References

1. di Cesare M, Sorić M, Bovet P, Miranda JJ, Bhutta Z, Stevens GA, et al. The epidemiological burden of obesity in childhood: a worldwide epidemic requiring urgent action. BMC Med 2019;17:212. https://doi.org/10.1186/s12916-019-1449-8
2. Garcidueñas-Fimbres TE, Paz-Graniel I, Nishi SK, Salas-Salvadó J, Babio N. Eating speed, eating frequency, and their relationships with diet quality, adiposity, and metabolic syndrome, or its components. Nutrients 2021;13:1687. https://doi.org/10.3390/nu13051687
3. Okubo H, Murakami K, Masayasu S, Sasaki S. The relationship of eating rate and degree of chewing to body weight status among preschool children in Japan: a nationwide cross-sectional study. Nutrients 2018;11:64. https://doi.org/10.3390/nu11010064
4. Kolay E, Bykowska-Derda A, Abdulsamad S, Kaluzna M, Samarzewska K, Ruchala M, et al. Self-reported eating speed is associated with indicators of obesity in adults: a systematic review and meta-analysis. Healthcare (Basel) 2021;9:1559. https://doi.org/10.3390/healthcare9111559
5. Paz-Graniel I, Babio N, Mendez I, Salas-Salvadó J. Association between eating speed and classical cardiovascular risk factors: a cross-sectional study. Nutrients 2019;11:83. https://doi.org/10.3390/nu11010083
6. Hamada Y, Miyaji A, Hayashi Y, Matsumoto N, Nishiwaki M, Hayashi N. Objective and subjective eating speeds are related to body composition and shape in female college students. J Nutr Sci Vitaminol (Tokyo) 2017;63:174-9. https://doi.org/10.3177/jnsv.63.174
7. Kokkinos A, le Roux CW, Alexiadou K, Tentolouris N, Vincent RP, Kyriaki D, et al. Eating slowly increases the postprandial response of the anorexigenic gut hormones, peptide YY and glucagon-like peptide-1. J Clin Endocrinol Metab 2010;95:333-7. https://doi.org/10.1210/jc.2009-1018
8. Ekuni D, Furuta M, Takeuchi N, Tomofuji T, Morita M. Self-reports of eating quickly are related to a decreased number of chews until first swallow, total number of chews, and total duration of chewing in young people. Arch Oral Biol 2012;57:981-6. https://doi.org/10.1016/j.archoralbio.2012.02.001
9. Spiegel TA, Wadden TA, Foster GD. Objective measurement of eating rate during behavioral treatment of obesity. Behav Ther 1991;22:61-7. https://doi.org/10.1016/S0005-7894(05)80244-8
10. van den Boer J, Werts M, Siebelink E, de Graaf C, Mars M. The availability of slow and fast calories in the Dutch diet: the current situation and opportunities for interventions. Foods 2017;6:87. https://doi.org/10.3390/foods6100087
11. Forde CG, van Kuijk N, Thaler T, de Graaf C, Martin N. Oral processing characteristics of solid savoury meal components, and relationship with food composition, sensory attributes and expected satiation. Appetite 2013;60:208-19. https://doi.org/10.1016/j.appet.2012.09.015
12. Fogel A, Goh AT, Fries LR, Sadananthan SA, Velan SS, Michael N, et al. Faster eating rates are associated with higher energy intakes during an ad libitum meal, higher BMI and greater adiposity among 4·5-year-old children: results from the Growing Up in Singapore Towards Healthy Outcomes (GUSTO) cohort. Br J Nutr 2017;117:1042-51. https://doi.org/10.1017/S0007114517000848
13. Viskaal-van Dongen M, Kok FJ, de Graaf C. Eating rate of commonly consumed foods promotes food and energy intake. Appetite 2011;56:25-31. https://doi.org/10.1016/j.appet.2010.11.141
14. Forde CG, Leong C, Chia-Ming E, McCrickerd K. Fast or slow-foods? Describing natural variations in oral processing characteristics across a wide range of Asian foods. Food Funct 2017;8:595-606. https://doi.org/10.1039/c6fo01286h
15. Poti JM, Mendez MA, Ng SW, Popkin BM. Is the degree of food processing and convenience linked with the nutritional quality of foods purchased by US households? Am J Clin Nutr 2015;101:1251-62. https://doi.org/10.3945/ajcn.114.100925
16. Teo PS, van Dam RM, Forde CG. Combined impact of a faster self-reported eating rate and higher dietary energy intake rate on energy intake and adiposity. Nutrients 2020;12:3264. https://doi.org/10.3390/nu12113264
17. Carnell S, Wardle J. Measuring behavioural susceptibility to obesity: validation of the child eating behaviour questionnaire. Appetite 2007;48:104-13. https://doi.org/10.1016/j.appet.2006.07.075
18. Jimeno-Martínez A, Maneschy I, Moreno LA, Bueno-Lozano G, de Miguel-Etayo P, Flores-Rojas K, et al. Reliability and validation of the Child Eating Behavior Questionnaire in 3- to 6-year-old Spanish children. FrontPsychol 2022;13:705912. https://doi.org/10.3389/fpsyg.2022.705912
19. Cole TJ, Lobstein T. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. Pediatr Obes 2012;7:284-94. https://doi.org/10.1111/j.2047-6310.2012.00064.x
20. VanItallie TB, Yang MU, Heymsfield SB, Funk RC, Boileau RA. Height-normalized indices of the body's fat-free mass and fat mass: potentially useful indicators of nutritional status. Am J Clin Nutr 1990;52:953-9. https://doi.org/10.1093/ajcn/52.6.953

21. Frost PH, Havel RJ. Rationale for use of non–high-density lipoprotein cholesterol rather than low-density lipoprotein cholesterol as a tool for lipoprotein cholesterol screening and assessment of risk and therapy. Am J Cardiol 1998;81:26B-31B. https://doi.org/10.1016/s0002-9149(98)00034-4
22. Schröder H, Fitó M, Estruch R, Martínez-González MA, Corella D, Salas-Salvadó J, et al. A short screener is valid for assessing Mediterranean diet adherence among older Spanish men and women. J Nutr 2011;141:1140-5. https://doi.org/10.3945/jn.110.135566
23. Verbestel V, De Henauw S, Bammann K, Barba G, Hadjigeorgiou C, Eiben G, et al. Are context-specific measures of parental-reported physical activity and sedentary behaviour associated with accelerometer data in 2-9-year-old European children? Public Health Nutr 2015;18:860-8. https://doi.org/10.1017/S136898001400086X
24. Fernández-Ballart JD, Piñol JL, Zazpe I, Corella D, Carrasco P, Toledo E, et al. Relative validity of a semi-quantitative food-frequency questionnaire in an elderly Mediterranean population of Spain. Br J Nutr 2010;103:1808-16. https://doi.org/10.1017/S0007114509993837
25. Farran A, Zamora R, Cervera P, Centre d'Enseyament Superior de Nutrició i Dietètica. Tablas de composición de alimentos del CESNID. 3rd ed. Universitat de Barcelona: McGraw-Hill Interamericana; 2004 [in Spanish].
26. Börnhorst C, Huybrechts I, Ahrens W, Eiben G, Michels N, Pala V, et al. Prevalence and determinants of misreporting among European children in proxy-reported 24 h dietary recalls. Br J Nutr 2013;109:1257-65. https://doi.org/10.1017/S0007114512003194
27. de Waal T, Pannekoek J, Scholtus S. Statistical data editing and imputation. Hoboken (NJ): John Wiley & Sons; 2011.
28. Yamane M, Ekuni D, Mizutani S, Kataoka K, Sakumoto-Kataoka M, Kawabata Y, et al. Relationships between eating quickly and weight gain in Japanese university students: a longitudinal study. Obesity (Silver Spring) 2014;22:2262-6. https://doi.org/10.1002/oby.20842
29. Zhu B, Haruyama Y, Muto T, Yamazaki T. Association between eating speed and metabolic syndrome in a three-year population-based cohort study. J Epidemiol 2015;25:332-6. https://doi.org/10.2188/jea.JE20140131
30. Okubo H, Miyake Y, Sasaki S, Tanaka K, Hirota Y. Rate of eating in early life is positively associated with current and later body mass index among young Japanese children: the Osaka Maternal and Child Health Study. Nutr Res 2017;37:20-8. https://doi.org/10.1016/j.nutres.2016.11.011
31. Ochiai H, Shirasawa T, Ohtsu T, Nishimura R, Morimoto A, Hoshino H, et al. The impact of eating quickly on anthropometric variables among schoolgirls: a prospective cohort study in Japan. Eur J Public Health 2014;24:691-5. https://doi.org/10.1093/eurpub/ckt120
32. Faith MS, Diewald LK, Crabbe S, Burgess B, Berkowitz RI. Reduced eating pace (RePace) behavioral intervention for children prone to or with obesity: does the turtle win the race? Obesity (Silver Spring) 2019;27:121-9. https://doi.org/10.1002/oby.22329
33. Tao L, Yang K, Huang F, Liu X, Li X, Luo Y, et al. Association between self-reported eating speed and metabolic syndrome in a Beijing adult population: a cross-sectional study. BMC Public Health 2018;18:855. https://doi.org/10.1186/s12889-018-5784-z
34. Yamagishi K, Sairenchi T, Sawada N, Sunou K, Sata M, Murai U, et al. Impact of speed-eating habit on subsequent body mass index and blood pressure among schoolchildren the Ibaraki Children's Cohort Study (IBA-CHIL). Circ J 2018;82:419-22. https://doi.org/10.1253/circj.CJ-17-0287
35. Nohara A, Maejima Y, Shimomura K, Kumamoto K, Takahashi M, Akuzawa M, et al. Self-awareness of fast eating and its impact on diagnostic components of metabolic syndrome among middle-aged Japanese males and females. Endocr Regul 2015;49:91-6. https://doi.org/10.4149/endo_2015_02_91
36. Otsuka R, Tamakoshi K, Yatsuya H, Wada K, Matsushita K, OuYang P, et al. Eating fast leads to insulin resistance: findings in middle-aged Japanese men and women. Prev Med 2008;46:154-9. https://doi.org/10.1016/j.ypmed.2007.07.031
37. Davis C, Bryan J, Hodgson J, Murphy K. Definition of the Mediterranean Diet; a literature review. Nutrients 2015;7:9139-53. https://doi.org/10.3390/nu7115459
38. Qiu M, Zhang Y, Long Z, He Y. Effect of protein-rich breakfast on subsequent energy intake and subjective appetite in children and adolescents: systematic review and meta–analysis of randomized controlled trials. Nutrients 2021;13:2840. https://doi.org/10.3390/nu13082840
39. Halliday TM, Liu SV, Moore LB, Hedrick VE, Davy BM. Adolescents perceive a low added sugar adequate fiber diet to be more satiating and equally palatable compared to a high added sugar low fiber diet in a randomized-crossover design controlled feeding pilot trial. Eat Behav 2018;30:9-15. https://doi.org/10.1016/j.eatbeh.2018.05.004
40. Forde CG, Mars M, de Graaf K. Ultra-processing or oral processing? A role for energy density and eating rate in moderating energy intake from processed foods. Curr Dev Nutr 2020;4:nzaa019. https://doi.org/10.1093/cdn/nzaa019
41. Hall KD, Ayuketah A, Brychta R, Cai H, Cassimatis T, Chen KY, et al. Ultra-processed diets cause excess calorie intake and weight gain: an inpatient randomized controlled trial of ad libitum food intake. Cell Metab 2019;30:67-77.e3. https://doi.org/10.1016/j.cmet.2019.05.008
42. Naude CE, Visser ME, Nguyen KA, Durao S, Schoonees A. Effects of total fat intake on bodyweight in children. Cochrane Database Syst Rev 2018;7:CD012960. https://doi.org/10.1002/14651858.CD012960.pub2

Table I. The 18-item questionnaire of adherence to the Mediterranean diet in children

Questions	Criterion for 1 point
1. Do you use extra-virgin olive oil as the main culinary fat?	Yes
2. Do you consume 3 or more tablespoons of olive oil per day (including that used for frying, salads or in meals away from home)? (1 tbsp=10 ml)	Yes
3. Do you consume 2 or more servings of vegetables per day? During the week, do you eat any of these servings as raw vegetables or salad? (1 serving= 50-80 g)	Yes
4. Do you consume 3 or more small fruits per day? (small fruit = 100 g)	Yes
5. Do you consume whole grains (bread, cereals, pasta, or rice) 3 or more times a week, instead of refined grains?	Yes
6. Do you consume at least 1 serving per day of fermented milk, plain yogurt or goat's or sheep's cheese? (1 commercial portion of fermented milk or yogurt or 25 g of cheese)	Yes
7. Do you consume 2-3 or more servings a week of legumes? (1 serving = 40 g raw weight)	Yes
8. Do you consume 3 or more servings a week of fish/seafood? (1 serving = 40-70 g)	Yes
9. Do you consume at least 3 servings a week of nuts? (1 serving = 15-20 g)	Yes
10. Do you consume preferably chicken, turkey, or rabbit instead of beef, pork, hamburgers, or sausage?	Yes
11. Do you consume 2 or more times a week cooked vegetables, pasta, rice, or other dishes seasoned with tomato, garlic, onion, or leek sauce simmered with olive oil (sofrito)?	Yes
12. Do you eat red meat, hamburgers, sausages, or derivative/processed meat products less than 2 times a week?	Yes
13. Do you eat less than 1 serving per day of butter or cream? (1 serving = 12 g)	Yes
14. Do you drink less than 2 glasses a week of carbonated and/or sugary beverages (sodas, coke, juices, nectars)?	Yes
15. Do you consume chips, gummy worms, sweets less than 1 time a week?	Yes
16. Do you consume dairy desserts, such as custard, ice cream, dairy smoothies, petit Suisse, vegetable drinks, etc, less than 1 time a week?	Yes
17. Do you consume pastries, stuffed cookies, sweets, or cakes less than 2 times a week?	Yes
18. Do you consume precooked or ready-to-eat foods less than 1 time a week?	Yes

Table II. Questionnaire for evaluating an active lifestyle in children

Questions	Criterion for 1 point
19. Does she/he walk or cycle to the school at least 3 days a week?	Yes
20. Does she/he play outdoors such as in parks or playgrounds, during leisure time, at least 3 days a week?	Yes
21. Does she/he participate in extracurricular sports or recreational activities like basketball, soccer, athletics, cycling, swimming, rhythmic gymnastics, other sports, traditional games, etc, at least 2 days a week?	Yes
22. Does she/he do strength and flexibility activities like dancing, yoga, or martial arts at least 4 days a week?	Yes
23. Does she/he preferentially use the stairs, such as at home?	Yes
24. Does she/he walk every day for at least 30 minutes, even if it is not continuous?	Yes
25. Does she/he participate in outdoors activities with the family during weekdays at least 1 time per week? (cycling, playing ball)	Yes
26. Does she/he participate in outdoors activities like family excursions during the weekends, at least 2 times per month?	Yes
27. During the leisure time, does she/he participate in activities that require more movement such as dancing, running, jumping, instead of activities like painting, sitting down to play, using the computer, etc?	Yes
28. Does she/he like the physical education classes at school?	Yes
29. During breaks at school, does she/he prefer activities like running, jumping or other activities instead to nonmoving games (talking, sitting down, playing with cars or dolls, etc)?	Yes
30. During weekdays, does she/he use audiovisual media, such as television, computers, tablets, or cell phones, less than 2 hours a day?	Yes
31. During weekends, does she/he use audiovisual media, such as television, computers, tablets, or cell phones, less than 2 hours a day?	Yes

Third article

"Adherence to a healthy lifestyle behavior composite score and cardiometabolic risk factors in the CORALS children cohort"

Tany E. Garcidueñas-Fimbres, Carlos Gómez-Martínez, Maria Pascual-Compte, Jose Manuel Jurado-Castro, Rosaura Leis, Luis A. Moreno, Santiago Navas-Carretero, Pilar Codoñer-Franch, Ana Moreira Echeverria, Belén Pastor-Villaescusa, Alicia López-Rubio, Sara Moroño García, Pilar De Miguel-Etayo, J. Alfredo Martínez, Inmaculada Velasco Aguayo, Rocío Vázquez-Cobela, Joaquín Escribano, María Luisa Miguel-Berges, María José De La Torre-Aguilar, Mercedes Gil-Campos, Jordi Salas-Salvadó, Nancy Babio on behalf of the CORALS study investigators.

- Publication phase: under review.
- Doctoral student's position: first author.

Summary of results

In the adjusted models, compared with the reference category of adherence to a healthy lifestyle behavior composite score, those participants in the 3rd tertile of adherence showed a decreased prevalence risk of overweight or obesity [OR, 0.4; 95% CI, 0.2, 0.6; P< 0.01] as well as lower waist circumference [β, -1.4 cm; 95% CI, -2.5, -0.4; P< 0.01), fat mass index [β, -0.3 kg/m2; 95% CI, -0.5, -0.1; P< 0.05], systolic blood pressure [β, -3.0 mmHg; 95% CI; -5.2, -0.9;

P< 0.01] and fasting plasma glucose concentration [β, -1.9 mg/dL; 95% CI, -3.5, -0.4; P< 0.05]. Slow eating category was one of the major contributors in these associations.

Conclusion

Higher adherence to a composite score comprised of the healthy behaviors of breastfeeding, sleep duration, physical activity, screentime, adherence to MedDiet and eating speed was associated with lower adiposity, systolic blood pressure and fasting plasma glucose concentration in a population of healthy preschool children. Slow eating speed appeared to be the healthy behavior with greater contribution to the individual associations with cardiometabolic risk factors, so it may be beneficial to consider this modifiable behavior in future strategies for reducing the risk of childhood obesity and adulthood diseases. Nevertheless, further long-term and interventional studies are required to confirm these associations.

ADHERENCE TO A HEALTHY LIFESTYLE BEHAVIOR COMPOSITE SCORE AND CARDIOMETABOLIC RISK FACTORS IN THE CORALS CHILDREN COHORT

Tany E. Garcidueñas-Fimbres[1,2,3], MD, MSc; Carlos Gómez-Martínez[1,2,3], MSc; Maria Pascual-Compte[2,3], MSc; Jose Manuel Jurado-Castro[4], PhD; Rosaura Leis[1,5,6,] MD, PhD; Luis A. Moreno[1,7,8], MD, PhD; Santiago Navas-Carretero[1,9,10], PhD; Pilar Codoñer-Franch[1,11], MD, PhD; Ana Moreira Echeverria[12], MD; Belén Pastor-Villaescusa[4], PhD; Alicia López-Rubio[5,6], MSc; Sara Moroño García[13], RN; Pilar De Miguel-Etayo[1,7,8], PhD; J. Alfredo Martínez[9], MD, PhD; Inmaculada Velasco Aguayo[4], PhD; Rocío Vázquez-Cobela[1,5,6], PhD; Joaquín Escribano[3,14], MD, PhD; María Luisa Miguel-Berges[1,7,8], PhD; María José De La Torre-Aguilar[4], MD, PhD; Mercedes Gil-Campos[1,4], MD, PhD; Jordi Salas-Salvadó[1,2,3*], MD, PhD; Nancy Babio[1,2,3*], PhD, on behalf of the CORALS study investigators.

Affiliations:

[1]Centro de Investigación Biomédica en Red de Fisiopatología de la Obesidad y Nutrición (CIBEROBN), Instituto de Salud Carlos III (ISCIII), Madrid, Spain.

[2]Universitat Rovira i Virgili, Departament de Bioquímica i Biotecnologia, Unitat de Nutrició Humana. Reus, Spain

[3]Institut d'Investigació Sanitària Pere Virgili (IISPV) Reus, Spain.

[4]Metabolism and Investigation Unit, Reina Sofia University Hospital. Maimónides Institute of Biomedicine Research of Córdoba (IMIBIC). University of Córdoba; Córdoba 14071, Spain.

[5]Unit of Pediatric Gastroenterology, Hepatology and Nutrition. Pediatric Service. Hospital Clínico Universitario de Santiago. 15706. Santiago de Compostela, Spain.

[6]Pediatric Nutrition Research Group, Health Research Institute of Santiago de Compostela (IDIS). Unit of Investigation in Nutrition, Growth and Human Development of Galicia-USC. 15706 Santiago de Compostela, Spain.

[7]Growth, Exercise, Nutrition and Development (GENUD) Research Group, University of Zaragoza, Zaragoza, Spain.

[8]Instituto Agroalimentario de Aragón (IA2). Instituto de Investigación Sanitaria de Aragón (IIS Aragón).

[9]University of Navarra, Center for Nutrition Research, 31008 Pamplona, Spain; University of Navarra, Fac Pharm & Nutr, Dept Nutr Food Sci & Physiol, 31008, Pamplona, Spain.

[10]IdisNA, Navarra Institute for Health Research, Pamplona, Spain.

[11]Dr. Peset University Hospital. Department of Pediatrics, Obstetrics and Gynecology, University of Valencia.

[12]Fundació Hospital Sant Joan de Deu Martorell.

[13]EDP Salut Sant Joan Baix Camp-ABS Riudoms.

[14]Paediatrics, Nutrition, and Development Research Unit, Universitat Rovira i Virgili.

*Corresponding authors: Nancy Babio [nancy.babio@urv.cat] and Jordi Salas-Salvadó [jordi.salas@urv.cat], Unitat de Nutrició Humana, Departament de Bioquímica i Biotecnología, Universitat Rovira i Virgili.

§ The CORALS study investigators are listed in the Supplementary material.

Short title:

Healthy lifestyle behaviors and cardiometabolic risk in children.

Funding

Supported by CONACYT (769789 to TEG-F), the University of Rovira i Virgili (2020PMF-PIPF-37 to CG-M). JS-S, senior author of this study, is partially supported by ICREA under the ICREA Academia program. Funds for the establishment of the CORALS cohort in the first year of the study (2019) were provided by an agreement between the Danone Institute from Spain and the CIBEROBN. Supported partially by the Generalitat Valenciana (PROMETEO 21/2021) and the Department of Education, Gobierno de Navarra (0000-RES1-2020-000338).

Conflict of interest:

The authors have declared no conflict of interest.

Data sharing statement

The datasets generated and analyzed during the current study are not publicly available due to data regulations and for ethical reasons, considering that this information might compromise research participants' consent because our participants only gave their consent for the use of their data by the original team of investigators. However, collaboration for data analyses can be requested by sending a letter to the CORALS steering Committee (estudiocoral@corals.es). The request would then be forwarded to all CORALS Steering Committee members for their deliberation.

