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Review

The burden of low back pain in children and adolescents with overweight and obesity: from pathophysiology to prevention and treatment strategies

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***Abstract*:** Nonspecific low back pain (LBP) is one of the most common causes of disability, affecting all individuals at least once in their lifetime. Such a condition is also becoming increasingly frequent in the pediatric population, especially in children and adolescents with overweight/obesity. Furthermore, new-onset LBP during adolescence has been demonstrated to be a strong predictor of developing LBP later in life, contributing to poorer outcomes and increasing social and medical costs. Several causes and different mechanisms have been considered for the development of LBP in pediatric individuals affected by obesity. For this reason, planning adequate prevention and treatment strategies, mainly through conservative lifestyle changes, would be crucial to anticipate the negative consequences of persisting LBP in adulthood. The aim of this narrative review was to characterize the relationship between LBP and overweight/obesity in the pediatric population, highlighting epidemiological and pathophysiological aspects. In addition, prevention and treatment approaches will be reviewed considering the need to reduce the burden of LBP on this population. According to our search, LBP was more frequent in children and adolescents with overweight and obesity and has been associated with several anthropometric and lifestyle factors, including lumbar hyperlordosis, sedentary habits, physical inactivity, carrying a heavy schoolbag, low vitamin D levels, psychosocial ill-being, and premature intervertebral disc degeneration. Most of these conditions may be addressed with conservative strategies mainly consisting of dietary adjustments, physical exercise, education programs, and physical therapy.

Keywords: adolescents, children, intervertebral disc, intervertebral disc degeneration, low back pain, obesity, overweight

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Background

Low back pain (LBP) is a common condition affecting almost every individual at least once in a lifetime. It represents the first cause of disability worldwide and is associated with a significant burden at both individual and societal levels.[1] Although traditionally investigated in aging populations, LBP has been increasingly reported in children and adolescents. Previous studies have shown that as many as 5% of children may be suffering from LBP at any time[2] and that LBP prevalence is higher in females and increases with age, ranging from 1 to 6% of children and 18 to 51% of adolescents.[3] Furthermore, new-onset LBP during adolescence has also been demonstrated to be a strong predictor of developing LBP later in life,[4] especially due to maladaptive beliefs and attitudes related to earlier pain events.[3] According to the biopsychosocial model, LBP is determined by the complex interplay between biological factors [intervertebral disc degeneration (IDD) associated with aging, obesity, and

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overload] and psychosocial factors, including depression, anxiety, social isolation, etc.[5,6] Several determinants related to the multidimensional nature of LBP have been characterized and are gaining increasing attention for their importance in contributing to poorer outcomes and increasing medical costs. Identifying early life risk factors for the development of LBP may be crucial to arrest this trend.[7]

Among all the conditions that may lead to the occurrence and persistence of LBP in the pediatric population, obesity holds a prominent role. Obesity is the most common nutritional disorder of the evolutive age as well as a growing public health issue affecting one out of three individuals under the age of 18.[8] According to the World Health Organization, >340 million children and adolescents in the world and >2 million in Italy are affected by obesity or overweight.[9] The worldwide prevalence of overweight and obesity has dramatically increased in the last decades, reaching 18% of the pediatric population in 2016.[8] Children and adolescents with overweight and obesity are at an increased risk of developing short- and long-term complications that may persist into adult life.[10] These include obesity itself, as well as metabolic, cardiovascular (i.e., hypertension, dyslipidemia, type 2 diabetes mellitus), and musculoskeletal disorders (i.e., pes planus, valgus knees), with a huge impact on global morbidity and mortality.[11] Among musculoskeletal complications affecting subjects with overweight and obesity, LBP has certainly great relevance. Indeed, obese children and adolescents present a three-fold higher risk of developing LBP compared to normal-weight peers.[12] By overloading spinal segments, especially the lumbar spine, overweight has been recognized as a major contributor to the development of nonspecific LBP, even in young individuals.[13] Moreover, proinflammatory adipokines and systemic inflammation that characterize individuals with obesity have also been imputed as possible promoters of IDD.[6] Therefore, understanding the relationship between LBP and overweight/obesity in children and adolescents would be crucial to obtain a timely diagnosis and plan the most appropriate prevention and treatment strategies.

Although the differential diagnosis of LBP in the pediatric age encompasses several different entities (including spondylolysis, Scheuermann's disease, spondyloarthropathies, spondylodiscitis, and tumors),[14] the purpose of this narrative review was to specifically investigate the relationship between nonspecific LBP and overweight/obesity in children and adolescents. Epidemiological and pathophysiological aspects will be reported, and then treatment and prevention strategies will be proposed to reduce the occurrence and tackle LBP in this population.

Epidemiology of LBP in children and adolescents

In contrast to conventional clinical knowledge, LBP seems to be a rather common and usually benign condition in children and adolescents, with a constantly increasing incidence in recent years. The reported lifetime prevalence of LBP in the pediatric age ranges from 4.7 to 74.4%, with wide variations among different countries and age groups.[15] These data bear significant relevance considering the strong association between the initial LBP episode and the subsequent high risk of developing chronic LBP during adulthood. Furthermore, several studies have also demonstrated that juvenile LBP is responsible for significantly higher annual medical and social costs (i.e., 19.5 billion USD for the 10- to 17-year age group in the United States and 100 million EUR in people under the age of 25 in Germany). These result from both indirect and direct expenses associated with parents' work absence and medical expenses, respectively.[15] Overall, LBP is a common condition in youth with increasing prevalence with age. Although most cases are not severe and do not require invasive treatments, 40.9% of children and adolescents will seek some form of care.[15] According to a large community study conducted by Watson *et al.*,[16] 94% of children with LBP between 11 and 14 years of age reported some sort of limitation in simple activities such as reaching up to a high shelf, sitting up in bed from a lying position, and bending down to wear socks.