ABSTRACT

Objective: To assess the associations between the adherence to a composite score comprised of 6 healthy lifestyle behaviors and several cardiometabolic risk factors in Spanish preschool children.

Methodology: Baseline cross-sectional analyses were conducted in the CORALS study cohort (n=1,371) aged 3-6 years. Six recognized healthy lifestyle behaviors (breastfeeding, sleep duration, physical activity, sedentary behaviors, adherence to the Mediterranean diet (MedDiet) and eating speed) were assessed in a composite score. Unadjusted and adjusted multiple linear and logistic regression models were fitted to assess the associations with certain cardiometabolic risk factors (weight status, waist circumference, fat mass index, blood pressure, fasting plasma glucose and lipid profile).

Results: In the adjusted models, compared with the reference category of adherence to a healthy lifestyle behavior composite score, those participants in the 3rd tertile of adherence showed a decreased prevalence risk of overweight or obesity [OR, 0.4; 95% CI, 0.2, 0.6; P< 0.01] as well as lower waist circumference [β, -1.4 cm; 95% CI, -2.5, -0.4; P< 0.01), fat mass index [β, -0.3 kg/m^2; 95% CI, -0.5, -0.1; P< 0.05], systolic blood pressure [β, -3.0 mmHg; 95% CI; -5.2, -0.9; P< 0.01] and fasting plasma glucose concentration [β, -1.9 mg/dL; 95% CI, -3.5, -0.4; P< 0.05]. Slow eating category was one of the major contributors in these associations.

Conclusions: Higher adherence to a healthy lifestyle behavior composite score was associated with lower adiposity and cardiometabolic risk in preschool children. Further long-term and interventional studies are required to confirm these associations.

Keywords: lifestyle behaviors, eating speed, childhood obesity, cardiometabolic risk, CORALS.

INTRODUCTION

Overweight and obesity in children is a global Public Health concern. The highest prevalence in Europe is observed in Mediterranean and Eastern European countries (1). The ALADINO 2019 study (2) reported, in Spanish children aged 6 to 9 years, a prevalence of overweight and obesity of 23.3% and 17.3% respectively. Furthermore, it is estimated that in 2030, around 9 million children of 5 to 9 years old will have obesity in Europe (3).

Obesity is the consequence of a complex bio-socioecological framework in which socio-political environment, individual factors, lifestyle behaviors, among others, interact (4,5). Childhood overweight and obesity has been associated with several long-term cardiometabolic disorders in adulthood (5). In this sense, consistently adiposity status from childhood to adulthood has been associated with higher risk of diabetes, hypertension, and lipid profile disorders (5).

Several modifiable lifestyle behaviors have been associated with obesity or cardiometabolic risk factors such as sedentary behaviors, lower levels of moderate-vigorous activity, unhealthy dietary patterns (including energy-dense and micronutrient-poor foods), reduced sleep duration, certain early life factors, among others (4). In recent decades, evidence has emerged on the associations between adherence to healthy lifestyle behaviors (e.g., diet, sleep, physical activity, screentime, etc.) and adiposity (6–9) or certain cardiometabolic disorders (7). However, none of these studies have assessed breastfeeding, eating speed and adherence to the MedDiet as components of the scores created. Adherence to the MedDiet (10,11) and eating speed (12,13) have been related with adiposity and cardiometabolic risk, however, evidence in children is very limited (18,19). On the other hand, the benefits from breastfeeding have been reported in early

life (14), and its duration was observed to be inversely associated with cardiometabolic risk (15) and weight gain (16) in childhood.

It is essential to identify other lifestyle behaviors that could be associated with adiposity and cardiometabolic disorders for a better understanding of the etiopathogenesis of childhood obesity, which would enable the design of more effective strategies to alleviate the consequences of this major public health concern. Hence, the aim of the present study was to assess cross-sectionally the relationships between adherence to a composite score comprised of 6 healthy lifestyle behaviors (breastfeeding, sleep duration, physical activity, sedentary behaviors, adherence to the MedDiet and eating speed) and its individual components with several cardiometabolic risk factors in children aged 3 to 6 years.

METHODS

1. Study design and participants

Cross-sectional analyses were conducted in baseline data from the first 1,371 participants recruited in the Childhood Obesity Risk Assessment Longitudinal Study (CORALS). This is a prospective ongoing multicenter study conducted in preschool children from 7 Spanish cities (Barcelona, Cordoba, Navarra, Reus, Santiago de Compostela, Valencia and Zaragoza) which aim is to identify and assess the risk factors that contribute to the development of obesity in childhood. Children were recruited between March 2019 and June 2021. A detailed description of the CORALS study can be found elsewhere (17), including recruitment details, eligibility and exclusion criteria.

For the present study, the following inclusion criteria were considered: a) having reported plausible energy intake on food frequency questionnaires, and b) to have available data on the duration of main meals (breakfast, lunch and dinner), physical activity,

breastfeeding, sleep duration, the 18-item questionnaire of adherence to the MedDiet and sedentary behaviors. Participants with current diagnosis of chronic diseases such as diabetes mellitus type 2, hypertension and familiar hypercholesterolemia were excluded from analyses.

2. Assessments

Parents or caregivers completed self-administered questionnaires assessing participant data: sociodemographic, dietary habits, lifestyle behaviors, early life and maternal factors, among others.

Exposure variables

Breastfeeding

The duration of total breastfeeding (months) and exclusive breastfeeding (yes/no) were assessed. Participants were categorized according to whether they were exclusively breastfed during the first 6 months of life (18).

Sleep duration

The sleep duration (hours/day) was evaluated through the question: "how long does your child sleep at night during weekdays and/on weekend days or holidays? And "how long does your child nap during weekdays and/on weekend days or holidays? Daily sleep duration was calculated (17) and categorized for age in "inadequate or could be adequate" (<10h/day or >13 h/day for children aged 3-5 years and <9 h/day or >11 h/day for children aged 6 years), or "adequate" (10-13h/day for children aged 3-5 years and 9-11h/day for children aged 6 years) according to the National Sleep Foundation's recommendations (19).

Sports and physical activities

A physical activity questionnaire was provided to parents or caregivers (17). The total time (hours) of sports and physical activities per week was estimated by summing the duration of physical education classes and extracurricular sports or physical activities. Then, two categories were created [<120 min/week (unhealthy behavior) or ≥120 min/week ("healthy behavior")] according to the WHO European Childhood Obesity Surveillance Initiative (20).

Screentime

Screentime for weekdays and weekend days was assessed by two questions: a) 'how long does your child watch television?' and b) 'how long does your child play in the computer/cell phone/game console?' Possible answers were "none", "0.5-1 h/day", "1-2h/day", "2-3 h/day", "3-4 h/day" or "more than 4 h/day". Total screentime was derived in a quantitative variable and <2 h/day was used as compliance with the recommendation (21).

Adherence to Mediterranean diet

An ad-hoc questionnaire adapted to children and comprised of 18 items (17) was used to evaluate the consumption of typical foods included in the MedDiet, as well as some foods not included in this dietary pattern (e.g., butter, cream, carbonated/sugary beverages, junk food, dairy desserts and pre-cooked/ready to eat food) that extends to certain foods commonly consumed by children (e.g., sweets, petit Suisse). The total score (0-18 points) was categorized by tertiles, in which the 3rd tertile represented the highest adherence to the MedDiet.

Eating speed

The duration of breakfast, lunch and dinner was assessed through the question "How long does it usually take for your child to eat?" Eating speed was estimated by summing the

total minutes required for eating the main meals and it was categorized by tertiles in fast, moderate, and slow eating categories. The slow eating category (3rd tertile) was considered the healthy lifestyle behavior.

Lifestyle behaviors assessment

Participants were categorized for each lifestyle behavior according to compliance (1 point) or not (0 points) with the following indicators: breastfeeding (<6 months=0 points; ≥6 months=1 point), sleep duration for age (inadequate/could be adequate=0 points; adequate=1 points), sports and physical activities (<2h/week=0 points; ≥2h/week=1 point) and screentime (<2h/day=1 point; ≥2h/day=0 points). Eating speed and adherence to the MedDiet were assessed by tertiles (T1=0 points, T2=0.5 points, T3=1 points) in which T3 corresponded to the slow eating category and the highest adherence to the MedDiet, respectively. Afterwards, the total score was calculated for each participant by summing the points obtained in each of the 6 healthy lifestyle behaviors (0-6 points). Hereafter, tertiles of combined adherence to these lifestyle behaviors were created (T1=lowest, T2=moderate and T3=highest).

Outcome variables

Adiposity

Weight and height were measured by trained registered dietitians. A precision scale (TANITA MS780SMA) was used to measure body weight (kg) and body fat mass (kg), by octopolar multifrequency bioelectrical impedance. The height and waist circumference (cm) were also evaluated by a portable stadiometer (SECA 213) and a measuring tape (SECA 201), respectively. Weight status was estimated according to body mass index (BMI) and categorized in underweight/normal weight or overweight/obesity according to

the cutoff points defined by Cole et al (22). The fat mass index (FMI) was calculated as body fat mass (kg)/squared height (23).

Cardiometabolic risk factors

Blood pressure was measured in non-dominant arm in triplicate at 5-minutes intervals. Total systolic and diastolic blood pressure values were estimated by the mean of all available data in both arms, if applicable.

Fasting plasma glucose and lipid profile (total cholesterol, high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), and triglycerides) were measured from blood samples collected in fasting conditions. The non-HDL-c was calculated (24).

Early life factors

Weight gain during pregnancy (kg) and birth weight (kg) were assessed. Birth weight was categorized in low (<2.5 kg), normal (2.5-3.9 kg), and high birth weight (>4 kg)(25).

Maternal factors

Maternal data was assessed, including educational level (primary or lower, secondary or academic-graduated or no reported) and socio-professional category (homemaker/student/retired/unemployed or employee). The BMI (kg/m^2) was estimated according to self-reported anthropometric measures and categorized according to WHO cut-offs (26). Then, it was re-categorized in underweight/normal weight (<25 kg/m^2) and overweight/obesity (≥25 kg/m^2).

Diet

A trained dietitian completed a semi-quantitative 125-item food frequency questionnaire (FFQ) for each participant, which was adapted from another FFQ validated in elderly

Spanish population (27). The consumption frequency options were as follows: never; 1-3 times per month; 1 time per week; 2-4 times per week; 5-6 times/week; 1 time per day; 2-3 times per day; 4-6 times per day and more than 6 times per day. The consumption of each food item in the FFQ was calculated by multiplying intake frequency by the portion specified in each case. The total energy and the intake of nutrients were estimated according to Spanish nutritional composition databases (28–30). Implausible energy intake was assessed according to Goldberg cut-offs adapted to children (31) and those participants with missing data or implausible reported energy intake were no included in final analyses.

3. Statistical analyses

CORALS database updated to December 2021 was used for the present cross-sectional study. One-factor ANOVA, Kruskal Wallis and chi-square tests were used to describe differences in baseline characteristics according to tertiles of adherence to the composite score comprised of 6 healthy lifestyle behaviors, which were reported as mean ± SD or median [interquartile range (IQR)] for continuous variables and as percentage (numbers) for categorical variables. Bonferroni's tests for multiple comparison were used in those statistically significant one-factor ANOVA analyses. Missing data <10% on covariates data was imputed to the mean (32). Multiple linear regression models [β; 95% confidence interval (CI)] were fitted to assess associations between tertiles of adherence to the composite score comprised of 6 healthy lifestyle behaviors (exposure), and outcome variables (waist circumference in cm, FMI in kg/m^2, systolic and diastolic blood pressure in mmHg, and lipid profile in mg/dL), except for weight status, for which logistic regression models [odds ratio (OR), 95% confidence interval] were fitted. Models were adjusted by recruitment center, according to the number of recruited participants in each center (<130, 130-200, >200), sex, age, mother's educational level (primary or lower,

secondary, academic-graduate or no reported data), maternal overweight/obesity status (yes/no) and birth weight category (low, normal, high). Individual associations between each lifestyle behavior and each outcome were also assessed by logistic or linear regression models to assess their contribution on the associations between the composite score comprised of 6 healthy lifestyle behaviors and outcomes. Each of these models were further adjusted by the other lifestyle behaviors, except for the one of interest. For all models, the reference category was the 1st tertile. Interaction analyses were conducted by sex. Due to the lack of validation of bioimpedance equations (TANITA MC780SMA) in children under 5 year-old, participants aged 3-4 year-old were excluded as sensitivity analyses for FMI. All statistical analyses were performed using Stata 14 software program (StataCorp), and statistical significance was set at a two-tailed p-value < 0.05.

RESULTS

A total of 1,371 participants attended the baseline visit at CORALS study, of which 49 participants were excluded from analyses for not meeting eligibility and inclusion criteria as well as 216 participants for having missing data or implausible reported energy intake. In addition, 139 participants were no longer included due to missing data on ≥1 variables required to estimate eating speed (n=119), adherence to the MedDiet (n=5) as well as total duration of physical activity (n=6) or daily sleep duration (n=9). Finally, 29 participants with diagnosis of chronic diseases were excluded. Hence, the final analyses included 938 children of which 49% were girls (**Figure 1**, online). Questionnaires were filled out by mothers who were main caregivers in the 88% of participants.

Participants had a mean age ± SD of 4.8 ± 1 year-old and showed a 14% and 8% prevalence of overweight and obesity, respectively. The total study population showed a mean of 6.8 ± 9 months of breastfeeding, 10.4 ± 1 h/day of sleep duration, 185 ± 115 mins/week of physical activity, 1.8 ± 1 h/day of screentime, 11 ± 3 points on the 18-item

MedDiet adherence questionnaire (T1= 8 ± 1 points; T2= 11 ± 1 points; T3=14 ± 1 points) and 78.1 ± 25 min/day of eating speed in the 3 main meals (T1= 53.8 min/day ± 9; T2= 76.3 ± 5 min/day; T3=108.3 ± 18 min/day). The general characteristics in the participants according to the categories of adherence to the composite score comprised of 6 healthy lifestyle behaviors are shown in **Table 1**. Mothers whose participants were in the 3rd tertile of adherence to the composite score comprised of 6 healthy lifestyle behaviors were more likely to have higher educational level and lower prevalence of overweight or obesity (all $p<0.05$). **Table 2** shows the lifestyle, adiposity and cardiometabolic risk characteristics in participants across the categories of adherence to the composite score. Compared to participants in the low category of adherence, those in the highest tertile of adherence were more likely to be breastfed for longer, to sleep and exercise more time, to have higher adherence to the MedDiet and to eat slower. Compared to the reference category of adherence to the composite score comprised of 6 healthy lifestyle behaviors, those children in the top category of adherence had a lower prevalence of overweight or obesity and values for fat mass index, systolic and diastolic blood pressure (all $p<0.05$).

Table 3 (online) summarizes the dietary characteristics across tertiles of adherence to the composite score comprised of 6 healthy lifestyle behaviors. Compared to participants in the reference category of adherence, participants in the 3rd tertile had lower intakes of total energy, carbohydrates, sodium and lower consumption of other dairy products, processed and derivatives meat products, tubers, pastries, sugar, candies, and sugary beverages (all $p<0.05$). Children in the highest category of adherence to the composite score comprised of 6 healthy lifestyle behaviors also reported higher intakes of protein, monounsaturated fatty acids, fiber, cheese, fish, seafood, vegetables, fruits, nuts and whole grains (all $p<0.05$).

The associations between the adherence to the composite score and several cardiometabolic risk factors are shown in **Table 4**. In unadjusted models, compared to children allocated in the lowest adherence category (<3 points), those in the 3rd tertile (>4 points) of adherence to the composite score were associated with a 60% lower risk of overweight or obesity prevalence, lower waist circumference, FMI and systolic and diastolic blood pressure. In adjusted models these associations remained, except for diastolic blood pressure. In addition, an inverse association was also observed between the highest tertile of adherence to the composite score and fasting plasma glucose concentration in the adjusted model [β coefficient (95%CI): -1.9 (-3.5, -0.4); p=0.013]. Interaction analyses between categories of adherence to the composite score and sex were not statistically significant. In sensitivity analyses, for which children aged under 5 years were excluded, the association between adherence to the composite score and FMI was not significant, but the negative direction remained.

The individual associations between each of the 6 healthy lifestyle behaviors and each outcome are shown in **Table 5** (online). An adequate sleep duration for age, screentime for <2 h/day and slow eating (>85 minutes in the 3 main meals) were associated with a lower prevalence risk of overweight or obesity (**Figure 2**). Screentime for <2 h/day and slow eating were associated with lower waist circumference and FMI. Exclusive breastfeeding to the first 6 months of age and slow eating were associated with lower systolic blood pressure. Physical activity for ≥2h/week was associated with decreased values of diastolic blood pressure and LDL-c levels. Slow eating was associated with lower fasting plasma glucose concentration. Higher adherence to the MedDiet (≥ 13 points) was inversely associated with serum triglyceride levels. Children whose parents or caregivers reported screentime for <2 h/day showed a higher total cholesterol and non-HDL-c. Slow eating was associated with lower OR for overweight/obesity, and β-

coefficients for FMI and systolic blood pressure, respectively. When these models were further adjusted by the other lifestyle behaviors except for the one of interest, most of the associations remained.

DISCUSSION

In the present study, higher adherence to a composite score comprised of the 6 healthy behaviors (breastfeeding, sleep duration, physical activity, screentime, eating speed and adherence to the MedDiet) was associated with lower adiposity and reduced levels of systolic blood pressure and fasting plasma glucose. No significant associations were observed for the remainder cardiometabolic risk factors. When assessing the individual associations among each of the healthy lifestyle behaviors and the cardiometabolic risk factors, eating speed showed to be one of the major contributors associated with lower adiposity (weight status, waist circumference and FMI) and was the only lifestyle behavior associated with lower fasting plasma glucose concentration. Additionally, those participants allocated in the highest tertile of adherence to the composite score comprised of 6 healthy lifestyle behaviors showed a lower energy intake and a dietary pattern closer to the MedDiet. Besides, higher educational level, lower BMI and overweight/obesity status was observed among the mothers of those participants allocated in the 3rd tertile of adherence to the composite score comprised of 6 healthy lifestyle behaviors.

Some observational studies (6–8) but not all (7,9) are in line with our results, as they reported associations between higher adherence to a healthy lifestyle pattern and lower adiposity in children of similar age (7). Discrepancies between studies could be partially explained by differences in the lifestyle behaviors assessment, although authors mainly assessed physical activity (7–9), sleep duration (7–9), sedentary behaviors (7–9), dietary components (6,7) and/or stress-related behaviors (8). Bawaked et al (7) conducted cross-sectional and longitudinal analyses in Spanish children in which a 5-item score was

created and categorized assigning a different punctuation to each tertile based on whether the behavior was favorable (extracurricular physical activity, sleep duration and plant-based food consumption) or unfavorable (screentime and consumption of ultra-processed food). In contrast with our results, in this study (7) no significant associations were reported for blood pressure, HDL-c or serum triglyceride levels, and fasting plasma glucose concentration was not assessed. Moreover, Bawaked et al (7) did not evaluate lifestyle behaviors based on their recommendations, which might differ according to age (e.g., sleep duration) and therefore, using an alternative method to assess these variables could lead to different results. In addition, a high prevalence of children with overweight or obesity was reported also in this study (7), which may not enable to discriminate appropriately the compliance with certain healthy lifestyle behaviors. On the other hand, some studies assessed components of the diet but not the whole dietary pattern. For example, Manios et al (6) assessed the eating frequency of certain foods (fruits, vegetables, total grains, dairy products, red meat, white meat/legumes, fish and seafood, unsaturated fats and sweets). Similarly, Kovács et al (8) explored the consumption of sweetened beverages and fruits/vegetables. However, in contrast with our study, none of these authors assessed eating speed, adherence to the MedDiet or breastfeeding as lifestyle behaviors potentially associated with adiposity and/or cardiometabolic risk factors.

Certain observational studies have also explored associations between physical activity and adiposity (7,8,33) or cardiometabolic risk factors (7,34,35). In line with our results, Väistö et al (34) reported, in children of 6-8 years of age, a negative association between physical activity and diastolic blood pressure and LDL-c. Moreover, additional studies conducted in European children (7,8,33) also reported significant associations between increased physical activity and lower adiposity. Additionally, in a Greek population of

children (35), lower triglyceride levels were observed in those children who exercised longer than 2 h/week. Nevertheless, only a few authors (8,33) assessed physical activity according to compliance with recommendations in early life and its association with certain cardiometabolic risk factors.

The evidence on associations between sleep duration and adiposity or cardiometabolic disorders in children is inconsistent. Yet, certain observational studies (7,33,36) are in line with our results as they also observed inverse associations between sleep duration and adiposity in European children and adolescents. In contrast, no significant associations with adiposity were reported in children from 8 European countries (8), while Bawaked et al (7) reported an inverse association between sleep duration at 4 year-old and systolic blood pressure at 7 years of age. Potential effects from sleep duration on energy expenditure (37) and nervous system (38) have been suggested, which may act as mechanisms in the regulation of adiposity and blood pressure, respectively.

The present study showed consistent results with previous observational studies conducted in preschool (7,8) and school children (8) as they observed inverse associations between high screentime and adiposity. In contrast, a meta-analysis (39) from observational and interventional studies reported small to very small evidence between sedentary behaviors and adiposity in children and adolescents. However, according to a systematic review (40), longer screen time could limit the time spent on physical activities, reinforcing sedentary behaviors which may explain adiposity outcomes. Additional studies with objective assessments of sedentary behaviors and further clinical trials are required to confirm these relationships. In the present study we also observed unexpected directions in the associations between healthy behavior for screentime and total cholesterol or non-HDL-c. In this regard, a cross-sectional study (41) conducted in Australian school children reported no significant associations between certain sedentary

behaviors and total cholesterol serum concentration. However, this study was conducted in a small sample of children, and total screentime was not assessed according to recommendations.

Regarding associations between eating speed and cardiometabolic risk factors, scarce studies have been conducted in children (42–46). In the present study we observed inverse associations between slow eating category and adiposity, systolic blood pressure and fasting plasma glucose concentration. Similar associations for adiposity were reported in a Finnish cross-sectional study (42) and an American clinical trial (43) but other cardiometabolic risk factors were no assessed. However, fast eating speed was recently positively associated with adiposity, systolic blood pressure and fasting plasma glucose in Spanish preschool children (17). Similarly, in an Asian population of children, Yamagishi et al (45) observed a positive association between eating speed and systolic blood pressure at 12 year-old but only in boys. Regarding the impact of eating speed on adiposity outcomes, we cannot discard the influence of diet quality since it has been suggested that dietary energy density and eating speed could regulate energy intake (47).

Although several prospective studies in adults have demonstrated an inverse association between adherence to the MedDiet and adiposity (48) or cardiometabolic risk factors (49–52), only few studies have assessed these relationships in children (35,53). In the present study, MedDiet was inversely related with serum triglyceride levels which is in line with previous studies (49,54) in adults. Furthermore, this association could be explained by some characteristics of the MedDiet (49) which is rich in fish, nuts, olive-oil, legumes as well as other plant-based foods (55) and low in refined cereals and sugar drinks.

Some results of the present article are also in line with a meta-analysis (56) which have shown that exclusive and long-term breastfeeding are associated with reduced risk of obesity in children. In contrast, a clinical trial (57) conducted in Belarussian infants,

aimed to achieve higher rates of exclusive and prolonged breastfeeding and reported that participants in the intervention group increased the duration and exclusivity of breastfeeding but showed no inverse effects for either low blood pressure or risk of obesity at 16 years of age. However, regarding the relationship between exclusive breastfeeding and overall cardiometabolic risk in children, it exists insufficient solid evidence (58) and therefore further studies are warranted.

The 'a priori' composite score comprised of 6 healthy lifestyle behaviors has been developed based on current evidence on diet and other lifestyle behaviors. However, possible interactions among the lifestyle behaviors assessed in the present study cannot be disregarded. For example, short sleep duration and high screentime or non-compliance with physical activity recommendations have been observed in association with higher adiposity in children (33). Additionally, an interaction between diet quality and physical activity was also reported (33), for which an unhealthy diet showed a significant impact according to the level of physical activity. Furthermore, additional putative lifestyle behaviors might have coexisted in participants and partially explain the results observed.

If further research could confirm the associations observed in the present article, this composite score comprised of 6 healthy lifestyle behaviors could become, in clinical practice, a useful tool that used as a simple checklist may contribute to the improvement of the prevention of adiposity and cardiometabolic disorders by early detection of lifestyle behaviors in childhood. Nevertheless, further research should be focused on more objective assessments of certain lifestyle behaviors such as direct visualization for eating speed and accelerometry for physical activity. Also, rigorous long-term and interventional studies should be conducted in children from different cultural contexts and may be extended to school children and adolescents.

The present study has some limitations that deserve to be mentioned: a) this is a cross-sectional study, so cause-effect conclusions should not be made; b) the studied population corresponded to Spanish preschool children and therefore the findings cannot be extrapolated to other populations; c) associations with residual confounding or undetected cardiometabolic disorders due to early age in the studied population cannot be dismissed; d) equations for bioelectrical bioimpedance have not been validated in children under 5 years of age, however, associations for FMI in sensitivity analyses, although not significant, showed the same direction that in the total population; e) physical activity was assessed by questionnaires which are limited in the identification of moderate-vigorous physical activities and for this reason we have not used the recommendation of 60 min/day but the cutoff reported in the WHO European Childhood Obesity Surveillance Initiative (20). In addition, some strengths also deserve to be mentioned. First, the studied population was comprised by a large sample size of the CORALS cohort from 7 Spanish centers, drew from general population. Second, data for lipids profile and fasting plasma glucose were available for the total studied population. Third, several measures were used to assess adiposity and cardiometabolic risk.

CONCLUSIONS

Higher adherence to a composite score comprised of the healthy behaviors for breastfeeding, sleep duration, physical activity, screentime, adherence to the MedDiet and eating speed was associated with lower adiposity, systolic blood pressure and fasting plasma glucose concentration in a population of healthy preschool children. Slow eating speed appeared to be the healthy behavior with greater contribution in the individual associations with adiposity and cardiometabolic risk factors, so it may be beneficial to consider this modifiable behavior in future strategies for reducing the risk of childhood

obesity and its comorbidities. Nevertheless, further long-term and interventional studies are required to confirm these associations.

ACKNOWLEDGMENTS

The authors would especially like to thank all the CORALS participants and their parents or caregivers as well as to the Health Centers and Primary Schools for their collaboration, to the CORALS personnel for their outstanding support, and to the staff of all associated primary care centers for their exceptional work. We want to particularly acknowledge the IISPV Biobank (PT20/00197) which is integrated in the ISCIII Platform for Biobanks and Biomodel.

ABBREVIATIONS

CORALS: Childhood Obesity Risk Assessment Longitudinal Study.

β: Beta coefficient.

CI: Confidence interval.

WHO: World Health Organization.

ALADINO: ALimentación, Actividad Física, Desarrollo INfantil y Obesidad.

MedDiet: Mediterranean diet.

FMI: Fat mass index.

HDL-c: High-density lipoprotein cholesterol.

LDL-c: Low-density lipoprotein cholesterol.

FFQ: Food frequency questionnaire.

CESNID: Centre d'Enseyament Superior de Nutrició i Dietètica.

IQR: Interquartile ratio.

OR: Odds ratio.

BMI: Body mass index.

ISCIII: Instituto Salud Carlos III

CONACYT: Consejo Nacional de Ciencia y Tecnología

ICREA: Institución Catalana de Investigación y Estudios Avanzados

CIBEROBN: Centro de Investigación Biomédica en Red de Fisiopatología de la Obesidad y Nutrición.

REFERENCES

1. WHO Regional office for Europe. WHO European Regional Obesity Report 2022. Copenhagen; 2022.

2. Agencia Española de Seguridad Alimentaria y Nutrición. Surveillance study on nutrition, physical activity, child development and obesity (ALADINO) [Internet]. 2020 Sep. Available from: https://www.aesan.gob.es/AECOSAN/docs/documentos/nutricion/observatorio/Brief_report_ALADINO_2019_NAOS.pdf

3. Lobstein T, Brinsden H, Neveux M, Barata Cavalcanti O, Barquera S, Baur L, et al. World Obesity Atlas 2022 [Internet]. 2022. Available from: https://www.worldobesity.org/resources/resource-library/world-obesity-atlas-2022

4. Jebeile H, Kelly AS, O'Malley G, Baur LA. Obesity in children and adolescents: epidemiology, causes, assessment, and management. Lancet Diabetes Endocrinol [Internet]. 2022 Mar; Available from: https://linkinghub.elsevier.com/retrieve/pii/S221385872200047X

5. Juonala M, Magnussen CG, Berenson GS, Venn A, Burns TL, Sabin MA, et al. Childhood Adiposity, Adult Adiposity, and Cardiovascular Risk Factors. New England Journal of Medicine [Internet]. 2011 Nov 17 [cited 2022 Aug 1];365(20):1876–85. Available from: http://www.nejm.org/doi/abs/10.1056/NEJMoa1010112

6. Manios Y, Kourlaba G, Grammatikaki E, Androutsos O, Moschonis G, Roma-Giannikou E. Development of a diet-lifestyle quality index for young children and its relation to obesity: the Preschoolers Diet-Lifestyle Index. Public Health Nutr [Internet]. 2010 Dec;13(12):2000–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/20409357

7. Bawaked RA, Fernández-Barrés S, Navarrete-Muñoz EM, González-Palacios S, Guxens M, Irizar A, et al. Impact of lifestyle behaviors in early childhood on obesity and cardiometabolic risk in children: Results from the Spanish INMA birth cohort study. Pediatr Obes [Internet]. 2020 Mar 2;15(3):e12590. Available from: https://onlinelibrary.wiley.com/doi/10.1111/ijpo.12590

8. Kovács E, Hunsberger M, Reisch L, Gwozdz W, Eiben G, de Bourdeaudhuij I, et al. Adherence to combined lifestyle factors and their contribution to obesity in the IDEFICS study. Obesity Reviews [Internet]. 2015 Dec 1 [cited 2021 Oct 20];16:138–50. Available from: https://onlinelibrary.wiley.com/doi/10.1111/obr.12349

9. Carson V, Tremblay MS, Chastin SFM. Cross-sectional associations between sleep duration, sedentary time, physical activity, and adiposity indicators among Canadian preschool-aged children using compositional analyses. BMC Public Health [Internet]. 2017 Nov 20;17(S5):848. Available from: https://bmcpublichealth.biomedcentral.com/articles/10.1186/s12889-017-4852-0

10. Kanellopoulou A, Giannakopoulou SP, Notara V, Antonogeorgos G, Rojas-Gil AP, Kornilaki EN, et al. The association between adherence to the Mediterranean diet and childhood obesity; the role of family structure: Results from an epidemiological study in 1728 Greek students. Nutr Health [Internet]. 2021 Mar 1 [cited 2022 May 2];27(1):39–47. Available from: https://journals.sagepub.com/doi/10.1177/0260106020952600

11. Seral-Cortes M, Larruy-García A, de Miguel-Etayo P, Labayen I, Moreno LA. Mediterranean Diet and Genetic Determinants of Obesity and Metabolic Syndrome in European Children and Adolescents. Genes (Basel) [Internet]. 2022 Feb 25;13(3):420. Available from: https://www.mdpi.com/2073-4425/13/3/420

12. Garcidueñas-Fimbres T, Paz-Graniel I, Nishi S, Salas-Salvadó J, Babio N. Eating Speed, Eating Frequency, and Their Relationships with Diet Quality, Adiposity, and

Metabolic Syndrome, or Its Components. Nutrients [Internet]. 2021 May 15;13(5):1687. Available from: https://www.mdpi.com/2072-6643/13/5/1687

13. Paz-Graniel I, Babio N, Mendez I, Salas-Salvadó J. Association between Eating Speed and Classical Cardiovascular Risk Factors: A Cross-Sectional Study. Nutrients [Internet]. 2019 Jan 4;11(1):83. Available from: https://www.mdpi.com/2072-6643/11/1/83

14. Patro-Gołąb B, Zalewski BM, Polaczek A, Szajewska H. Duration of Breastfeeding and Early Growth: A Systematic Review of Current Evidence. Breastfeeding Medicine [Internet]. 2019 May 1;14(4):218–29. Available from: https://www.liebertpub.com/doi/10.1089/bfm.2018.0187

15. Wong PD, Anderson LN, Dai DDW, Parkin PC, Maguire JL, Birken CS, et al. The Association of Breastfeeding Duration and Early Childhood Cardiometabolic Risk. J Pediatr [Internet]. 2018 Jan [cited 2021 Aug 26];192:80-85.e1. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0022347617313252

16. Silveira JAC, Colugnati FAB, Poblacion AP, Taddei JAAC. The role of exclusive breastfeeding and sugar-sweetened beverage consumption on preschool children's weight gain. Pediatr Obes [Internet]. 2015 Apr;10(2):91–7. Available from: https://onlinelibrary.wiley.com/doi/10.1111/ijpo.236

17. Garcidueñas-Fimbres TE, Paz-Graniel I, Gómez-Martínez C, Jurado-Castro JM, Leis R, Escribano J, et al. Associations Between Eating Speed, Diet Quality, Adiposity, and Cardiometabolic Risk Factors. J Pediatr [Internet]. 2023 Jan [cited 2022 Sep 26];252:31-39.e1. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0022347622007594

18. World Health Organization. Infant and young child feeding. https://www.who.int/es/news-room/fact-sheets/detail/infant-and-young-child-feeding. 2021.

19. Hirshkowitz M, Whiton K, Albert SM, Alessi C, Bruni O, DonCarlos L, et al. National Sleep Foundation's sleep time duration recommendations: methodology and results summary. Sleep Health [Internet]. 2015 Mar;1(1):40–3. Available from: https://linkinghub.elsevier.com/retrieve/pii/S2352721815000157

20. World Health Organization. Regional Office for Europe. WHO European Childhood Obesity Surveillance Initiative (COSI) Report on the fourth round of data collection [Internet]. 2021. Available from: https://apps.who.int/iris/handle/10665/341189

21. Bar-On ME, Broughton DD, Buttross S, Corrigan S, Gedissman A, González De Rivas MR, et al. Children, Adolescents, and Television. Pediatrics [Internet]. 2001 Feb 1 [cited 2022 Sep 22];107(2):423–6. Available from: https://publications.aap.org/pediatrics/article/107/2/423/66273/Children-Adolescents-and-Television

22. Cole TJ, Lobstein T. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. Pediatr Obes [Internet]. 2012 Aug;7(4):284–94. Available from: https://onlinelibrary.wiley.com/doi/10.1111/j.2047-6310.2012.00064.x

23. VanItallie TB, Yang MU, Heymsfield SB, Funk RC, Boileau RA. Height-normalized indices of the body's fat-free mass and fat mass: potentially useful indicators of nutritional status. Am J Clin Nutr [Internet]. 1990 Dec 1;52(6):953–9. Available from: https://academic.oup.com/ajcn/article/52/6/953/4651256

24. Frost PH, Havel RJ. Rationale for Use of Non–High-Density Lipoprotein Cholesterol Rather Than Low-Density Lipoprotein Cholesterol as a Tool for Lipoprotein Cholesterol Screening and Assessment of Risk and Therapy. Am J Cardiol [Internet]. 1998 Feb [cited 2021 Nov 16];81(4):26B-31B. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0002914998000344

25. Yerushalmy J. The classification of newborn infants by birth weight and gestational age. J Pediatr [Internet]. 1967 Aug;71(2):164–72. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0022347667800672

26. World Health Organ Tech Rep Ser. Obesity: Preventing and Managing the Global Epidemic: report of a WHO consultation [Internet]. 2000. 1–253 p. Available from: https://apps.who.int/iris/handle/10665/42330

27. Fernández-Ballart JD, Piñol JL, Zazpe I, Corella D, Carrasco P, Toledo E, et al. Relative validity of a semi-quantitative food-frequency questionnaire in an elderly Mediterranean population of Spain. British Journal of Nutrition [Internet]. 2010 Jun 28 [cited 2023 Mar 15];103(12):1808–16. Available from: https://www.cambridge.org/core/product/identifier/S0007114509993837/type/journal_article

28. Farran A, Zamora R, Cervera P, Centre d'Enseyament Superior de Nutrició i Dietètica. Tablas de composición de alimentos del CESNID. 3rd ed. Mc- Graw-Hill / Interamericana, Edicions Universitat de Barcelona, editors. Vol. 78. 2004. 1–247 p.

29. Babio Sánchez N, de la Serra Besora Moreno M, Lladó Bellette N, Salas Salvadó Jordi. CELIACBASE. Tabla de composición de productos sin gluten . Publicacions URV; 2018. 1–152 p.

30. Babio Nancy, Dragusan Lica Natalia, De Las Heras Sara, Blanquer Maria. Tabla de Composición de Productos y Alternativas Vegetales. 1st ed. Reus: Publicacions URV; 2022. 1–388 p.

31. Börnhorst C, Huybrechts I, Ahrens W, Eiben G, Michels N, Pala V, et al. Prevalence and determinants of misreporting among European children in proxy-reported 24 h dietary recalls. British Journal of Nutrition [Internet]. 2013 Apr 14 [cited 2022 Mar 7];109(7):1257–65. Available from:

https://www.cambridge.org/core/product/identifier/S0007114512003194/type/journal_article

32. Ton de Waal, Jeroen Pannekoek, Sander Scholtus. Statistical Data Editing and Imputation. John Wiley & Sons, editor. Vol. 53. 2011.

33. Wilkie HJ, Standage M, Gillison FB, Cumming SP, Katzmarzyk PT. Multiple lifestyle behaviours and overweight and obesity among children aged 9–11 years: results from the UK site of the International Study of Childhood Obesity, Lifestyle and the Environment. BMJ Open [Internet]. 2016 Feb 1 [cited 2021 Oct 20];6(2):e010677. Available from: https://bmjopen.bmj.com/content/6/2/e010677

34. Väistö J, Eloranta AM, Viitasalo A, Tompuri T, Lintu N, Karjalainen P, et al. Physical activity and sedentary behaviour in relation to cardiometabolic risk in children: cross-sectional findings from the Physical Activity and Nutrition in Children (PANIC) Study. International Journal of Behavioral Nutrition and Physical Activity [Internet]. 2014 Dec 26 [cited 2022 Aug 5];11(1):55. Available from: https://ijbnpa.biomedcentral.com/articles/10.1186/1479-5868-11-55

35. Lampropoulou M, Chaini M, Rigopoulos N, Evangeliou A, Papadopoulou-Legbelou K, Koutelidakis AE. Association between Serum Lipid Levels in Greek Children with Dyslipidemia and Mediterranean Diet Adherence, Dietary Habits, Lifestyle and Family Socioeconomic Factors. Nutrients 2020, Vol 12, Page 1600 [Internet]. 2020 May 29 [cited 2021 Oct 25];12(6):1600. Available from: https://www.mdpi.com/2072-6643/12/6/1600/htm

36. Hense S, Pohlabeln H, de Henauw S, Eiben G, Molnar D, Moreno LA, et al. Sleep Duration and Overweight in European Children: Is the Association Modified by Geographic Region? Sleep [Internet]. 2011 Jul 1 [cited 2022 Sep 21];34(7):885. Available from: https://academic.oup.com/sleep/article/34/7/885/2596044/Sleep-Duration-and-Overweight-in-European-Children

37. Benedict C, Hallschmid M, Lassen A, Mahnke C, Schultes B, Schiöth HB, et al. Acute sleep deprivation reduces energy expenditure in healthy men. Am J Clin Nutr [Internet]. 2011 Jun 1 [cited 2022 Jul 13];93(6):1229–36. Available from: https://academic.oup.com/ajcn/article/93/6/1229/4597726

38. DelRosso LM, Mogavero MP, Ferri R. Effect of Sleep Disorders on Blood Pressure and Hypertension in Children. Curr Hypertens Rep [Internet]. 2020 Nov 7;22(11):88. Available from: https://link.springer.com/10.1007/s11906-020-01100-x

39. Biddle SJH, García Bengoechea E, Wiesner G. Sedentary behaviour and adiposity in youth: a systematic review of reviews and analysis of causality. International Journal of Behavioral Nutrition and Physical Activity [Internet]. 2017 Dec 28 [cited 2020 Apr 10];14(1):43. Available from: http://ijbnpa.biomedcentral.com/articles/10.1186/s12966-017-0497-8

40. Carson V, Hunter S, Kuzik N, Gray CE, Poitras VJ, Chaput JP, et al. Systematic review of sedentary behaviour and health indicators in school-aged children and youth: an update. Applied Physiology, Nutrition, and Metabolism [Internet]. 2016 Jun;41(6 (Suppl. 3)):S240–65. Available from: http://www.nrcresearchpress.com/doi/10.1139/apnm-2015-0630

41. Robinson S, Daly RM, Ridgers ND, Salmon J. Screen-Based Behaviors of Children and Cardiovascular Risk Factors. J Pediatr [Internet]. 2015 Dec 1 [cited 2022 Aug 8];167(6):1239–45. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0022347615010008

42. Eloranta AM, Lindi V, Schwab U, Tompuri T, Kiiskinen S, Lakka HM, et al. Dietary factors associated with overweight and body adiposity in Finnish children aged 6–8 years: the PANIC Study. Int J Obes [Internet]. 2012 Jul 5;36(7):950–5. Available from: http://www.nature.com/articles/ijo201289

43. Faith MS, Diewald LK, Crabbe S, Burgess B, Berkowitz RI. Reduced Eating Pace (RePace) Behavioral Intervention for Children Prone to or with Obesity: Does the Turtle Win the Race? Obesity [Internet]. 2019 Jan 4 [cited 2020 Jul 20];27(1):121–9. Available from: https://onlinelibrary.wiley.com/doi/10.1002/oby.22329

44. Okubo H, Murakami K, Masayasu S, Sasaki S. The Relationship of Eating Rate and Degree of Chewing to Body Weight Status among Preschool Children in Japan: A Nationwide Cross-Sectional Study. Nutrients [Internet]. 2018 Dec 29;11(1):64. Available from: http://www.mdpi.com/2072-6643/11/1/64

45. Yamagishi K, Sairenchi T, Sawada N, Sunou K, Sata M, Murai U, et al. Impact of Speed-Eating Habit on Subsequent Body Mass Index and Blood Pressure Among Schoolchildren ― The Ibaraki Children's Cohort Study (IBACHIL) ―. Circulation Journal [Internet]. 2018 [cited 2021 Aug 19];82(2):419–22. Available from: https://www.jstage.jst.go.jp/article/circj/82/2/82_CJ-17-0287/_article

46. Okubo H, Miyake Y, Sasaki S, Tanaka K, Hirota Y. Rate of eating in early life is positively associated with current and later body mass index among young Japanese children: the Osaka Maternal and Child Health Study. Nutrition Research [Internet]. 2017 Jan;37:20–8. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0271531716304006

47. van den Boer J, Werts M, Siebelink E, de Graaf C, Mars M. The Availability of Slow and Fast Calories in the Dutch Diet: The Current Situation and Opportunities for Interventions. Foods [Internet]. 2017 Oct 2 [cited 2021 Aug 19];6(10):87. Available from: http://www.mdpi.com/2304-8158/6/10/87

48. Lotfi K, Saneei P, Hajhashemy Z, Esmaillzadeh A. Adherence to the Mediterranean Diet, Five-Year Weight Change, and Risk of Overweight and Obesity: A Systematic Review and Dose–Response Meta-Analysis of Prospective Cohort Studies.

Advances in Nutrition [Internet]. 2022 Feb 1 [cited 2022 Oct 9];13(1):152–66. Available from: https://academic.oup.com/advances/article/13/1/152/6342524

49. Bakaloudi DR, Chrysoula L, Kotzakioulafi E, Theodoridis X, Chourdakis M. Impact of the Level of Adherence to Mediterranean Diet on the Parameters of Metabolic Syndrome: A Systematic Review and Meta-Analysis of Observational Studies. Nutrients [Internet]. 2021 Apr 30 [cited 2022 Oct 10];13(5). Available from: http://www.ncbi.nlm.nih.gov/pubmed/33946280

50. Guasch-Ferré M, Willett WC. The Mediterranean diet and health: a comprehensive overview. J Intern Med [Internet]. 2021 Sep 23 [cited 2022 Oct 9];290(3):549–66. Available from: https://onlinelibrary.wiley.com/doi/10.1111/joim.13333

51. Nissensohn M, Román-Viñas B, Sánchez-Villegas A, Piscopo S, Serra-Majem L. The Effect of the Mediterranean Diet on Hypertension: A Systematic Review and Meta-Analysis. J Nutr Educ Behav [Internet]. 2016 Jan [cited 2022 Oct 9];48(1):42-53.e1. Available from: https://linkinghub.elsevier.com/retrieve/pii/S1499404615006673

52. Sarsangi P, Salehi-Abargouei A, Ebrahimpour-Koujan S, Esmaillzadeh A. Association between Adherence to the Mediterranean Diet and Risk of Type 2 Diabetes: An Updated Systematic Review and Dose–Response Meta-Analysis of Prospective Cohort Studies. Advances in Nutrition [Internet]. 2022 Oct 2 [cited 2022 Oct 9];13(5):1787–98. Available from: https://academic.oup.com/advances/article/13/5/1787/6574405

53. Martino F, Martino E, Versacci P, Niglio T, Zanoni C, Puddu PE. Lifestyle and awareness of cholesterol blood levels among 29159 community school children in Italy. Nutrition, Metabolism and Cardiovascular Diseases [Internet]. 2019 Aug 1;29(8):802–7. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0939475319302078

54. Kastorini CM, Milionis HJ, Esposito K, Giugliano D, Goudevenos JA, Panagiotakos DB. The Effect of Mediterranean Diet on Metabolic Syndrome and its Components. J Am Coll Cardiol [Internet]. 2011 Mar 15 [cited 2023 Feb 28];57(11):1299–313. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0735109710050679

55. Davis C, Bryan J, Hodgson J, Murphy K. Definition of the Mediterranean Diet; A Literature Review. Nutrients [Internet]. 2015 Nov 5 [cited 2022 Jan 10];7(11):9139–53. Available from: http://www.mdpi.com/2072-6643/7/11/5459

56. Horta BL, Loret de Mola C, Victora CG. Long-term consequences of breastfeeding on cholesterol, obesity, systolic blood pressure and type 2 diabetes: a systematic review and meta-analysis. Acta Paediatr [Internet]. 2015 Dec 1 [cited 2022 Sep 6];104:30–7. Available from: https://onlinelibrary.wiley.com/doi/10.1111/apa.13133

57. Martin RM, Kramer MS, Patel R, Rifas-Shiman SL, Thompson J, Yang S, et al. Effects of Promoting Long-term, Exclusive Breastfeeding on Adolescent Adiposity, Blood Pressure, and Growth Trajectories: A Secondary Analysis of a Randomized Clinical Trial. JAMA Pediatr [Internet]. 2017 Jul 3 [cited 2022 Jul 13];171(7):e170698. Available from: http://www.ncbi.nlm.nih.gov/pubmed/28459932

58. Wisnieski L, Kerver J, Holzman C, Todem D, Margerison-Zilko C. Breastfeeding and Risk of Metabolic Syndrome in Children and Adolescents: A Systematic Review. Journal of Human Lactation [Internet]. 2018 Aug 3 [cited 2022 May 13];34(3):515–25. Available from: http://journals.sagepub.com/doi/10.1177/0890334417737038

Table 1. General characteristics of the studied participants across categories of adherence to the composite score comprised of 6 healthy lifestyle behaviors.

	Tertiles of adherence to the healthy lifestyle behavior composite score			
	T1 (<3 points) n=392	**T2** (3-4 points) n=296	**T3** (>4 points) n=250	**p value**
Age, years	4.8 ± 1.0	4.8 ± 1.1	4.9 ± 1.1	0.682
Girls, % (n)	49.0 (192)	50.7 (150)	48.4 (121)	0.852
Early life factors				
Birth weight, kg	3.3 ± 0.6	3.2 ± 0.6	3.3 ± 0.5	0.677
Birth weight				0.525
Low birth weight, % (n)	7.1 (28)	8.5 (25)	5.2 (13)	
Normal birth weight, % (n)	86.0 (337)	84.8 (251)	89.6 (224)	
High birth weight, % (n)	6.9 (27)	6.8 (20)	5.2 (13)	
Mother weight gain during pregnancy, kg	12.5 ± 4.6	12.5 ± 4.5	12.5 ± 4.6	0.974
Maternal factors				
Age, years	40.4 ± 5.5	41.2 ± 5.2	41.3 ± 7.5	0.138
BMI, kg/m^2	25.7 ± 5.3^{ab}	24.6 ± 5.0^{a}	23.9 ± 4.2^{b}	**<0.001**
Weight status				**<0.001**
Underweight or normal weight, % (n)	53.1 (208)	65.5 (194)	69.6 (174)	
Overweight or obesity, % (n)	46.9 (184)	34.5 (102)	30.4 (76)	
Educational level				**<0.001**
Primary or lower, % (n)	15.1 (59)	9.1 (27)	2.0 (5)	
Secondary, % (n)	46.9 (184)	35.8 (106)	34.4 (86)	
Academic – graduated or no reported data, % (n)	38.0 (149)	55.1 (163)	63.6 (159)	

Socio-professional category, % (n)				0.362
Homemaker/student/retired/unemployed	30.9 (121)	27.4 (81)	26.0 (65)	
Employee	69.1 (271)	72.6 (215)	74.0 (185)	

Data are expressed as mean ± SD or median [IQR] for continuous variables and percentages (numbers) for categorical variables.

P-values were calculated by the chi-square or ANOVA test for categorical and continuous variables, respectively. P values <0.05 were considered significant.

The Bonferroni's test for multiple comparisons was used for the results of maternal BMI. Significant differences (p value <0.05) between categories of adherence to the healthy lifestyle behaviors composite score are expressed as: a=T1vs.T2; b=T1vs.T3 and c=T2vs.T3.

Table 2. Lifestyle and cardiometabolic risk factors in the studied population across categories of adherence to the composite score comprised of 6 healthy lifestyle behaviors.

Lifestyle behaviors				
Breastfeeding, months	4.9 ± 7.4[ab]	6.8 ± 9.0[ac]	9.9 ± 10.2[bc]	**<0.001**
Exclusive Breastfeeding, % (n)	15.8 (62)	31.4 (93)	71.2 (178)	**<0.001**
Total sleep duration, hours/day	10.2 ± 1.0[ab]	10.5 ± 0.7[a]	10.6 ± 0.7[b]	**<0.001**
Sleeping pattern for age				**<0.001**
Inadequate, % (n)	36.2 (142)	11.5 (34)	5.2 (13)	
Adequate, % (n)	63.8 (250)	88.5 (262)	94.8 (237)	
Sports and physical activities, minutes/week	154.6 ± 114.1[ab]	195.8 ± 117.7[ac]	220.2 ± 100.4[bc]	**<0.001**
Healthy behavior (≥120 min/week), % (n)	53.1 (208)	77.4 (229)	92.4 (231)	**<0.001**
Screentime, hours/day	2.4 ± 1.1[ab]	1.6 ± 0.9[ac]	1.3 ± 0.6[bc]	**<0.001**
Healthy behavior (<2 h/day), % (n)	37.8 (148)	75.0 (222)	94.8 (237)	**<0.001**
Adherence to Mediterranean diet, 0-18 points	9.6 ± 2.5[ab]	10.8 ± 2.6[ac]	12.3 ± 2.4[bc]	**<0.001**
Eating speed, minutes/day	69.7 ± 22.1[ab]	78.6 ± 23.2[ac]	90.6 ± 26.2[bc]	**<0.001**
Adiposity				
BMI, kg/m^2	16.7 ± 2.3[b]	16.3 ± 2.0	15.9 ± 1.8[b]	**<0.001**
Weight status				**<0.001**
Underweight or normal weight, % (n)	70.2 (275)	80.7 (239)	88.4 (221)	
Overweight or obesity, % (n)	29.9 (117)	19.3 (57)	11.6 (29)	
Waist circumference, cm	52.6 ± 7.7	52.2 ± 6.3	51.4 ± 5.8	0.103
Fat mass index, kg/m^2	4.1 ± 1.4[ab]	3.8 ± 1.2[a]	3.6 ± 1.2[b]	**<0.001**

Cardiometabolic risk factors				
Systolic blood pressure, mmHg	105.2 ± 12.5[b]	104.0 ± 14.4[c]	100.4 ± 11.8[bc]	**<0.001**
Diastolic blood pressure, mmHg	65.8 ± 11.8[b]	66.0 ± 13.6[c]	62.2 ± 11.4[bc]	**<0.001**
Fasting plasma glucose, mg/dL	77.8 ± 8.5	77.5 ± 10.6	77.2 ± 8.9	0.759
Total cholesterol, mg/dL	163.5 [146-181]	165 [149-181]	169 [148-187]	0.280
HDL cholesterol, mg/dL	57 [49-67]	57 [49-64]	57 [49-66]	0.883
LDL cholesterol, mg/dL	94 [79-108]	96 [84-111]	94.5 [82-112.3]	0.300
Non-HDL cholesterol, mg/dL	104 [89-122]	107 [95-124]	107 [94-125.4]	0.176
Triglycerides, mg/dL	53 [44-68]	53 [43-65]	52 [43-64]	0.783

Abbreviations: BMI, body mass index, HDL, high-density lipoprotein; LDL low-density lipoprotein.

Data are expressed as mean ± SD or median [IQR] for continuous variables and percentages (numbers) for categorical variables.

P-values were calculated by the chi-square or ANOVA test for categorical and continuous variables, respectively. P values <0.05 were considered significant.

The Bonferroni's test for multiple comparisons was used in those significant results calculated by analysis of covariance. Significant differences (p value <0.05) between categories of adherence to the 6-healthy lifestyle behaviors composite score are expressed as: a=T1vs.T2; b=T1vs.T3 and c=T2vs.T3.

Table 3. Dietary characteristics of the study participants across categories of adherence to the composite score comprised of 6 healthy lifestyle behaviors.

	Tertiles of adherence to the healthy lifestyle behavior composite score			
	T1 (<3 points)	**T2** (3-4 points)	**T3** (>4 points)	**p value**
	n=392	n=296	n=250	
Dietary intake contribution				
Total energy intake, kcal/day	1752 ± 378[ab]	1674 ± 352[a]	1642 ± 304[b]	**<0.001**
Carbohydrates, % of total energy intake	43.5 ± 5.7[b]	42.7 ± 5.0	42.2 ± 5.6[b]	**0.009**
Proteins, % of total energy intake	14.7 ± 2.1[ab]	15.2 ± 2.1[a]	15.2 ± 2.1[b]	**<0.001**
Protein intake per body weight, g/kg/day	3.2 ± 1.0	3.3 ± 0.9	3.4 ± 0.9	0.058
Total fat, % of total energy intake	41.9 ± 5.8	42.1 ± 5.4	42.6 ± 6.3	0.293
Saturated fatty acids, % of total energy intake	14.0 ± 2.0	14.0 ± 2.0	13.8 ± 2.0	0.579
Monounsaturated fatty acids, % of total energy intake	18.5 ± 4.4[b]	19.0 ± 4.1	19.6 ± 5.0[b]	**0.013**
Polyunsaturated fatty acids, % of total energy intake	6.3 ± 1.9	6.0 ± 1.4	6.0 ± 1.5	**0.034**
Fiber, g/1,000 kcal	8.0 ± 2.1[ab]	8.5 ± 2.3[ac]	9.2 ± 2.5[bc]	**<0.001**
≥14 gr/1000 kcal, % (n)	1.3 (5)	3.4 (10)	6.4 (16)	**0.002**
Sodium, mg/dL	2383 ± 737[b]	2322 ± 694	2182 ± 672[b]	**0.002**
Dairy products				

Milk, g/day	329.4 ± 227.8	331.1 ± 242.9	291.8 ± 216.1	0.078
Yogurt, g/day	110.4 ± 98.4	118.1 ± 97.2	104.1 ± 80.1	0.214
Cheese, g/day	11.9 ± 12.0[b]	11.5 ± 12.6[c]	14.6 ± 11.3[bc]	**0.006**
Other dairy products, g/day	104.3 ± 102.9[ab]	75.7 ± 85.4[a]	64.9 ± 73.1[b]	**<0.001**
Protein foods				
White meat, g/day	24.2 ± 9.5	23.9 ± 9.8	24.7 ± 8.2	0.598
Unprocessed red meat, g/day	18.8 ± 13.6	19.3 ± 11.9	18.6 ± 12.6	0.809
Processed and derivatives meat products, g/day	26.6 ± 16.2[b]	25.2 ± 14.6	23.5 ± 13.0[b]	**0.036**
Egg, g/day	22.7 ± 9.8	24.4 ± 9.1	23.3 ± 8.3	0.053
Fish and seafood, g/day	30.6 ± 17.7[ab]	35.8 ± 17.3[a]	36.3 ± 17.0[b]	**<0.001**
Vegetables and fruits				
Vegetables, g/day	70.6 ± 53.6[b]	75.3 ± 47.3[c]	88.3 ± 51.6[bc]	**<0.001**
Tubers, g/day	42.8 ± 20.0[b]	40.0 ± 19.7	37.6 ± 17.9[b]	**0.003**
Fruits, g/day	163.2 ± 114.1[ab]	193.2 ± 113.1[ac]	217.9 ± 130.4[bc]	**<0.001**
Nuts				
Nuts, g/day	3.1 ± 4.2[b]	3.6 ± 5.3	4.5 ± 5.4[b]	**0.001**
Cereals and Legumes				
Legumes, g/day	14.5 ± 8.3	13.6 ± 6.1	14.1 ± 6.1	0.248
Refined cereals, g/day	74.5 ± 36.9	70.8 ± 32.4	71.1 ± 36.2	0.312

Whole cereals, g/day	5.8 ± 13.2[b]	8.2 ± 15.0[c]	11.4 ± 18.8[bc]	**<0.001**
Miscellaneous				
Oil and fats, g/day	26.0 ± 15.9	25.9 ± 14.6	27.8 ± 16.8	0.303
Pastries, g/day	43.5 ± 31.8[b]	39.0 ± 28.6	35.9 ± 25.1[b]	**0.005**
Sugars and candies, g/day	14.9 ± 11.4[ab]	12.8 ± 10.6[a]	11.1 ± 9.1[b]	**<0.001**
Beverages				
Water, ml/day	855.3 ± 359.4	846.2 ± 359.8	865.4 ± 409.9	0.837
Sugary beverages, ml/day	132.3 ± 145.8[ab]	101.1 ± 109.2[a]	92.5 ± 107.9[b]	**<0.001**
Tea and infusions, ml/day	6.3 ± 27.6	7.6 ± 33.4	6.9 ± 26.1	0.855

Data are expressed as mean ± SD.

P-values were calculated by the ANOVA. The p values <0.05 were considered significant.

The Bonferroni's test for multiple comparisons was used for those significant results calculated by analysis of covariance. Significant differences (p value <0.05) between categories of adherence to the 6-healthy lifestyle behaviors composite score are expressed as: a=T1vs. T2; b=T1 vs. T3 and c=T2 vs. T3.

Table 4. Associations between the adherence to the composite score comprised of 6 healthy lifestyle behaviors and several cardiometabolic risk factors.

	Tertiles of adherence to the healthy lifestyle behavior composite score		
	T1 (<3 points)	**T2** (3-4 points)	**T3** (>4 points)
Adiposity			
Weight status, n[#]	392	296	250
Crude model	1 (ref.)	0.6 (0.4, 0.8) **	0.3 (0.2, 0.5) **
Adjusted model	1 (ref.)	0.6 (0.4, 0.9) *	0.4 (0.2, 0.6) **
Waist circumference, n[¥]	388	293	247
Crude model	0 (ref.)	-0.3 (-1.4, 0.7)	-1.2 (-2.3, -0.1) *
Adjusted model	0 (ref.)	-0.4 (-1.3, 0.6)	-1.4 (-2.5, -0.4) **
Fat mass index, n[¥]	342	265	222
Crude model	0 (ref.)	-0.3 (-0.5, -0.1) **	-0.5 (-0.7, -0.3) **
Adjusted model	0 (ref.)	-0.2 (-0.4, 0.1)	-0.3 (-0.5, -0.1) *
Cardiometabolic risk factors			
Systolic blood pressure, n[¥]	376	278	231
Crude model	0 (ref.)	-1.2 (-3.2, 0.8)	-4.8 (-6.9, -2.7) **
Adjusted model	0 (ref.)	-0.1 (-2.1, 1.9)	-3.0 (-5.2, -0.9) **
Diastolic blood pressure, n[¥]	376	278	233

Crude model	0 (ref.)	0.1 (-1.8, 2.1)	-3.6 (-5.6, -1.6) **
Adjusted model	0 (ref.)	1.1 (-0.8, 2.9)	-2.0 (-4.1, 0.0)
Fasting plasma glucose, n[¥]	340	237	208
Crude model	0 (ref.)	-0.2 (-1.8, 1.3)	-0.6 (-2.2, 1.0)
Adjusted model	0 (ref.)	-0.8 (-2.3, 0.6)	-1.9 (-3.5, -0.4) *
Total cholesterol, n[¥]	338	237	207
Crude model	0 (ref.)	2.6 (-2.1, 7.3)	3.3 (-1.6, 8.1)
Adjusted model	0 (ref.)	2.3 (-2.4, 7.0)	2.1 (-3.0, 7.2)
HDL cholesterol, n[¥]	336	237	207
Crude model	0 (ref.)	-0.9 (-3.2, 1.3)	0.3 (-2.1, 2.6)
Adjusted model	0 (ref.)	-1.3 (-3.6, 0.9)	-0.5 (-3.0, 2.0)
LDL cholesterol, n[¥]	314	227	196
Crude model	0 (ref.)	3.1 (-1.1, 7.3)	1.4 (-3.0, 5.8)
Adjusted model	0 (ref.)	2.7 (-1.5, 7.0)	-0.0 (-4.5, 4.5)
Non-HDL cholesterol, n[¥]	336	237	207
Crude model	0 (ref.)	3.9 (-0.3, 8.1)	3.4 (-1.0, 7.7)
Adjusted model	0 (ref.)	4.0 (-0.2, 8.3)	3.0 (-1.6, 7.6)
Triglycerides, n[¥]	337	237	207
Crude model	0 (ref.)	-1.1 (-4.6, 2.4)	0.5 (-3.2, 4.2)

Adjusted model	0 (ref.)	-0.0 (-3.5, 3.5)	2.9 (-0.9, 6.7)

Abbreviations: CI, confidence interval, HDL cholesterol, high-density lipoprotein cholesterol; LDL cholesterol, low-density lipoprotein cholesterol; OR, odds ratio.

Tertiles of adherence to the composite score comprised of 6 healthy lifestyle behaviors (exposure). [#] Multivariable logistic regression model: weight status was a dichotomous outcome (underweight or normal weight -1- and overweight or obesity -2-) and results were expressed in OR (95% CI). [¥] Multivariable lineal regression models: waist circumference in cm; fat mass index in kg/m^2; systolic and diastolic blood pressure in mmHg; fasting plasma glucose and lipid profile in mg/dL as outcomes which results were expressed in β (95%CI).

All models were adjusted by recruitment center (3 categories according to the number of participants), sex, age and mother's educational level (primary or lower, secondary, academic-graduate or no reported data), birth weight (low/normal/high) and maternal overweight/obesity (yes/no). *P values <0.05; **P values <0.01.

Table 5. Associations between each of the 6 healthy lifestyle behaviors included in the composite score and cardiometabolic risk factors.

Adiposity		
		Additional adjustment §
Weight Status (overweight/obesity: no/yes)[#]		
Exclusive breastfeeding to the first 6 months	0.8 (0.6, 1.2)	0.8 (0.6, 1.2)
Adequate sleep duration for age	0.6 (0.4, 0.9) *	0.6 (0.4, 0.9) **
Physical activity for ≥2 h/week	0.9 (0.6, 1.3)	1.0 (0.7, 1.5)
Screentime for <2 h/day	0.6 (0.4, 0.8) **	0.6 (0.4, 0.9) *
Higher adherence to the Mediterranean diet (T3 ≥ 13 points)	0.7 (0.5, 1.2)	0.9 (0.5, 1.4)
Slow eating (T3 > 85 min. in the 3 main meals)	0.4 (0.2, 0.6) **	0.4 (0.2, 0.6) **
Waist circumference, cm[¥]		
Exclusive breastfeeding to the first 6 months	-0.3 (-1.2, 0.6)	-0.3 (1.2, 0.6)
Adequate sleep duration for age	-0.9 (-2.0, 0.1)	-0.9 (-2.0, 0.1)
Physical activity for ≥2 h/week	-0.3 (-1.3, 0.6)	-0.1 (-1.1, 0.8)
Screentime for <2 h/day	-1.3 (-2.1, -0.4) **	-1.2 (-2.1, -0.3) **
Higher adherence to the Mediterranean diet (T3 ≥ 13 points)	-0.0 (-1.0, 1.0)	0.3 (-0.7, 1.3)
Slow eating (T3 > 85 min. in the 3 main meals)	-2.2 (-3.2, -1.1) **	-2.2 (-3.2, -1.2) **

Fat mass index, kg/m²¥		
Exclusive breastfeeding to the first 6 months	0.0 (-0.2, 0.2)	0.0 (-0.1, 0.2)
Adequate sleep duration for age	-0.2 (-0.4, 0.0)	-0.2 (-0.4, 0.1)
Physical activity for ≥2 h/week	-0.1 (-0.3, 0.1)	-0.1 (-0.3, 0.1)
Screentime for <2 h/day	-0.2 (-0.4, -0.0) *	-0.2 (-0.4, -0.0) *
Higher adherence to the Mediterranean diet (T3 ≥ 13 points)	-0.1 (-0.3, 0.1)	-0.1 (-0.3, 0.2)
Slow eating (T3 > 85 min. in the 3 main meals)	-0.3 (-0.5, -0.1) **	-0.3 (-0.5, -0.1) **
Cardiometabolic risk factors		
Systolic blood pressure, mmHg¥		
Exclusive breastfeeding to the first 6 months	-2.0 (-3.8, -0.2) *	-1.8 (-3.6, -0.0) *
Adequate sleep duration for age	1.2 (-0.9, 3.4)	1.3 (-0.9, 3.4)
Physical activity for ≥2 h/week	-1.5 (-3.5, 0.4)	-1.4 (-3.4, 0.6)
Screentime for <2 h/day	-0.8 (-2.7, 1.0)	-0.6 (-2.5, 1.3)
Higher adherence to the Mediterranean diet (T3 ≥ 13 points)	-2.2 (-4.4, 0.0)	-1.9 (-4.2, 0.3)
Slow eating (T3 > 85 min. in the 3 main meals)	-2.3 (-4.4, -0.3) *	-2.0 (-4.1, 0.1)
Diastolic blood pressure, mmHg¥		
Exclusive breastfeeding to the first 6 months	-1.3 (-2.9, 0.4)	-1.2 (-2.9, 0.5)
Adequate sleep duration for age	0.3 (-1.7, 2.2)	0.3 (-1.7, 2.3)
Physical activity for ≥2 h/week	-1.9 (-3.8, -0.1) *	-1.8 (-3.7, 0.1)

Screentime for <2 h/day	-0.3 (-2.1, 1.4)	-0.4 (-2.1, 1.4)
Higher adherence to the Mediterranean diet (T3 ≥ 13 points)	0.5 (-1.6, 2.6)	0.7 (-1.5, 2.8)
Slow eating (T3 > 85 min. in the 3 main meals)	-1.1 (-3.1, 0.8)	-1.0 (-3.0, 1.0)
Fasting plasma glucose, mg/dL[¥]		
Exclusive breastfeeding to the first 6 months	-0.5 (-1.7, 0.8)	-0.4 (-1.7, 0.8)
Adequate sleep duration for age	-0.4 (-1.8, 1.1)	-0.5 (-2.0, 0.9)
Physical activity for ≥2 h/week	-0.4 (-1.8, 1.1)	-0.2 (-1.6, 1.3)
Screentime for <2 h/day	-0.0 (-1.3, 1.2)	0.2 (-1.1, 1.5)
Higher adherence to the Mediterranean diet (T3 ≥ 13 points)	-1.0 (-2.5, 0.5)	-0.9 (-2.4, 0.6)
Slow eating (T3 > 85 min. in the 3 main meals)	-2.5 (-4.0, -1.1) **	-2.5 (-3.9, -1.0) **
Total cholesterol, mg/dL[¥]		
Exclusive breastfeeding to the first 6 months	-3.9 (-8.1, 0.3)	-4.0 (-8.2, 0.2)
Adequate sleep duration for age	2.7 (-2.2, 7.5)	2.1 (-2.7, 7.0)
Physical activity for ≥2 h/week	-2.9 (-7.6, 1.9)	-3.0 (-7.7, 1.7)
Screentime for <2 h/day	6.8 (2.6, 11.0) **	6.8 (2.5, 11.1) **
Higher adherence to the Mediterranean diet (T3 ≥ 13 points)	-1.4 (-3.5, 6.3)	0.3 (-4.6, 5.2)
Slow eating (T3 > 85 min. in the 3 main meals)	3.6 (-1.3, 8.5)	3.7 (-1.2, 8.6)
HDL cholesterol, mg/dL[¥]		
Exclusive breastfeeding to the first 6 months	-0.9 (-3.0, 1.1)	-1.0 (-3.1, 1.0)

Adequate sleep duration for age	-1.3 (-3.6, 1.1)	-1.4 (-3.8, 0.9)
Physical activity for ≥2 h/week	-0.5 (-2.8, 1.8)	-0.5 (-2.8, 1.8)
Screentime for <2 h/day	1.7 (-0.3, 3.8)	2.0 (-0.1, 4.1)
Higher adherence to the Mediterranean diet (T3 ≥ 13 points)	-1.6 (-4.0, 0.7)	-2.0 (-4.4, 0.4)
Slow eating (T3 > 85 min. in the 3 main meals)	0.9 (-1.5, 3.3)	0.9 (-1.5, 3.3)
LDL cholesterol, mg/dL[¥]		
Exclusive breastfeeding to the first 6 months	-3.7 (-7.4, 0.0)	-3.6 (-7.3, 0.1)
Adequate sleep duration for age	3.1 (-1.2, 7.4)	2.9 (-1.4, 7.2)
Physical activity for ≥2 h/week	-4.3 (-8.5, -0.1) *	-4.3 (-8.5, 0.0) *
Screentime for <2 h/day	3.4 (-0.4, 7.3)	3.0 (-0.9, 6.9)
Higher adherence to the Mediterranean diet (T3 ≥ 13 points)	3.7 (-0.7, 8.0)	3.1 (-1.2, 7.5)
Slow eating (T3 > 85 min. in the 3 main meals)	2.6 (-1.6, 6.9)	2.7 (-1.5, 7.0)
Non-HDL cholesterol, mg/dL[¥]		
Exclusive breastfeeding to the first 6 months	-2.7 (-6.5, 1.0)	-2.7 (-6.5, 1.0)
Adequate sleep duration for age	3.8 (-0.5, 8.1)	3.4 (-1.0, 7.7)
Physical activity for ≥2 h/week	-2.9 (-7.1, 1.4)	-3.0 (-7.2, 1.3)
Screentime for <2 h/day	5.7 (1.9, 9.5) **	5.4 (1.5, 9.2) **
Higher adherence to the Mediterranean diet (T3 ≥ 13 points)	3.3 (-1.1, 7.7)	2.4 (-2.0, 6.8)
Slow eating (T3 > 85 min. in the 3 main meals)	2.6 (-1.7, 7.0)	2.7 (-1.6, 7.1)

Triglycerides, mg/dL[¥]		
Exclusive breastfeeding to the first 6 months	1.5 (-1.6, 4.6)	1.5 (-1.6, 4.6)
Adequate sleep duration for age	3.6 (-0.0, 7.2)	3.6 (-0.0, 7.2)
Physical activity for ≥2 h/week	3.4 (-0.2, 6.9)	2.8 (-0.7, 6.4)
Screentime for <2 h/day	-0.4 (-3.6, 2.7)	-0.5 (-3.7, 2.7)
Higher adherence to the Mediterranean diet (T3 ≥ 13 points)	-4.2 (-7.9, -0.6) *	-4.3 (-7.9, -0.6) *
Slow eating (T3 > 85 min. in the 3 main meals)	2.8 (-0.9, 6.4)	3.0 (-0.7, 6.6)

Abbreviations: CI, confidence interval, HDL cholesterol, high-density lipoprotein cholesterol; LDL cholesterol, low-density lipoprotein cholesterol; OR, odds ratio.

Tertiles of adherence to the composite score comprised of 6 healthy lifestyle behaviors (exposure). [#] Multivariable logistic regression model: weight status was a dichotomous outcome (underweight or normal weight -1- and overweight or obesity -2-) and results were expressed in OR (95% CI). [¥] Multivariable lineal regression models: waist circumference in cm; fat mass index in kg/m^2; systolic and diastolic blood pressure in mmHg; fasting plasma glucose and lipid profile in mg/dL as outcomes which results were expressed in β (95%CI).

All models were adjusted by recruitment center (3 categories according to the number of participants), sex, age and mother's educational level (primary or lower, secondary, academic-graduate or no reported data), birth weight (low/normal/high) and maternal overweight/obesity (yes/no).

§ Each model was further adjusted by the other lifestyle behaviors, except for the one of interest. * P values <0.05 ** P values <0.01

VI. DISCUSSION

General discussion

In the present thesis, the summary of the main findings is that several lifestyle behaviors, including MedDiet, eating speed, eating frequency, breastfeeding, sleep duration, physical activity and screentime, may play an important role on the onset of obesity and its associated cardiometabolic risk in children. However, their contribution to these health outcomes may not be equally distributed. In this sense, fast-eating speed was observed in association with increased adiposity, higher levels of systolic blood pressure and fasting plasma glucose but lower adherence to the MedDiet. On the other hand, slow eating speed showed a major role in the concomitant associations observed between healthy behaviors for breastfeeding, sleep duration, physical activity, screentime, adherence to MedDiet and eating speed and lower adiposity as well as cardiometabolic risk, specifically systolic blood pressure levels and fasting plasma glucose concentration. Furthermore, in individual associations, slow eating was the lifestyle behavior associated with lower fasting plasma glucose concentration and, increased adherence to the MedDiet had a solely contribution to lowering serum triglycerides levels.

In line with the results of the present thesis, similar associations have been reported between eating speed and eating frequency and adiposity[193–196] and/or cardiometabolic risk [197–199] in children with

different ethnicities. In contrast, positive associations between eating frequency and adiposity have also been observed [200] but only in children with central obesity, who were more likely to show lower levels of physical activity which may support this finding. On the other hand, eating speed has been associated to several measures of adiposity, including percentage of body fat mass[201] and waist-to-height ratio[202] but to our knowledge, no published articles have considered fat mass index. Regarding evidence on the associations between fast-eating speed and increased systolic blood pressure or fasting plasma glucose concentration in children, similar results have been reported but only for systolic blood pressure[203].

With respect to slow eating, a Finnish observational study[204] and an American clinical trial[196] conducted in children, also observed associations between slow eating and favorable levels of adiposity however, associations with cardiometabolic risk factors were not assessed in none of them.

It is important to highlight that evidence on the associations between eating speed and health outcomes, has been commonly reported in terms of the fast-eating category, being slow eating usually the reference category. Additionally, it should be noted that the process of eating and oral proccessing comprises many other factors[205–211], including satiety signals[212], all of which may impact directly or indirectly on adiposity and cardiometabolic risk. In this sense, eating pace (e.g., eating speed, eating rate or energy intake

rate) have been related to sensory and dietary characteristics such as certain textures[213–215], tastes[216,217] and food composition[216], all of which could play a role on dietary and eating behaviors[218,219] which is evidenced by the positive associations that have been reported between eating speed and sweet-tasting [216] or ultra-processed foods [220].

Regarding the observed association between fast-eating category and decreased adherence to MedDiet, at present no comparisons can be made due to lack of similar published articles. However, this result could have received a substantial contribution by certain characteristics of the MedDiet which is widely recognized by its benefits on health[221–225] and comprises plenty plant-based foods, whole grains, legumes, olive oil, nuts, dairy and fish/seafood, moderate consumption of wine, poultry, eggs and lower intake of red and processed meat as well as other processed foods[226,227]. Moreover, the consumption of minimally processed diets, which may be extended to MedDiet, has been reported in association with satiety signals [228] which could explain differences in eating speed. On the other hand, previous studies[229,230] have observed greater adherence to MedDiet associated with lower serum triglyceride levels which is in line with the results of the present thesis, yet in contrast with Lampropoulou et al [231].

With regard to the impact of MedDiet on cardiometabolic risk, it is important to consider that multiple bioactive compounds are higher

in plant-based diets which include fiber dietary, minerals, vitamins, fatty acids, proteins, some carbohydrates and polyphenols, all of which have been suggested to play a direct role in obesity through inflammation and cardiometabolic risk factors[232].

Results of the present thesis are also consistent with several cross-sectional [111,135,163,164] and longitudinal[111] studies regarding the associations between the co-occurrence of healthy lifestyle behaviors and decreased adiposity in children however, only Bawaked et al.[111] assessed cardiometabolic risk, as measured by HDL-c, triglycerides and blood pressure, but no significant results were observed at baseline or in the follow-up. In this sense, it must be noted that the methodology of the composite score comprised of 6 healthy lifestyle behaviors, created for the purposes of this thesis, differs from previously designed lifestyle indexes targeting populations of children[111,135,163,164]. This is mainly due to differences in the assessment of lifestyle behaviors, in which compliance with recommendations for lifestyle behaviors where mostly disregarded and adherence to the MedDiet, eating speed or breastfeeding were not considered as lifestyle behaviors.

On the other hand, regarding the contribution of breastfeeding and in line with the present thesis, several benefits on adiposity[233] and cardiometabolic profile[93,234] have been reported in breastfed children however, evidence on exclusive breastfeeding is controversial at present[95,235] mainly due to insufficient long-term

and interventional studies[236]. Otherwise, it is worth noting that sedentary behaviors assessment usually focuses on the screentime, so additional related factors that might also have health consequences are rarely assessed, such as the type of digital media/device and the content to which the children are exposed (educational, recreational, etc.)[237], including advertisements[144], as well as the setting in which digital media is used (school, home, mealtimes or others).

Additionally, we cannot discard the existence of interactions between the lifestyle behaviors assessed for the purposes of the present thesis, which may explain some of the results. In this sense, increased adiposity has been observed in relation to the combined associations of higher screentime but lower sleep duration and physical activity levels [135] . Similarly, increased screentime as well as consumption of saturated fats and sugar-sweetened beverages but lower protein intake have been reported in association with a detrimental cardiometabolic profile in children [238]. Besides, higher adherence to the MedDiet has been associated with higher physical activity[132,239,240] but lower screentime[240,241], which also may lead to differences on cardiometabolic health.

Limitations and Strengths

Certain limitations of the present thesis required to be mentioned in order to avoid misunderstandings about the results. First, this thesis is not comprised of long-term and/or interventional studies so it is not possible to draw cause-effect conclussions or to discard bidirectional associations, especially between eating speed and diet quality. Second, it might exist residual confounding or undetected cardiometabolic disorders due to early age in the studied population. Third, results cannot be extrapolated to other populations since analyses have been conducted in a population of Spanish preschool children. Fourth, data has been collected through questionnaires however, eating speed was not estimated based on parents' self-perception (e.g., low, mild or fast eating speed) but on the total minutes required to eat each meal. Furthermore, analyses assessing the associations between eating speed, adiposity and cardiometabolic risk factors were replicated considering the CEBQ scale "slowness in eating" as main exposure. Fifth, equations for bioelectrical bioimpedance are not validated in children aged <5 years and therefore, sensitivity analyses were conducted. Finally, even though the 18-item MedDiet questionnaire has been adapted to children according to a questionnaire validated in an elderly population, the results should be interpreted with caution, since validation is in progress.

Limitations and Strenghts

The present thesis has also strengths that merit to be mentioned. First, the analyses were conducted on a population comprised of a large sample size from the 7 CORALS study recruitment centers. Second, a specific and homogeneous methodology was used across all participants. Third, data from blood samples collected was available for the total studied population. Fourth, cardiometabolic risk was assessed by several adiposity measures and cardiometabolic risk factors. Fifth, several confounding variables were considered for the purposes of the present thesis, as well as sensitivity analyses. Additionally, further adjustment by all those lifestyle behaviors that were not of interest when assessing the individual associations between each of the 6 healthy lifestyle behaviors and cardiometabolic risk outcomes.

VII. CONCLUSIONS

English

In overall, the main conclusion of the present thesis is that eating speed is cross-sectionally associated with adiposity and levels of cardiometabolic risk factors in healthy preschool children, individually and as part of a composite score that also includes other lifestyle behaviors such as diet quality, exclusive breastfeeding, physical activity, sleep duration and sedentary behaviors.

Hypothesis n° 1:

Fast-eating speed is associated with increased adiposity and levels of certain cardiometabolic risk factors as well as with lower diet quality.

- Compared with slow eating, those participants allocated in the fast-eating category showed higher levels of adiposity (overweight or obesity, higher waist circumference, FMI), and increased levels of certain cardiometabolic risk factors (systolic blood pressure and fasting plasma glucose) but lower adherence to the MedDiet.

Hypothesis n° 2:

Higher diet quality, slow eating and the compliance with recommendations for breastfeeding, physical activity, sleep duration and sedentary behaviors are associated with lower

prevalence of overweight or obesity and lower levels of cardiometabolic risk factors which could lead to decreased risk of disease in childhood.

- Higher adherence to a composite score comprised of 6 healthy lifestyle behaviors was associated with lower adiposity, systolic blood pressure levels and fasting plasma glucose concentration in preschool children.
- Slow eating showed to be a major contributor in the associations observed with adiposity and cardiometabolic risk factors and has a sole contribution in lowering fasting plasma glucose concentration.
- Higher adherence to the MedDiet was negatively associated with serum triglycerides levels.

The results of the present thesis could favor the design of enhanced strategies for the prevention and/or treatment of childhood obesity and its associated comorbidities, contributing to alleviate one of the greatest concerns of Public Health: childhood obesity and the future health of the younger generations. From this perspective, the fact that a simple checklist of relevant lifestyle behaviors enables the early detection of potential detrimental effects on cardiometabolic health in children is becoming more feasible. However, further research is warranted due to the scarce number of longitudinal studies and rigorous clinical trials as well as a greater number of under-powered studies in children.

Castellano

En general, la principal conclusión de la presente tesis es que la velocidad de la ingesta se asocia transversalmente con la adiposidad y los niveles de factores de riesgo cardiometabólico en niños preescolares sanos, individualmente y como parte de un sistema de puntuación que también incluyó la calidad de la dieta, lactancia materna exclusiva, actividad física, duración del sueño y conductas sedentarias.

Hipótesis n° 1:

La velocidad a la que se come rápido se asocia con un aumento de la adiposidad y de los niveles de determinados factores de riesgo cardiometabólico, así como con una menor calidad de la dieta.

- En comparación con comer despacio, los participantes asignados a la categoría de comer rápido presentaron mayores niveles de adiposidad (sobrepeso u obesidad, mayor perímetro de cintura, índice de masa grasa) y niveles de ciertos factores de riesgo cardiometabólico (presión arterial sistólica y glucosa plasmática en ayunas) pero menor adherencia a la dieta Mediterránea.

Hipótesis n° 2:

Conclusiones

Una mayor calidad de la dieta, comer lento y cumplir con las recomendaciones para lactancia materna, actividad física, duración del sueño y tiempo de pantallas se asocian a una menor prevalencia de sobrepeso u obesidad y a disminución de los niveles de algunos factores de riesgo cardiometabólico, lo que podría conducir a menor riesgo de enfermedad en la infancia.

- Una mayor adherencia a una puntuación compuesta de 6 comportamientos de estilo de vida saludables se asoció con una menor adiposidad, presión arterial sistólica y concentración de glucosa en plasma en ayunas en niños en edad preescolar.
  - Comer lento mostró una mayor contribución en las asociaciones observadas para la adiposidad y los factores de riesgo cardiometabólico, y fue el único comportamiento de estilo de vida asociado a una menor concentración de glucosa plasmática en ayunas.
- La mayor adherencia a la MedDiet se asoció negativamente con los niveles séricos de triglicéridos.

En este sentido, los resultados de la presente tesis podrían favorecer el diseño de mejores estrategias de prevención y/o tratamiento de la obesidad infantil y sus comorbilidades asociadas que puedan contribuir a paliar una de las mayores preocupaciones de la Salud Pública: la obesidad infantil y la salud futura de las nuevas generaciones. Desde esta perspectiva, el hecho de que una simple

Conclusiones

lista de comprobación de los comportamientos de estilo de vida relevantes permita la detección precoz de posibles efectos perjudiciales sobre la salud cardiometabólica de los niños es cada vez más factible. Sin embargo, son necesario más estudios debido a que existe un escaso número de estudios longitudinales y ensayos clínicos rigurosos en niños y los estudios de poca potencia estadística impera.

VIII. FUTURE PERSPECTIVES

Future Perspectives

At present, childhood obesity can be considered a pandemic due to its increasing global prevalence and incidence and, regrettably, the earlier its onset, the greater the risk of comorbidities in childhood and adulthood. In this sense, prevention and treatment of childhood obesity is challenging due to the wide network of related etiological factors and although the present thesis provides evidence on certain risk factors potentially associated to this pathology, it is still insufficient for a complete understanding of the mechanisms that could trigger its onset and/or favor its prevalence. Therefore, some of the gaps that should be addressed to this purpose can be summarized as follows:

- Further long-term observational prospective and interventional studies should be conducted in populations of children from different cultural contexts assessing the associations between eating speed, and diet quality with cardiometabolic risk and including several measures of adiposity, such as FMI. It is important to highlight the importance of considering eating speed and diet quality as well as their possible interactions with other lifestyle behaviors due to their potential impact on childhood obesity, especially in early life.
- Evidence on the role of exclusive breastfeeding in certain health outcomes in children is controversial and very limited, especially regarding its association with cardiometabolic risk

factors. In order to confirm our results, further research assessing the concomitant impact of lifestyle behaviors on cardiometabolic risk, including adiposity, should consider exclusive breastfeeding as an exposure rather than as a confounder, which is the most common analysis conducted.

- To the extent possible, additional studies in children should focus lifestyle behaviors assessment on objective measures such as direct visualization of the eating pace and accelerometry for physical activity.
- It is important to establish a consensus on the definitions of eating pace measures, especially for eating speed and eating rate. Furthermore, research to improve the understanding of the mechanisms underlying the impact that these eating behaviors may have on cardiometabolic health is sorely needed. In this sense, an interesting approach that currently has scarcely been explored in children is the influence of sensory properties, as well as food composition, on the regulation of eating speed.
- Future studies should examine the role of eating speed and oral processing on glucose metabolism in children as well as their relationship with other related-hormones (e.g., insulin, GLP-1) since the results of the present thesis are consistent with evidence in adults however, in children there are no studies published to date that have assessed this association.

Future Perspectives

- Further research is warranted to confirm the association between eating speed and diet quality, clarify its direction and assess their roles in the system of central appetite and energy balance, which could improve the understanding of the potential mechanisms underlying childhood obesity and its related cardiometabolic disorders.
- In terms of recommendations for lifestyle behaviors in children, it is key to review current guidelines and update them, based on actual research, if appropriate. However, it should be noted that using recommendations rather than raw variables (e.g., screentime) could lead to misinformation if strict cutoffs for categorical variables are applied, since the risk could be unequal across participants in the same category (e.g., in a child with a reported screentime of 2.5 h/day than in another with 4h/day).
- Furthermore, other potential lifestyle-related factors should be considered for future research. For example, the type of digital media as well as the setting in which is used, and the content may lead to differences in health outcomes.
- Finally, it would be a major advance in the understanding of the mechanisms of childhood obesity if further research could confirm the results of this thesis through Omics sciences.

IX. REFERENCES

1. Alderman H, Behrman JR, Glewwe P, Fernald L, Walker S. *Evidence of Impact of Interventions on Growth and Development during Early and Middle Childhood*. The International Bank for Reconstruction and Development / The World Bank; 2017. Accessed May 6, 2022. http://www.ncbi.nlm.nih.gov/pubmed/23541370

2. Mummert A, Schoen M, Lampl M. Growth and Life Course Health Development. In: *Handbook of Life Course Health Development*. Springer International Publishing; 2018:405-429. doi:10.1007/978-3-319-47143-3_17

3. Delemarre-van de Waal HA. Environmental factors influencing growth and pubertal development. *Environ Health Perspect*. 1993;101(suppl 2):39-44. doi:10.1289/ehp.93101s239

4. Kliegman RM, Blum NJ, Shah SS, et al. *Nelson. Tratado de Pediatría*. 21st ed.; 2020.

5. Sadler TW. *Langman's Medical Embryology*. 14ª. (Wolters Kluwer, ed.).; 2019.

6. Merrick J. Child health and human development over the lifespan. *Front Public Health*. 2013;1. doi:10.3389/fpubh.2013.00001

7. Walker SP, Wachs TD, Meeks Gardner J, et al. Child development: risk factors for adverse outcomes in developing countries. *The Lancet*. 2007;369(9556):145-157. doi:10.1016/S0140-6736(07)60076-2

8. Geserick M, Vogel M, Gausche R, et al. Acceleration of BMI in Early Childhood and Risk of Sustained Obesity. *New England Journal of Medicine*. 2018;379(14):1303-1312. doi:10.1056/NEJMoa1803527

9. di Gravio C, Krishnaveni G v., Somashekara R, et al. Comparing BMI with skinfolds to estimate age at adiposity rebound and its associations with cardio-metabolic risk markers in adolescence. *Int J Obes*. 2019;43(4):683-690. doi:10.1038/s41366-018-0144-8

10. González L, Corvalán C, Pereira A, Kain J, Garmendia ML, Uauy R. Early adiposity rebound is associated with metabolic risk in 7-year-old children. *Int J Obes*. 2014;38(10):1299-1304. doi:10.1038/ijo.2014.97

11. Sumińska M, Podgórski R, Bogusz-Górna K, Skowrońska B, Mazur A, Fichna M. Historical and cultural aspects of obesity: From a symbol of wealth and prosperity to the epidemic of the 21st century. *Obesity Reviews*. 2022;23(6):e13440. doi:10.1111/obr.13440

12. World Health Organization (WHO). Obesity and overweight. Published 2020. Accessed November 13, 2020. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight

13. Wärnberg J, Nova E, Moreno LA, et al. Inflammatory proteins are related to total and abdominal adiposity in a healthy adolescent population: the AVENA Study. *Am J Clin Nutr*. 2006;84(3):505-512. doi:10.1093/ajcn/84.3.505

14. Nappo A, Iacoviello L, Fraterman A, et al. High-sensitivity C-reactive protein is a predictive factor of adiposity in children: Results of the identification and prevention of dietary-and lifestyle-induced health effects in children and infants (IDEFICS) study. *J Am Heart Assoc*. 2013;2(3). doi:10.1161/JAHA.113.000101

15. Lobstein T, Brinsden H, Neveux M, et al. *World Obesity Atlas 2022*.; 2022. Accessed August 1, 2022.

https://www.worldobesity.org/resources/resource-library/world-obesity-atlas-2022

16. World Health Organization. Regional Office for Europe. *WHO European Regional Obesity : Report 2022*.

17. Agencia Española de Seguridad Alimentaria y Nutrición. *Surveillance Study on Nutrition, Physical Activity, Child Development and Obesity (ALADINO).*; 2020. Accessed August 1, 2022. https://www.aesan.gob.es/AECOSAN/docs/documentos/nutricion/observatorio/Brief_report_ALADINO_2019_NAOS.pdf

18. Kumar S, Kelly AS. Review of Childhood Obesity: From Epidemiology, Etiology, and Comorbidities to Clinical Assessment and Treatment. *Mayo Clin Proc*. 2017;92(2):251-265. doi:10.1016/j.mayocp.2016.09.017

19. Cypess AM. Reassessing Human Adipose Tissue. Ingelfinger JR, ed. *New England Journal of Medicine*. 2022;386(8):768-779. doi:10.1056/NEJMra2032804

20. Sutaria S, Devakumar D, Yasuda SS, Das S, Saxena S. Is obesity associated with depression in children? Systematic review and meta-analysis. *Arch Dis Child*. 2019;104(1):64-74. doi:10.1136/archdischild-2017-314608

21. Kumar S, Kaufman T. Childhood obesity. *Panminerva Med*. 2018;60(4):200-212. doi:10.23736/S0031-0808.18.03557-7

22. Horesh A, Tsur AM, Bardugo A, Twig G. Adolescent and Childhood Obesity and Excess Morbidity and Mortality in Young Adulthood—a

Systematic Review. *Curr Obes Rep*. 2021;10(3):301-310. doi:10.1007/s13679-021-00439-9

23. Adair LS, Fall CHD, Osmond C, et al. Associations of linear growth and relative weight gain during early life with adult health and human capital in countries of low and middle income: findings from five birth cohort studies. *Lancet*. 2013;382(9891):525-534. doi:10.1016/S0140-6736(13)60103-8

24. Ward ZJ, Long MW, Resch SC, Giles CM, Cradock AL, Gortmaker SL. Simulation of Growth Trajectories of Childhood Obesity into Adulthood. *New England Journal of Medicine*. 2017;377(22):2145-2153. doi:10.1056/NEJMoa1703860

25. Cunningham SA, Kramer MR, Narayan KMV. Incidence of Childhood Obesity in the United States. *New England Journal of Medicine*. 2014;370(5):403-411. doi:10.1056/NEJMoa1309753

26. Juhola J, Magnussen CG, Viikari JSA, et al. Tracking of Serum Lipid Levels, Blood Pressure, and Body Mass Index from Childhood to Adulthood: The Cardiovascular Risk in Young Finns Study. *J Pediatr*. 2011;159(4):584-590. doi:10.1016/j.jpeds.2011.03.021

27. Tirosh A, Shai I, Afek A, et al. Adolescent BMI Trajectory and Risk of Diabetes versus Coronary Disease. *N Engl J Med*. 2011;364(14):1315. doi:10.1056/NEJMOA1006992

28. Jacobs DR, Woo JG, Sinaiko AR, et al. Childhood Cardiovascular Risk Factors and Adult Cardiovascular Events. *New England Journal of Medicine*. 2022;386(20):1877-1888. doi:10.1056/NEJMoa2109191

29. Bjerregaard LG, Jensen BW, Ängquist L, Osler M, Sørensen TIA, Baker JL. Change in Overweight from Childhood to Early Adulthood and Risk of Type 2 Diabetes. *N Engl J Med*. 2018;378(14):1302-1312. doi:10.1056/NEJMoa1713231

30. Weihrauch-Blüher S, Schwarz P, Klusmann JH. Childhood obesity: increased risk for cardiometabolic disease and cancer in adulthood. *Metabolism*. 2019;92:147-152. doi:10.1016/j.metabol.2018.12.001

31. Eisenmann JC, Laurson KR, DuBose KD, Smith BK, Donnelly JE. Construct validity of a continuous metabolic syndrome score in children. *Diabetol Metab Syndr*. 2010;2(1):8. doi:10.1186/1758-5996-2-8

32. Li X, Keown-Stoneman CDG, Lebovic G, et al. The association between body mass index trajectories and cardiometabolic risk in young children. *Pediatr Obes*. 2020;15(8). doi:10.1111/ijpo.12633

33. Carsley SE, Anderson LN, Plumptre L, et al. Severe Obesity, Obesity, and Cardiometabolic Risk in Children 0 to 6 Years of Age. *Childhood Obesity*. 2017;13(5):415-424. doi:10.1089/chi.2017.0004

34. Lund MA v, Thostrup AH, Frithioff-Bøjsøe C, et al. Low-grade inflammation independently associates with cardiometabolic risk in children with overweight/obesity. *Nutr Metab Cardiovasc Dis*. 2020;30(9):1544-1553. doi:10.1016/j.numecd.2020.04.024

35. Maximova K, Kuhle S, Davidson Z, Fung C, Veugelers PJ. Cardiovascular risk-factor profiles of normal and overweight children and adolescents: insights from the Canadian Health Measures Survey. *Can J Cardiol*. 2013;29(8):976-982. doi:10.1016/j.cjca.2012.08.013

36. Kelishadi R, Mirmoghtadaee P, Najafi H, Keikha M. Systematic review on the association of abdominal obesity in children and adolescents with cardio-metabolic risk factors. *J Res Med Sci*. 2015;20(3):294-307. http://www.ncbi.nlm.nih.gov/pubmed/26109978

37. Kamel M, Smith BT, Wahi G, Carsley S, Birken CS, Anderson LN. Continuous cardiometabolic risk score definitions in early childhood: a scoping review. *Obesity Reviews*. 2018;19(12):1688-1699. doi:10.1111/obr.12748

38. Kelly AS, Steinberger J, Jacobs DR, Hong CP, Moran A, Sinaiko AR. Predicting cardiovascular risk in young adulthood from the metabolic syndrome, its component risk factors, and a cluster score in childhood. *International Journal of Pediatric Obesity*. 2011;6(2-2):e283-e289. doi:10.3109/17477166.2010.528765

39. Anderson LN, Lebovic G, Hamilton J, et al. Body Mass Index, Waist Circumference, and the Clustering of Cardiometabolic Risk Factors in Early Childhood. *Paediatr Perinat Epidemiol*. 2016;30(2):160-170. doi:10.1111/ppe.12268

40. Muñoz-Hernando J, Escribano J, Ferré N, et al. Usefulness of the waist-to-height ratio for predicting cardiometabolic risk in children and its suggested boundary values. *Clinical Nutrition*. 2022;41(2):508-516. doi:10.1016/j.clnu.2021.12.008

41. MacPherson M, de Groh M, Loukine L, Prud'homme D, Dubois L. Prevalence of metabolic syndrome and its risk factors in Canadian children and adolescents: Canadian Health Measures Survey Cycle 1 (2007–2009) and Cycle 2 (2009–2011). *Health Promotion and Chronic*

Disease Prevention in Canada. 2016;36(2):32-40. doi:10.24095/hpcdp.36.2.03

42. Freedman DS, Khan LK, Dietz WH, Srinivasan SR, Berenson GS. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa Heart Study. *Pediatrics*. 2001;108(3):712-718. doi:10.1542/peds.108.3.712

43. Alberti KGMM, Eckel RH, Grundy SM, et al. Harmonizing the Metabolic Syndrome. *Circulation*. 2009;120(16):1640-1645. doi:10.1161/CIRCULATIONAHA.109.192644

44. Steinberger J, Daniels SR, Eckel RH, et al. Progress and challenges in metabolic syndrome in children and adolescents: a scientific statement from the American Heart Association Atherosclerosis, Hypertension, and Obesity in the Young Committee of the Council on Cardiovascular Disease in the Young; Council on Cardiovascular Nursing; and Council on Nutrition, Physical Activity, and Metabolism. *Circulation*. 2009;119(4):628-647. doi:10.1161/CIRCULATIONAHA.108.191394

45. Noubiap JJ, Nansseu JR, Lontchi-Yimagou E, et al. Global, regional, and country estimates of metabolic syndrome burden in children and adolescents in 2020: a systematic review and modelling analysis. *Lancet Child Adolesc Health*. 2022;6(3):158-170. doi:10.1016/S2352-4642(21)00374-6

46. Suárez Carmona W, Sánchez Oliver A, González Jurado J. Fisiopatología de la obesidad: Perspectiva actual. *Revista chilena de nutrición*. 2017;44(3):226-233. doi:10.4067/S0717-75182017000300226

47. Gjermeni E, Kirstein AS, Kolbig F, et al. Obesity–An Update on the Basic Pathophysiology and Review of Recent Therapeutic Advances. *Biomolecules*. 2021;11(10). doi:10.3390/BIOM11101426

48. Fernández-Sánchez A, Madrigal-Santillán E, Bautista M, et al. Inflammation, oxidative stress, and obesity. *Int J Mol Sci*. 2011;12(5):3117-3132. doi:10.3390/ijms12053117

49. Sun K, Kusminski CM, Scherer PE. Adipose tissue remodeling and obesity. *Journal of Clinical Investigation*. 2011;121(6):2094-2101. doi:10.1172/JCI45887

50. Deng Y, Scherer PE. Adipokines as novel biomarkers and regulators of the metabolic syndrome. *Ann N Y Acad Sci*. 2010;1212:E1-E19. doi:10.1111/j.1749-6632.2010.05875.x

51. Tong Y, Xu S, Huang L, Chen C. Obesity and insulin resistance: Pathophysiology and treatment. *Drug Discov Today*. 2022;27(3):822-830. doi:10.1016/j.drudis.2021.11.001

52. Poirier P, Giles TD, Bray GA, et al. Obesity and Cardiovascular Disease: Pathophysiology, Evaluation, and Effect of Weight Loss. *Circulation*. 2006;113(6):898-918. doi:10.1161/CIRCULATIONAHA.106.171016

53. Kotnik P, Fischer Posovszky P, Wabitsch M. Endocrine and Metabolic Effects of Adipose Tissue in Children and Adolescents. *Zdr Varst*. 2015;54(2):131-138. doi:10.1515/sjph-2015-0020

54. Gauthier KI, Krajicek MJ. Obesogenic environment: A concept analysis and pediatric perspective. *Journal for Specialists in Pediatric Nursing*. 2013;18(3):202-210. doi:10.1111/jspn.12027

55. Swinburn B, Egger G, Raza F. Dissecting Obesogenic Environments: The Development and Application of a Framework for Identifying and Prioritizing Environmental Interventions for Obesity. *Prev Med (Baltim)*. 1999;29(6):563-570. doi:10.1006/pmed.1999.0585

56. Lopez-Gonzalez D, Partida-Gaytán A, Wells JC, et al. Obesogenic Lifestyle and Its Influence on Adiposity in Children and Adolescents, Evidence from Mexico. *Nutrients 2020, Vol 12, Page 819*. 2020;12(3):819. doi:10.3390/NU12030819

57. Llewellyn C, Wardle J. Behavioral susceptibility to obesity: Gene–environment interplay in the development of weight. *Physiol Behav*. 2015;152:494-501. doi:10.1016/j.physbeh.2015.07.006

58. Ziauddeen N, Roderick PJ, Macklon NS, Alwan NA. Predicting childhood overweight and obesity using maternal and early life risk factors: a systematic review. *Obesity Reviews*. 2018;19(3):302-312. doi:10.1111/obr.12640

59. Bawaked RA, Gomez SF, Homs C, et al. Association of eating behaviors, lifestyle, and maternal education with adherence to the Mediterranean diet in Spanish children. *Appetite*. 2018;130:279-285. doi:10.1016/j.appet.2018.08.024

60. Solmi M, Köhler CA, Stubbs B, et al. Environmental risk factors and nonpharmacological and nonsurgical interventions for obesity: An umbrella review of meta-analyses of cohort studies and randomized controlled trials. *Eur J Clin Invest*. 2018;48(12):e12982. doi:10.1111/eci.12982

61. Price RA. Genetics of Human Obesity. *Annals of Behavioral Medicine*. 1987;9(1):9-14. doi:10.1207/s15324796abm0901_2

62. Bouchard C, Perusse L. Heredity and Body Fat. *Annu Rev Nutr*. 1988;8(1):259-277. doi:10.1146/annurev.nu.08.070188.001355

63. Frayling TM, Timpson NJ, Weedon MN, et al. A common variant in the FTO gene is associated with body mass index and predisposes to childhood and adult obesity. *Science*. 2007;316(5826):889-894. doi:10.1126/science.1141634

64. Locke AE, Kahali B, Berndt SI, et al. Genetic studies of body mass index yield new insights for obesity biology. *Nature*. 2015;518(7538):197-206. doi:10.1038/nature14177

65. Kraja AT, Vaidya D, Pankow JS, et al. A Bivariate Genome-Wide Approach to Metabolic Syndrome. *Diabetes*. 2011;60(4):1329-1339. doi:10.2337/db10-1011

66. Wu Y, Perng W, Peterson KE. Precision Nutrition and Childhood Obesity: A Scoping Review. *Metabolites*. 2020;10(6):235. doi:10.3390/metabo10060235

67. Silventoinen K, Jelenkovic A, Sund R, et al. Genetic and environmental effects on body mass index from infancy to the onset of adulthood: an individual-based pooled analysis of 45 twin cohorts participating in the COllaborative project of Development of Anthropometrical measures in Twins (CODATwins) study. *Am J Clin Nutr*. 2016;104(2):371-379. doi:10.3945/ajcn.116.130252

68. Schrempft S, van Jaarsveld CHM, Fisher A, et al. Variation in the Heritability of Child Body Mass Index by Obesogenic Home

Environment. *JAMA Pediatr*. 2018;172(12):1153-1160. doi:10.1001/jamapediatrics.2018.1508

69. Pineros-Leano M, Grafft N, Aguayo L. Childhood obesity risk factors by race and ethnicity. *Obesity (Silver Spring)*. 2022;30(8):1670-1680. doi:10.1002/oby.23500

70. Song M, Zheng Y, Qi L, Hu FB, Chan AT, Giovannucci EL. Longitudinal Analysis of Genetic Susceptibility and BMI Throughout Adult Life. *Diabetes*. 2018;67(2):248-255. doi:10.2337/db17-1156

71. Hammami N, Chaurasia A, Bigelow P, Leatherdale ST. Gender differences in the longitudinal association between multilevel latent classes of chronic disease risk behaviours and body mass index in adolescents. *Health Promotion and Chronic Disease Prevention in Canada*. 2020;40(9):259-266. doi:10.24095/hpcdp.40.9.01

72. Onat A, Karadeniz Y, Tusun E, Yüksel H, Kaya A. Advances in understanding gender difference in cardiometabolic disease risk. *Expert Rev Cardiovasc Ther*. 2016;14(4):513-523. doi:10.1586/14779072.2016.1150782

73. Heitkamp M, Siegrist M, Molnos S, et al. Obesity Genes and Weight Loss during Lifestyle Intervention in Children with Obesity. *JAMA Pediatr*. 2021;175(1). doi:10.1001/jamapediatrics.2020.5142

74. Jebeile H, Kelly AS, O'Malley G, Baur LA. Obesity in children and adolescents: epidemiology, causes, assessment, and management. *Lancet Diabetes Endocrinol*. 2022;10(5):351-365. doi:10.1016/S2213-8587(22)00047-X

75. Ford ND, Behrman JR, Hoddinott JF, et al. Exposure to improved nutrition from conception to age 2 years and adult cardiometabolic disease risk: a modelling study. *Lancet Glob Health*. 2018;6(8):e875-e884. doi:10.1016/S2214-109X(18)30231-6

76. Giles LC, Whitrow MJ, Davies MJ, Davies CE, Rumbold AR, Moore VM. Growth trajectories in early childhood, their relationship with antenatal and postnatal factors, and development of obesity by age 9 years: results from an Australian birth cohort study. *Int J Obes*. 2015;39(7):1049-1056. doi:10.1038/ijo.2015.42

77. Nyasordzi J, Conrad J, Goletzke J, et al. Early life factors and their relevance for markers of cardiometabolic risk in early adulthood. *Nutrition, Metabolism and Cardiovascular Diseases*. 2021;31(7):2109-2121. doi:10.1016/j.numecd.2021.03.024

78. Navarro P, Mehegan J, Murrin CM, Kelleher CC, Phillips CM. Associations between a maternal healthy lifestyle score and adverse offspring birth outcomes and childhood obesity in the Lifeways Cross-Generation Cohort Study. *International Journal of Obesity* . 2020;44(11):2213-2224. doi:10.1038/s41366-020-00652-x

79. Mourtakos SP, Tambalis KD, Panagiotakos DB, et al. Maternal lifestyle characteristics during pregnancy, and the risk of obesity in the offspring: a study of 5,125 children. *BMC Pregnancy Childbirth*. 2015;15(1):66. doi:10.1186/s12884-015-0498-z

80. Dhana K, Zong G, Yuan C, et al. Lifestyle of women before pregnancy and the risk of offspring obesity during childhood through early adulthood. *Int J Obes (Lond)*. 2018;42(7):1275. doi:10.1038/S41366-018-0052-Y

81. Burnett AJ, Lamb KE, McCann J, Worsley A, Lacy KE. Parenting styles and the dietary intake of pre-school children: a systematic review. *Psychol Health*. 2020;35(11):1326-1345. doi:10.1080/08870446.2020.1743842

82. Cadenas-Sanchez C, Henriksson P, Henriksson H, et al. Parental body mass index and its association with body composition, physical fitness and lifestyle factors in their 4-year-old children: results from the MINISTOP trial. *Eur J Clin Nutr*. 2017;71(10):1200-1205. doi:10.1038/ejcn.2017.62

83. Hurley KL, Pallan MJ, Lancashire ER, et al. An exploration of the longitudinal relation between parental feeding practices and child anthropometric adiposity measures from the West Midlands Active Lifestyle and Healthy Eating in Schoolchildren (WAVES) Study. *American Journal of Clinical Nutrition*. 2018;108(6):1316-1323. doi:10.1093/ajcn/nqy241

84. Tenjin K, Sekine M, Yamada M, Tatsuse T. Relationship Between Parental Lifestyle and Dietary Habits of Children: A Cross-Sectional Study. *J Epidemiol*. 2020;30(6):253-259. doi:10.2188/jea.JE20190015

85. Pathirana MM, Lassi ZS, Roberts CT, Andraweera PH. Cardiovascular risk factors in offspring exposed to gestational diabetes mellitus *in utero* : systematic review and meta-analysis. *J Dev Orig Health Dis*. 2020;11(6):599-616. doi:10.1017/S2040174419000850

86. Fernández-Barrés S, Romaguera D, Valvi D, et al. Mediterranean dietary pattern in pregnant women and offspring risk of overweight and abdominal obesity in early childhood: The INMA birth cohort study. *Pediatr Obes*. 2016;11(6):491-499. doi:10.1111/ijpo.12092

87. Fernández-Barrés S, Vrijheid M, Manzano-Salgado CB, et al. The Association of Mediterranean Diet during Pregnancy with Longitudinal Body Mass Index Trajectories and Cardiometabolic Risk in Early Childhood. *Journal of Pediatrics*. 2019;206:119-127.e6. doi:10.1016/j.jpeds.2018.10.005

88. Kanellopoulou A, Giannakopoulou SP, Notara V, et al. The association between adherence to the Mediterranean diet and childhood obesity; the role of family structure: Results from an epidemiological study in 1728 Greek students. *Nutr Health*. 2021;27(1):39-47. doi:10.1177/0260106020952600

89. Martín-Calvo N, Goni L, Tur JA, Martínez JA. Low birth weight and small for gestational age are associated with complications of childhood and adolescence obesity: Systematic review and meta-analysis. *Obesity Reviews*. 2022;23(S1). doi:10.1111/obr.13380

90. Ashtree DN, McGuinness AJ, Plummer M, Sun C, Craig JM, Scurrah KJ. Developmental origins of cardiometabolic health outcomes in twins: A systematic review and meta-analysis. *Nutr Metab Cardiovasc Dis*. 2020;30(10):1609-1621. doi:10.1016/j.numecd.2020.06.010

91. Martín-Calvo N, Goni L, Tur JA, Martínez JA. Low birth weight and small for gestational age are associated with complications of childhood and adolescence obesity: Systematic review and meta-analysis. *Obesity Reviews*. 2022;23(S1):e13380. doi:10.1111/OBR.13380

92. Yakoob MY, Lo CW. Nutrition (Micronutrients) in Child Growth and Development: A Systematic Review on Current Evidence, Recommendations and Opportunities for Further Research. *Journal of*

Developmental and Behavioral Pediatrics. 2017;38(8):665-679. doi:10.1097/DBP.0000000000000482

93. Horta BL, Loret de Mola C, Victora CG. Long-term consequences of breastfeeding on cholesterol, obesity, systolic blood pressure and type 2 diabetes: a systematic review and meta-analysis. *Acta Paediatr*. 2015;104:30-37. doi:10.1111/apa.13133

94. Vinke PC, Tigelaar C, Küpers LK, Corpeleijn E. The Role of Children's Dietary Pattern and Physical Activity in the Association Between Breastfeeding and BMI at Age 5: The GECKO Drenthe Cohort. *Matern Child Health J*. 2021;25(2):338-348. doi:10.1007/s10995-020-03063-6

95. Patro-Gołąb B, Zalewski BM, Polaczek A, Szajewska H. Duration of Breastfeeding and Early Growth: A Systematic Review of Current Evidence. *Breastfeeding Medicine*. 2019;14(4):218-229. doi:10.1089/bfm.2018.0187

96. Kheir F, Feeley N, Maximova K, Drapeau V, Henderson M, van Hulst A. Breastfeeding duration in infancy and dietary intake in childhood and adolescence. *Appetite*. 2021;158:104999. doi:10.1016/j.appet.2020.104999

97. D'Hollander CJ, Keown-Stoneman CDG, Birken CS, et al. Timing of Introduction to Solid Food, Growth, and Nutrition Risk in Later Childhood. *J Pediatr*. 2022;240:102-109.e3. doi:10.1016/j.jpeds.2021.08.076

98. Switkowski KM, Aris IM, Gingras V, Oken E, Young JG. Estimated causal effects of complementary feeding behaviors on early childhood diet

quality in a US cohort. *Am J Clin Nutr*. 2022;115(4):1105-1114. doi:10.1093/ajcn/nqac003

99. Nigg CR, Ul Anwar MM, Braun K, et al. A Review of Promising Multicomponent Environmental Child Obesity Prevention Intervention Strategies by the Children's Healthy Living Program. *J Environ Health*. 2016;79(3):18-26. http://www.ncbi.nlm.nih.gov/pubmed/29120137

100. Yang Z, Phung H, Hughes AM, Sherwood S, Harper E, Kelly P. Trends in overweight and obesity by socioeconomic status in Year 6 school children, Australian Capital Territory, 2006-2018. *BMC Public Health*. 2019;19(1). doi:10.1186/S12889-019-7645-9/FIGURES/4

101. Lioret S, Campbell KJ, McNaughton SA, et al. Lifestyle Patterns Begin in Early Childhood, Persist and Are Socioeconomically Patterned, Confirming the Importance of Early Life Interventions. *Nutrients*. 2020;12(3):724. doi:10.3390/nu12030724

102. D'Souza NJ, Kuswara K, Zheng M, et al. A systematic review of lifestyle patterns and their association with adiposity in children aged 5–12 years. *Obesity Reviews*. 2020;21(8). doi:10.1111/obr.13029

103. St Pierre C, ver Ploeg M, Dietz WH, et al. Food Insecurity and Childhood Obesity: A Systematic Review. *Pediatrics*. 2022;150(1). doi:10.1542/peds.2021-055571

104. Suga H. Household food unavailability due to financial constraints affects the nutrient intake of children. *Eur J Public Health*. 2019;29(5):816-820. doi:10.1093/eurpub/cky263

105. Marcos-Pasero H, Aguilar-Aguilar E, de la Iglesia R, et al. “GENYAL” Study to Childhood Obesity Prevention: Methodology and Preliminary Results. *Front Nutr*. 2022;9:777384. doi:10.3389/fnut.2022.777384

106. Hilpert M, Brockmeier K, Dordel S, et al. Sociocultural Influence on Obesity and Lifestyle in Children: A Study of Daily Activities, Leisure Time Behavior, Motor Skills, and Weight Status. *Obes Facts*. 2017;10(3):168-178. doi:10.1159/000464105

107. Ghenadenik AE, Kakinami L, van Hulst A, Henderson M, Barnett TA. Neighbourhoods and obesity: A prospective study of characteristics of the built environment and their association with adiposity outcomes in children in Montreal, Canada. *Prev Med (Baltim)*. 2018;111:35-40. doi:10.1016/j.ypmed.2018.02.018

108. van Hulst A, Roy-Gagnon MH, Gauvin L, Kestens Y, Henderson M, Barnett TA. Identifying risk profiles for childhood obesity using recursive partitioning based on individual, familial, and neighborhood environment factors. *International Journal of Behavioral Nutrition and Physical Activity*. 2015;12(1):17. doi:10.1186/s12966-015-0175-7

109. Bailey LC, Forrest CB, Zhang P, Richards TM, Livshits A, DeRusso PA. Association of Antibiotics in Infancy With Early Childhood Obesity. *JAMA Pediatr*. 2014;168(11):1063-1069. doi:10.1001/JAMAPEDIATRICS.2014.1539

110. Plaza-Díaz J, Molina-Montes E, Soto-Méndez MJ, et al. Clustering of Dietary Patterns and Lifestyles Among Spanish Children in the EsNuPI Study. *Nutrients*. 2020;12(9):1-25. doi:10.3390/NU12092536

111. Bawaked RA, Fernández-Barrés S, Navarrete-Muñoz EM, et al. Impact of lifestyle behaviors in early childhood on obesity and cardiometabolic risk in children: Results from the Spanish INMA birth cohort study. *Pediatr Obes*. 2020;15(3):e12590. doi:10.1111/ijpo.12590

112. Bae JH, Lee H. The effect of diet, exercise, and lifestyle intervention on childhood obesity: A network meta-analysis. *Clinical Nutrition*. 2021;40(5):3062-3072. doi:10.1016/j.clnu.2020.11.006

113. Haapala EA, Väistö J, Ihalainen JK, et al. Associations of physical activity, sedentary time, and diet quality with biomarkers of inflammation in children. *Eur J Sport Sci*. 2022;22(6):906-915. doi:10.1080/17461391.2021.1892830

114. Fang J, Gong C, Wan Y, Xu Y, Tao F, Sun Y. Polygenic risk, adherence to a healthy lifestyle, and childhood obesity. *Pediatr Obes*. 2019;14(4). doi:10.1111/ijpo.12489

115. Garcidueñas-Fimbres TE, Paz-Graniel I, Gómez-Martínez C, et al. Associations Between Eating Speed, Diet Quality, Adiposity, and Cardiometabolic Risk Factors. *J Pediatr*. 2023;252:31-39.e1. doi:10.1016/j.jpeds.2022.08.024

116. Hejazi K, Ferrari F. Effects of Physical Exercise on Cardiometabolic Biomarkers and Inflammatory Markers in Children: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. *Biol Res Nurs*. 2022;24(4):519-529. doi:10.1177/10998004221099573

117. Carson V, Hunter S, Kuzik N, et al. Systematic review of sedentary behaviour and health indicators in school-aged children and youth: an

update. *Applied Physiology, Nutrition, and Metabolism*. 2016;41(6 (Suppl. 3)):S240-S265. doi:10.1139/apnm-2015-0630

118. Plumptre L, Anderson LN, Chen Y, et al. Longitudinal Analysis of Sleep Duration and Cardiometabolic Risk in Young Children. *Childhood Obesity*. 2017;13(4):291-299. doi:10.1089/chi.2016.0279

119. Metallinos-Katsaras ES, Freedson PS, Fulton JE, Sherry B. The association between an objective measure of physical activity and weight status in preschoolers. *Obesity*. 2007;15(3):686-694. doi:10.1038/OBY.2007.571

120. Pangrazi RP, Beighle A, Vehige T, Vack C. Impact of Promoting Lifestyle Activity for Youth (PLAY) on Children's Physical Activity. *Journal of School Health*. 2003;73(8):317-321. doi:10.1111/j.1746-1561.2003.tb06589.x

121. Laguna M, Ruiz JR, Lara MT, Aznar S. Recommended levels of physical activity to avoid adiposity in Spanish children. *Pediatr Obes*. 2013;8(1):62-69. doi:10.1111/J.2047-6310.2012.00086.X

122. Sirico F, Bianco A, D'Alicandro G, et al. Effects of Physical Exercise on Adiponectin, Leptin, and Inflammatory Markers in Childhood Obesity: Systematic Review and Meta-Analysis. *Childhood Obesity*. 2018;14(4):207-217. doi:10.1089/chi.2017.0269

123. Cao Y, Zhu L, Liu J. Effects of aerobic exercise on obese children with metabolic syndrome: a systematic review and meta-analysis. *Journal of Pediatric Endocrinology and Metabolism*. 2021;34(9):1069-1079. doi:10.1515/jpem-2021-0295

124. de Lima TR, Martins PC, Torre GL, Mannocci A, Silva KS, Silva DAS. Association between muscle strength and risk factors for metabolic syndrome in children and adolescents: a systematic review. *J Pediatr Endocrinol Metab*. 2021;34(1):1-12. doi:10.1515/jpem-2020-0135

125. World Health Organization. *WHO Guidelines on Physical Activity and Sedentary Behaviour.*; 2020. https://www.who.int/publications/i/item/9789240015128

126. World Health Organization. *Guidelines on Physical Activity, Sedentary Behaviour and Sleep for Children under 5 Years of Age.*; 2019. https://apps.who.int/iris/handle/10665/311664

127. Peplies J, Börnhorst C, Günther K, et al. Longitudinal associations of lifestyle factors and weight status with insulin resistance (HOMA-IR) in preadolescent children: the large prospective cohort study IDEFICS. *International Journal of Behavioral Nutrition and Physical Activity 2016 13:1*. 2016;13(1):1-12. doi:10.1186/S12966-016-0424-4

128. Chen T, Lin J, Lin Y, et al. Effects of aerobic exercise and resistance exercise on physical indexes and cardiovascular risk factors in obese and overweight school-age children: A systematic review and meta-analysis. Hsu YP, ed. *PLoS One*. 2021;16(9):e0257150. doi:10.1371/journal.pone.0257150

129. Lampropoulou M, Chaini M, Rigopoulos N, Evangeliou A, Papadopoulou-Legbelou K, Koutelidakis AE. Association between Serum Lipid Levels in Greek Children with Dyslipidemia and Mediterranean Diet Adherence, Dietary Habits, Lifestyle and Family Socioeconomic Factors. *Nutrients 2020, Vol 12, Page 1600*. 2020;12(6):1600. doi:10.3390/NU12061600

130. Cao M, Tang Y, Li S, Zou Y. Effects of High-Intensity Interval Training and Moderate-Intensity Continuous Training on Cardiometabolic Risk Factors in Overweight and Obesity Children and Adolescents: A Meta-Analysis of Randomized Controlled Trials. *Int J Environ Res Public Health*. 2021;18(22):11905. doi:10.3390/ijerph182211905

131. Duval C, Rouillier MA, Rabasa-Lhoret R, Karelis AD. High intensity exercise: Can it protect you from a fast food diet? *Nutrients*. 2017;9(9). doi:10.3390/nu9090943

132. Tambalis KD, Panagiotakos DB, Psarra G, Sidossis LS. Concomitant Associations between Lifestyle Characteristics and Physical Activity Status in Children and Adolescents. *J Res Health Sci*. 2019;19(1):e00439. http://www.ncbi.nlm.nih.gov/pubmed/31133628

133. Stiglic N, Viner RM. Effects of screentime on the health and well-being of children and adolescents: a systematic review of reviews. *BMJ Open*. 2019;9(1):e023191. doi:10.1136/bmjopen-2018-023191

134. Bar-On ME, Broughton DD, Buttross S, et al. Children, Adolescents, and Television. *Pediatrics*. 2001;107(2):423-426. doi:10.1542/peds.107.2.423

135. Wilkie HJ, Standage M, Gillison FB, Cumming SP, Katzmarzyk PT. Multiple lifestyle behaviours and overweight and obesity among children aged 9–11 years: results from the UK site of the International Study of Childhood Obesity, Lifestyle and the Environment. *BMJ Open*. 2016;6(2):e010677. doi:10.1136/BMJOPEN-2015-010677

136. Jamnik J, Keown-Stoneman C, Eny KM, Maguire JL, Birken CS. Mealtime media use and cardiometabolic risk in children. *Public Health Nutr*. 2022;25(3):670-679. doi:10.1017/S1368980020003821

137. Falbe J, Rosner B, Willett WC, Sonneville KR, Hu FB, Field AE. Adiposity and different types of screen time. *Pediatrics*. 2013;132(6):e1497-505. doi:10.1542/peds.2013-0887

138. Wachira LJM, Muthuri SK, Ochola SA, Onywera VO, Tremblay MS. Screen-based sedentary behaviour and adiposity among school children: Results from international study of childhood obesity, lifestyle and the environment (iscole) - Kenya. *PLoS One*. 2018;13(6). doi:10.1371/journal.pone.0199790

139. Avery A, Anderson C, McCullough F. Associations between children's diet quality and watching television during meal or snack consumption: A systematic review. *Matern Child Nutr*. 2017;13(4). doi:10.1111/MCN.12428

140. Ghobadi S, Hassanzadeh-Rostami Z, Salehi-Marzijarani M, et al. Association of eating while television viewing and overweight/obesity among children and adolescents: a systematic review and meta-analysis of observational studies. *Obesity Reviews*. 2018;19(3):313-320. doi:10.1111/obr.12637

141. Tabares-Tabares M, Moreno Aznar LA, Aguilera-Cervantes VG, León-Landa E, López-Espinoza A. Screen use during food consumption: Does it cause increased food intake? A systematic review. *Appetite*. 2022;171:105928. doi:10.1016/j.appet.2022.105928

142. Robinson CA, Domoff SE, Kasper N, Peterson KE, Miller AL. The healthfulness of children's meals when multiple media and devices are present. *Appetite*. 2022;169:105800. doi:10.1016/j.appet.2021.105800

143. Zimmerman FJ, Bell JF. Associations of television content type and obesity in children. *Am J Public Health*. 2010;100(2):334-340. doi:10.2105/AJPH.2008.155119

144. Campos D, Escudero-Marín M, Snitman CM, et al. The Nutritional Profile of Food Advertising for School-Aged Children via Television: A Longitudinal Approach. *Children*. 2020;7(11):230. doi:10.3390/children7110230

145. Chaput JP, Gray CE, Poitras VJ, et al. Systematic review of the relationships between sleep duration and health indicators in school-aged children and youth. *Applied Physiology, Nutrition, and Metabolism*. 2016;41(6 (Suppl. 3)):S266-S282. doi:10.1139/apnm-2015-0627

146. Liu J, Ji X, Pitt S, et al. Childhood sleep: physical, cognitive, and behavioral consequences and implications. *World Journal of Pediatrics*. Published online November 23, 2022:1. doi:10.1007/s12519-022-00647-w

147. Matricciani L, Paquet C, Galland B, Short M, Olds T. Children's sleep and health: A meta-review. *Sleep Med Rev*. 2019;46:136-150. doi:10.1016/j.smrv.2019.04.011

148. Hirshkowitz M, Whiton K, Albert SM, et al. National Sleep Foundation's sleep time duration recommendations: methodology and results

summary. *Sleep Health*. 2015;1(1):40-43. doi:10.1016/j.sleh.2014.12.010

149. Ohayon M, Wickwire EM, Hirshkowitz M, et al. National Sleep Foundation's sleep quality recommendations: first report. *Sleep Health*. 2017;3(1):6-19. doi:10.1016/j.sleh.2016.11.006

150. Schmied EA, Full KM, Lin SF, Gregorio-Pascual P, Ayala GX. Sleep health among U.S. Hispanic/Latinx children: An examination of correlates of meeting sleep duration recommendations. *Sleep Health*. 2022;8(6):615-619. doi:10.1016/j.sleh.2022.07.002

151. Newton AT, Honaker SM, Reid GJ. Risk and protective factors and processes for behavioral sleep problems among preschool and early school-aged children: A systematic review. *Sleep Med Rev*. 2020;52:101303. doi:10.1016/j.smrv.2020.101303

152. Glasgow TE, Adams EL, Ksinan A, et al. Sleep onset, duration, or regularity: which matters most for child adiposity outcomes? *Int J Obes*. Published online May 12, 2022:1-8. doi:10.1038/s41366-022-01140-0

153. Felső R, Lohner S, Hollódy K, Erhardt É, Molnár D. Relationship between sleep duration and childhood obesity: Systematic review including the potential underlying mechanisms. *Nutrition, Metabolism and Cardiovascular Diseases*. 2017;27(9):751-761. doi:10.1016/j.numecd.2017.07.008

154. Wu Y, Gong Q, Zou Z, Li H, Zhang X. Short sleep duration and obesity among children: A systematic review and meta-analysis of prospective

studies. *Obes Res Clin Pract*. 2017;11(2):140-150. doi:10.1016/j.orcp.2016.05.005

155. Li L, Zhang S, Huang Y, Chen K. Sleep duration and obesity in children: A systematic review and meta-analysis of prospective cohort studies. *J Paediatr Child Health*. 2017;53(4):378-385. doi:10.1111/jpc.13434

156. Miller MA, Kruisbrink M, Wallace J, Ji C, Cappuccio FP. Sleep duration and incidence of obesity in infants, children, and adolescents: a systematic review and meta-analysis of prospective studies. *Sleep*. 2018;41(4). doi:10.1093/sleep/zsy018

157. Deng X, He M, He D, Zhu Y, Zhang Z, Niu W. Sleep duration and obesity in children and adolescents: evidence from an updated and dose–response meta-analysis. *Sleep Med*. 2021;78:169-181. doi:10.1016/j.sleep.2020.12.027

158. Plumptre L, Anderson LN, Chen Y, et al. Longitudinal Analysis of Sleep Duration and Cardiometabolic Risk in Young Children. *Childhood Obesity*. 2017;13(4):291-299. doi:10.1089/chi.2016.0279

159. Gimeno GP, Rupérez AI, Vázquez-Cobela R, et al. Sleep duration and blood pressure in Spanish children with obesity. *Proceedings of the Nutrition Society*. 2020;79(OCE2):E592. doi:10.1017/S0029665120005418

160. Sun J, Wang M, Yang L, Zhao M, Bovet P, Xi B. Sleep duration and cardiovascular risk factors in children and adolescents: A systematic review. *Sleep Med Rev*. 2020;53:101338. doi:10.1016/j.smrv.2020.101338

161. Ward AL, Reynolds AN, Kuroko S, Fangupo LJ, Galland BC, Taylor RW. Bidirectional associations between sleep and dietary intake in 0–5 year old children: A systematic review with evidence mapping. *Sleep Med Rev*. 2020;49:101231. doi:10.1016/j.smrv.2019.101231

162. Antczak D, Lonsdale C, Lee J, et al. Physical activity and sleep are inconsistently related in healthy children: A systematic review and meta-analysis. *Sleep Med Rev*. 2020;51:101278. doi:10.1016/j.smrv.2020.101278

163. Manios Y, Kourlaba G, Grammatikaki E, Androutsos O, Moschonis G, Roma-Giannikou E. Development of a diet-lifestyle quality index for young children and its relation to obesity: The Preschoolers Diet-Lifestyle Index. *Public Health Nutr*. 2010;13(12):2000-2009. doi:10.1017/S1368980010000698

164. Kovács E, Hunsberger M, Reisch L, et al. Adherence to combined lifestyle factors and their contribution to obesity in the IDEFICS study. *Obesity Reviews*. 2015;16:138-150. doi:10.1111/obr.12349

165. Meyer U, Schindler C, Bloesch T, et al. Combined impact of negative lifestyle factors on cardiovascular risk in children: A randomized prospective study. *Journal of Adolescent Health*. 2014;55(6):790-795. doi:10.1016/j.jadohealth.2014.07.007

166. Manios Y, Kourlaba G, Grammatikaki E, et al. Development of a lifestyle-diet quality index for primary schoolchildren and its relation to insulin resistance: The Healthy Lifestyle-Diet Index. *Eur J Clin Nutr*. 2010;64(12):1399-1406. doi:10.1038/ejcn.2010.172

167. Gregory JW. Prevention of Obesity and Metabolic Syndrome in Children. *Front Endocrinol (Lausanne)*. 2019;10:669. doi:10.3389/fendo.2019.00669

168. World Health Organization. *Report of the Commission on Ending Childhood Obesity*.; 2016. https://www.who.int/publications/i/item/9789241510066

169. Al-Jawaldeh A, Hammerich A, Doggui R, Engesveen K, Lang K, McColl K. Implementation of WHO Recommended Policies and Interventions on Healthy Diet in the Countries of the Eastern Mediterranean Region: From Policy to Action. *Nutrients 2020, Vol 12, Page 3700*. 2020;12(12):3700. doi:10.3390/NU12123700

170. Mayr HL, Cohen F, Isenring E, Soenen S, Marshall S. Multidisciplinary lifestyle intervention in children and adolescents - results of the project GRIT (Growth, Resilience, Insights, Thrive) pilot study. *BMC Pediatrics 2020 20:1*. 2020;20(1):1-16. doi:10.1186/S12887-020-02069-X

171. Fornari E, Maffeis C. Treatment of Metabolic Syndrome in Children. *Front Endocrinol (Lausanne)*. 2019;10:702. doi:10.3389/FENDO.2019.00702

172. Hampl SE, Hassink SG, Skinner AC, et al. Clinical Practice Guideline for the Evaluation and Treatment of Children and Adolescents With Obesity. *Pediatrics*. 2023;151(2). doi:10.1542/peds.2022-060640

173. Dabas A, Seth A. Prevention and Management of Childhood Obesity. *The Indian Journal of Pediatrics*. 2018;85(7):546-553. doi:10.1007/s12098-018-2636-x

174. Salam RA, Padhani ZA, Das JK, et al. Effects of Lifestyle Modification Interventions to Prevent and Manage Child and Adolescent Obesity: A Systematic Review and Meta-Analysis. *Nutrients*. 2020;12(8):1-23. doi:10.3390/nu12082208

175. Bondyra-Wiśniewska B, Myszkowska-Ryciak J, Harton A. Impact of Lifestyle Intervention Programs for Children and Adolescents with Overweight or Obesity on Body Weight and Selected Cardiometabolic Factors—A Systematic Review. *Int J Environ Res Public Health*. 2021;18(4):2061. doi:10.3390/ijerph18042061

176. Mehdizadeh A, Nematy M, Vatanparast H, Khadem-Rezaiyan M, Emadzadeh M. Impact of Parent Engagement in Childhood Obesity Prevention Interventions on Anthropometric Indices among Preschool Children: A Systematic Review. *Childhood Obesity*. 2020;16(1):3-19. doi:10.1089/chi.2019.0103

177. Gato-Moreno M, Martos-Lirio MF, Leiva-Gea I, et al. Early Nutritional Education in the Prevention of Childhood Obesity. *Int J Environ Res Public Health*. 2021;18(12):6569. doi:10.3390/ijerph18126569

178. Labayen I, Medrano M, Arenaza L, et al. Effects of Exercise in Addition to a Family-Based Lifestyle Intervention Program on Hepatic Fat in Children With Overweight. *Diabetes Care*. 2020;43(2):306-313. doi:10.2337/DC19-0351

179. Arenaza L, Medrano M, Oses M, et al. The effect of a family-based lifestyle education program on dietary habits, hepatic fat and adiposity markers in 8–12-year-old children with overweight/obesity. *Nutrients*. 2020;12(5). doi:10.3390/nu12051443

180. World Health Organization. Infant and young child feeding. Published June 9, 2021. Accessed November 13, 2020. https://www.who.int/news-room/fact-sheets/detail/infant-and-young-child-feeding

181. World Health Organ Tech Rep Ser. *Obesity: Preventing and Managing the Global Epidemic: Report of a WHO Consultation*.; 2000. Accessed May 25, 2022. https://apps.who.int/iris/handle/10665/42330

182. Farran A, Zamora R, Cervera P, Centre d'Enseyament Superior de Nutrició i Dietètica. *Tablas de Composición de Alimentos Del CESNID*. Vol 78. 3rd ed. (Mc- Graw-Hill / Interamericana, Edicions Universitat de Barcelona, eds.).; 2004.

183. Babio Sánchez N, de la Serra Besora Moreno M, Lladó Bellette N, Salas Salvadó Jordi. *CELIACBASE. Tabla de Composición de Productos Sin Gluten* . Publicacions URV; 2018.

184. Babio Nancy, Dragusan Lica Natalia, De Las Heras Sara, Blanquer Maria. *Tabla de Composición de Productos y Alternativas Vegetales*. 1st ed. Publicacions URV; 2022.

185. Schröder H, Fitó M, Estruch R, et al. A Short screener is valid for assessing mediterranean diet adherence among older spanish men and women. *Journal of Nutrition*. 2011;141(6):1140-1145. doi:10.3945/jn.110.135566

186. Verbestel V, de Henauw S, Bammann K, et al. Are context-specific measures of parental-reported physical activity and sedentary behaviour associated with accelerometer data in 2–9-year-old

European children? *Public Health Nutr*. 2015;18(5):860-868. doi:10.1017/S136898001400086X

187. Cole TJ, Lobstein T. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. *Pediatr Obes*. 2012;7(4):284-294. doi:10.1111/J.2047-6310.2012.00064.X

188. VanItallie TB, Yang MU, Heymsfield SB, Funk RC, Boileau RA. Height-normalized indices of the body's fat-free mass and fat mass: potentially useful indicators of nutritional status. *Am J Clin Nutr*. 1990;52(6):953-959. doi:10.1093/ajcn/52.6.953

189. Frost PH, Havel RJ. Rationale for Use of Non–High-Density Lipoprotein Cholesterol Rather Than Low-Density Lipoprotein Cholesterol as a Tool for Lipoprotein Cholesterol Screening and Assessment of Risk and Therapy. *Am J Cardiol*. 1998;81(4):26B-31B. doi:10.1016/S0002-9149(98)00034-4

190. Jimeno-Martínez A, Maneschy I, Moreno LA, et al. Reliability and Validation of the Child Eating Behavior Questionnaire in 3- to 6-Year-Old Spanish Children. *Front Psychol*. 2022;13. doi:10.3389/fpsyg.2022.705912

191. Yerushalmy J. The classification of newborn infants by birth weight and gestational age. *J Pediatr*. 1967;71(2):164-172. doi:10.1016/S0022-3476(67)80067-2

192. World Health Organization. Regional Office for Europe. *WHO European Childhood Obesity Surveillance Initiative (COSI) Report on the Fourth Round of Data Collection*.; 2021. Accessed January 27, 2022. https://apps.who.int/iris/handle/10665/341189

193. Fogel A, Goh AT, Fries LR, et al. Faster eating rates are associated with higher energy intakes during an ad libitum meal, higher BMI and greater adiposity among 4·5-year-old children: results from the Growing Up in Singapore Towards Healthy Outcomes (GUSTO) cohort. *British Journal of Nutrition*. 2017;117(7):1042-1051. doi:10.1017/S0007114517000848

194. Okubo H, Murakami K, Masayasu S, Sasaki S. The Relationship of Eating Rate and Degree of Chewing to Body Weight Status among Preschool Children in Japan: A Nationwide Cross-Sectional Study. *Nutrients*. 2018;11(1):64. doi:10.3390/nu11010064

195. Franko DL, Striegel-Moore RH, Thompson D, et al. The relationship between meal frequency and body mass index in black and white adolescent girls: More is less. *Int J Obes*. 2008;32(1):23-29. doi:10.1038/sj.ijo.0803654

196. Faith MS, Diewald LK, Crabbe S, Burgess B, Berkowitz RI. Reduced Eating Pace (RePace) Behavioral Intervention for Children Prone to or with Obesity: Does the Turtle Win the Race? *Obesity*. 2019;27(1):121-129. doi:10.1002/oby.22329

197. Kelishadi R, Qorbani M, Motlagh ME, Heshmat R, Ardalan G, Bahreynian M. Association of eating frequency with anthropometric indices and blood pressure in children and adolescents: The CASPIAN-IV Study. *J Pediatr (Rio J)*. 2016;92(2):156-167. doi:10.1016/j.jped.2015.05.009

198. Jääskeläinen A, Schwab U, Kolehmainen M, Pirkola J, Järvelin MR, Laitinen J. Associations of meal frequency and breakfast with obesity and metabolic syndrome traits in adolescents of Northern Finland

Birth Cohort 1986. *Nutrition, Metabolism and Cardiovascular Diseases*. 2013;23(10):1002-1009. doi:10.1016/j.numecd.2012.07.006

199. Murakami K, Livingstone MBE. Associations of eating frequency with adiposity measures, blood lipid profiles and blood pressure in British children and adolescents. *British Journal of Nutrition*. 2014;111(12):2176-2183. doi:10.1017/S0007114514000452

200. Jennings A, Cassidy A, van Sluijs EMF, Griffin SJ, Welch AA. Associations between eating frequency, adiposity, diet, and activity in 9-10 year old healthy-weight and centrally obese children. *Obesity*. 2012;20(7):1462-1468. doi:10.1038/oby.2012.72

201. Ochiai H, Shirasawa T, Ohtsu T, et al. The impact of eating quickly on anthropometric variables among schoolgirls: a prospective cohort study in Japan. *The European Journal of Public Health*. 2014;24(4):691-695. doi:10.1093/eurpub/ckt120

202. Ochiai H, Shirasawa T, Nanri H, et al. Eating quickly is associated with waist-to-height ratio among Japanese adolescents: A cross-sectional survey. *Archives of Public Health*. 2016;74(1). doi:10.1186/s13690-016-0130-3

203. Yamagishi K, Sairenchi T, Sawada N, et al. Impact of Speed-Eating Habit on Subsequent Body Mass Index and Blood Pressure Among Schoolchildren — The Ibaraki Children's Cohort Study (IBACHIL) —. *Circulation Journal*. 2018;82(2):419-422. doi:10.1253/circj.CJ-17-0287

204. Eloranta AM, Lindi V, Schwab U, et al. Dietary factors associated with overweight and body adiposity in Finnish children aged 6-8 years: The PANIC Study. *Int J Obes*. 2012;36(7):950-955. doi:10.1038/ijo.2012.89

205. Monnier L, Schlienger JL, Colette C, Bonnet F. The obesity treatment dilemma: Why dieting is both the answer and the problem? A mechanistic overview. *Diabetes Metab*. 2021;47(3):101192. doi:10.1016/j.diabet.2020.09.002

206. Santos AF, Fernandes C, Fernandes M, Santos AJ, Veríssimo M. Associations between Emotion Regulation, Feeding Practices, and Preschoolers' Food Consumption. *Nutrients*. 2022;14(19):4184. doi:10.3390/nu14194184

207. Lasschuijt M, Mars M, de Graaf C, Smeets PAM. How oro-sensory exposure and eating rate affect satiation and associated endocrine responses-a randomized trial. *Am J Clin Nutr*. 2020;111(6):1137-1149. doi:10.1093/ajcn/nqaa067

208. Gómez-Zúñiga RS, Wintergerst A. Effect of food portion on masticatory parameters in 8- to 10-year-old children. *J Texture Stud*. 2023;54(1):67-75. doi:10.1111/jtxs.12724

209. Krop EM, Hetherington MM, Nekitsing C, Miquel S, Postelnicu L, Sarkar A. Influence of oral processing on appetite and food intake – A systematic review and meta-analysis. *Appetite*. 2018;125:253-269. doi:10.1016/j.appet.2018.01.018

210. Slyper A. Oral Processing, Satiation and Obesity: Overview and Hypotheses. *Diabetes Metab Syndr Obes*. 2021;Volume 14:3399-3415. doi:10.2147/DMSO.S314379

211. Forde CG, Stieger M. Metabolomic Impacts of Food Oral Processing. In: *Function Chemistry, Function and Analysis No. 30. Oral Processing*

and Consumer Perception: Biophysics, Food Microstructures and Health. ; 2022. www.rsc.org

212. Slyper AH, Kopfer K, Huang WM, Re'em Y. Increased hunger and speed of eating in obese children and adolescents. *Journal of Pediatric Endocrinology and Metabolism*. 2014;27(5-6). doi:10.1515/jpem-2013-0271

213. Forde CG, van Kuijk N, Thaler T, de Graaf C, Martin N. Oral processing characteristics of solid savoury meal components, and relationship with food composition, sensory attributes and expected satiation. *Appetite*. 2013;60(1):208-219. doi:10.1016/j.appet.2012.09.015

214. Forde CG, Leong C, Chia-Ming E, McCrickerd K. Fast or slow-foods? Describing natural variations in oral processing characteristics across a wide range of Asian foods. *Food Funct*. 2017;8(2):595-606. doi:10.1039/c6fo01286h

215. Teo PS, Lim AJ, Goh AT, et al. Texture-based differences in eating rate influence energy intake for minimally processed and ultra-processed meals. *Am J Clin Nutr*. 2022;116(1):244-254. doi:10.1093/ajcn/nqac068

216. van den Boer J, Werts M, Siebelink E, de Graaf C, Mars M. The Availability of Slow and Fast Calories in the Dutch Diet: The Current Situation and Opportunities for Interventions. *Foods*. 2017;6(10):87. doi:10.3390/foods6100087

217. Viskaal-van Dongen M, Kok FJ, de Graaf C. Eating rate of commonly consumed foods promotes food and energy intake. *Appetite*. 2011;56(1):25-31. doi:10.1016/j.appet.2010.11.141

218. Teo PS, Tso R, van Dam RM, Forde CG. Taste of Modern Diets: The Impact of Food Processing on Nutrient Sensing and Dietary Energy Intake. *J Nutr*. 2022;152(1):200-210. doi:10.1093/jn/nxab318

219. Forde CG, de Graaf K. Influence of Sensory Properties in Moderating Eating Behaviors and Food Intake. *Front Nutr*. 2022;9:841444. doi:10.3389/fnut.2022.841444

220. Forde CG, Mars M, de Graaf K. Ultra-Processing or Oral Processing? A Role for Energy Density and Eating Rate in Moderating Energy Intake from Processed Foods. *Curr Dev Nutr*. 2020;4(3). doi:10.1093/cdn/nzaa019

221. Soltani S, Jayedi A, Shab-Bidar S, Becerra-Tomás N, Salas-Salvadó J. Adherence to the Mediterranean Diet in Relation to All-Cause Mortality: A Systematic Review and Dose-Response Meta-Analysis of Prospective Cohort Studies. *Advances in Nutrition*. 2019;10(6):1029-1039. doi:10.1093/advances/nmz041

222. Martinez-Lacoba R, Pardo-Garcia I, Amo-Saus E, Escribano-Sotos F. Mediterranean diet and health outcomes: a systematic meta-review. *Eur J Public Health*. 2018;28(5):955-961. doi:10.1093/eurpub/cky113

223. Rosato V, Temple NJ, la Vecchia C, Castellan G, Tavani A, Guercio V. Mediterranean diet and cardiovascular disease: a systematic review and meta-analysis of observational studies. *Eur J Nutr*. 2019;58(1):173-191. doi:10.1007/s00394-017-1582-0

224. Sofi F, Abbate R, Gensini GF, Casini A. Accruing evidence on benefits of adherence to the Mediterranean diet on health: an updated

systematic review and meta-analysis. *Am J Clin Nutr*. 2010;92(5):1189-1196. doi:10.3945/ajcn.2010.29673

225. Esposito K, Maiorino MI, Bellastella G, Chiodini P, Panagiotakos D, Giugliano D. A journey into a Mediterranean diet and type 2 diabetes: a systematic review with meta-analyses. *BMJ Open*. 2015;5(8):e008222. doi:10.1136/bmjopen-2015-008222

226. D'Alessandro A, Lampignano L, De Pergola G. Mediterranean Diet Pyramid: A Proposal for Italian People. A Systematic Review of Prospective Studies to Derive Serving Sizes. *Nutrients*. 2019;11(6):1296. doi:10.3390/nu11061296

227. Guasch-Ferré M, Willett WC. The Mediterranean diet and health: a comprehensive overview. *J Intern Med*. 2021;290(3):549-566. doi:10.1111/JOIM.13333

228. Fardet A. Minimally processed foods are more satiating and less hyperglycemic than ultra-processed foods: a preliminary study with 98 ready-to-eat foods. *Food Funct*. 2016;7(5):2338-2346. doi:10.1039/C6FO00107F

229. Bakaloudi DR, Chrysoula L, Kotzakioulafi E, Theodoridis X, Chourdakis M. Impact of the Level of Adherence to Mediterranean Diet on the Parameters of Metabolic Syndrome: A Systematic Review and Meta-Analysis of Observational Studies. *Nutrients*. 2021;13(5):1514. doi:10.3390/nu13051514

230. Kastorini CM, Milionis HJ, Esposito K, Giugliano D, Goudevenos JA, Panagiotakos DB. The Effect of Mediterranean Diet on Metabolic

Syndrome and its Components. *J Am Coll Cardiol*. 2011;57(11):1299-1313. doi:10.1016/j.jacc.2010.09.073

231. Lampropoulou M, Chaini M, Rigopoulos N, Evangeliou A, Papadopoulou-Legbelou K, Koutelidakis AE. Association between Serum Lipid Levels in Greek Children with Dyslipidemia and Mediterranean Diet Adherence, Dietary Habits, Lifestyle and Family Socioeconomic Factors. *Nutrients*. 2020;12(6):1600. doi:10.3390/nu12061600

232. Ramírez-Moreno E, Arias-Rico J, Jiménez-Sánchez RC, et al. Role of Bioactive Compounds in Obesity: Metabolic Mechanism Focused on Inflammation. *Foods*. 2022;11(9):1232. doi:10.3390/foods11091232

233. Eny KM, Chen S, Anderson LN, et al. Breastfeeding duration, maternal body mass index, and birth weight are associated with differences in body mass index growth trajectories in early childhood. *American Journal of Clinical Nutrition*. 2018;107(4):584-592. doi:10.1093/ajcn/nqx081

234. Wong PD, Anderson LN, Dai DDW, et al. The Association of Breastfeeding Duration and Early Childhood Cardiometabolic Risk. *J Pediatr*. 2018;192:80-85.e1. doi:10.1016/j.jpeds.2017.09.071

235. Martin RM, Kramer MS, Patel R, et al. Effects of Promoting Long-term, Exclusive Breastfeeding on Adolescent Adiposity, Blood Pressure, and Growth Trajectories: A Secondary Analysis of a Randomized Clinical Trial. *JAMA Pediatr*. 2017;171(7):e170698. doi:10.1001/jamapediatrics.2017.0698

236. Wisnieski L, Kerver J, Holzman C, Todem D, Margerison-Zilko C. Breastfeeding and Risk of Metabolic Syndrome in Children and Adolescents: A Systematic Review. *Journal of Human Lactation*. 2018;34(3):515-525. doi:10.1177/0890334417737038

237. Aglipay M, Vanderloo LM, Cost KT, Maguire JL, Birken CS. The Digital Media Environment and Cardiovascular Risk in Children. *Canadian Journal of Cardiology*. 2020;36(9):1440-1447. doi:10.1016/J.CJCA.2020.04.015

238. Roberge JB, van Hulst A, Barnett TA, et al. Lifestyle Habits, Dietary Factors, and the Metabolically Unhealthy Obese Phenotype in Youth. *J Pediatr*. 2019;204:46-52.e1. doi:10.1016/j.jpeds.2018.08.063

239. García-Hermoso A, Ezzatvar Y, López-Gil JF, Ramírez-Vélez R, Olloquequi J, Izquierdo M. Is adherence to the Mediterranean diet associated with healthy habits and physical fitness? A systematic review and meta-analysis including 565 421 youths. *British Journal of Nutrition*. 2022;128(7):1433-1444. doi:10.1017/S0007114520004894

240. Iaccarino Idelson P, Scalfi L, Valerio G. Adherence to the Mediterranean Diet in children and adolescents: A systematic review. *Nutrition, Metabolism and Cardiovascular Diseases*. 2017;27(4):283-299. doi:10.1016/j.numecd.2017.01.002

241. Martino F, Martino E, Versacci P, Niglio T, Zanoni C, Puddu PE. Lifestyle and awareness of cholesterol blood levels among 29159 community school children in Italy. *Nutrition, Metabolism and Cardiovascular Diseases*. 2019;29(8):802-807. doi:10.1016/j.numecd.2019.05.060

X. APPENDIXES

Scientific contributions

Publications derived from the present doctoral thesis

Garcidueñas-Fimbres TE, Paz-Graniel I, Nishi SK, Salas-Salvadó J, Babio N. Eating Speed, Eating Frequency, and Their Relationships with Diet Quality, Adiposity, and Metabolic Syndrome, or Its Components. Nutrients. 2021, 13, 1687. doi:10.3390/nu13051687

Garcidueñas-Fimbres TE, Paz-Graniel I, Gómez-Martínez C, et al. Associations Between Eating Speed, Diet Quality, Adiposity, and Cardiometabolic Risk Factors. *J Pediatr*. 2023;252:31-39.e1. doi:10.1016/j.jpeds.2022.08.024

Participation in national and international conferences

Title of the work: Eating speed, diet quality and cardiometabolic factors in Spanish children: The CORALS study.
Name of the conference: 39th International Symposium on Diabetes and Nutrition. Athens, Greece. June, 16th-19th, 2021.
Organising entity: Diabetes and Nutrition Study Group.
Authors: Tany E. Garcidueñas Fimbres; Indira Paz Graniel; Carlos Gomez Martínez; José Manuel Jurado Castro; Rosaura Leis; Joaquín Escribano; Luis A. Moreno; Santiago Navas Carretero; Olga Portoles; Karla A. Pérez Vega; Mercedes Gil Campos; Alicia López Rubio; Cristina Rey Reñones; Pilar De Miguel Etayo; J. Alfredo Martínez; Katherine Flores Rojas; Rocío Vázquez Cobela; Verónica Luque; Maria Luisa Miguel Berges; Belén Pastor Villaescusa; Francisco Jesús Llorente Cantarero; Jordi Salas Salvadó; Nancy Babio.

Title of the work: Eating speed is associated to diet quality and cardiometabolic risk factors in children aged 3-6 years old: The CORAL Study.

Appendixes

Name of the conference: Congreso XII Symposium CIBER Fisiopatología de la Obesidad y Nutrición [online event]. October 26th-28th, 2021.
Organizing entity: CIBER FISIOPATOLOGIA DE LA OBESIDAD Y NUTRICION (CIBEROBN).
Authors: Tany E . Garcidueñas Fimbres; Indira Paz Graniel; Maria Pascual Compte; José Jurado Castro; Rocío Vázquez Cobela; Luis A Moreno; Santiago Navas Carretero; Pilar Codoñer; Karla Alejandra Perez; Katherine Flores Rojas; Rosaura Leis; Pilar De Miguel Etayo; Carlos Gomez Martínez; Alfredo Martínez; Dolores Corella; Ana Moreira; Mercedes Gil Campos; Jordi Salas Salvadó; Nancy Babio.

Title of the work: EATING SPEED, EATING FREQUENCY AND THEIR RELATIONSHIPS WITH DIET QUALITY, ADIPOSITY AND METABOLIC SYNDROME OR ITS COMPONENTS.
Name of the conference: 38th International Symposium on Diabetes and Nutrition [online event]. June 21th-24th, 2021.
Organizing entity: Diabetes and Nutrition Study Group.
Authors: Tany E. Garcidueñas Fimbres; Indira Paz Graniel; Stephanie K. Nishi; Jordi Salas Salvadó; Nancy Babio.

Research stay

Research center: SickKids Research Institute.
City: Toronto, Canada.
Supervisor: Prof. Catherine Birken.
Duration: 3 months and 13 days (September – December, 2022).
Objective: To develop a child lifestyle behaviours composite score that is strongly associated with cardiometabolic risk in the cohort of TARGet Kids study.

UNIVERSITAT
ROVIRA i VIRGILI