In addition, a higher incidence of LBP is reported in females compared to males. Sex differences, along with the time of onset, may be related to the earlier age of puberty onset and growth spurt, and to the different distribution of adipose tissue and body composition.[17] Females are more likely to be overweight during adolescence, with a peripheral adipose tissue distribution, while abdominal visceral fat accumulation is more common among males.[18] According to a previous population-based prospective cohort study, overweight was associated with incident LBP rather than with

persistent LBP in both sexes. In this study, a body mass index (BMI) between 7 and 16 years old was significantly associated with LBP in females at 18 years of age, independently of smoking, physical activity, and family socioeconomic status. In males, the BMI at 16–18 years predicted incident LBP at 18 years. According to the authors, early IDD, psychosocial factors, psychosomatic symptoms (among females), and different pain perceptions (in both males and females) were the main risk factors for LBP in their young cohort.[18]

Pathophysiology of LBP in children and adolescents with overweight and obesity

Anthropometric and lifestyle factors

Like adults, most LBP episodes in the pediatric population are nonspecific and self-limited. However, children and adolescents may also be affected by specific causes of LBP requiring immediate attention and dedicated care, which have been extensively discussed elsewhere. Indeed, nonspecific LBP should be a diagnosis of exclusion. The most common specific musculoskeletal causes of LBP in children and adolescents include spondylolysis with or without isthmic spondylolisthesis, segmentation defects, disc herniation, apophyseal ring fracture, Scheuermann's disease, facet joint syndrome, sacroiliitis, and trauma.[14] Interestingly, smoking, late puberty and being overweight have all been associated with an increased risk of undergoing discectomy due to lumbar disc herniation in adolescents.[19] On the other hand, increased pelvic incidence and lumbar lordosis, which are common in overweight children and adolescents, are also significantly correlated with a higher risk of spondylolysis and isthmic spondylolisthesis.[20,21]

Previous studies have considered the role of different anthropometric factors on the risk of developing LBP, such as sitting height, sitting posture, and lumbar spine flexibility, although often reporting contradictory results with a minor impact on LBP natural history.[2,22] Similarly, based on the empirical observation of a higher number of patients in the adolescent age, it has also been postulated that LBP may derive from an imbalance of growth rates between the vertebrae and the surrounding muscle and ligamentous tissues, especially during the pubertal spurt. However, no association was found in longitudinal studies.[23] Excessive load bearing resulting from overweight would require additional mechanical demands possibly participating in the development of LBP. The accumulation of adipose tissue in the abdominal area, combined with the loss of tone in the muscles of the abdominal wall, leads to lumbar hyperlordosis[24] and compensatory thoracic hyperkyphosis with head protraction.[25] Collectively, such alterations of sagittal balance may promote LBP due to impaired load transmission across the spine.

A sedentary and inactive lifestyle may also significantly contribute to the development of LBP, while LBP itself may encourage physical inactivity, resulting in decreased social interactions and negative lifestyle changes that may foster obesity and exacerbate LBP, in a vicious cycle. A sedentary lifestyle may result in diminished bone mineral density, poor flexibility, and weight gain. For example, it has been reported that children watching television for <1 h/day did not show a greater risk of LBP compared to children watching none. However, watching television for 1–2 h and >2 h was correlated with a 70% and 210% higher risk to develop LBP, respectively.[26] In this regard, a recent cross-sectional study has demonstrated that physical inactivity from childhood to adolescence in combination with a sedentary behavior doubled the likelihood of having LBP.[27]

The relationship between the development of LBP and carrying a heavy schoolbag has been highly debated in the literature, often with contradictory results. This may be due to several reasons, such as the cross-sectional design of most studies and the highly heterogeneous definition of schoolbag weight.[2] LBP has been evaluated in relation to perceived schoolbag heaviness,[28] absolute,[29] or relative schoolbag weight (calculated as the ratio between bag and body weight).[30,31] Several studies have shown that weighty bags are indeed associated with a higher risk of developing LBP.[15] In a recent cross-sectional study conducted in an adolescent population from Kuwait,[31] LBP was significantly associated with perceived schoolbag heaviness as well as relative bag weight. However, no significant association was found in the latter after adjusting for confounders in the multivariate model. Similarly, previous studies have shown that children considering their schoolbag heavy reported up to 60% higher odds of LBP.[28,32] Although not consistent, such quantification of schoolbag weight is worth further consideration. Children and adolescents with LBP may perceive their bags as heavy because of preexisting pain

and discomfort related to carrying loads, independently of their actual weight.[33] Interestingly, an elegant study from Nicolet and colleagues[34] showed that adolescents with a history of LBP tended to overestimate bag heaviness, whereas subjects without a history of LBP were more likely to underestimate it. In this regard, psychological factors, such as fear avoidance and kinesiophobia (both known causes of LBP), may play a key role.[5]

From a biomechanical point of view, carrying a schoolbag induces an increase in trunk forward lean (TFN), defined as the angle between a vertical line passing through the great trochanter and a line connecting the great trochanter with the acromion on a lateral view. Incremented TFN values are correlated with higher stresses at the lumbosacral junction, possibly triggering LBP.[35] Moreover, TFN significantly increases with relative schoolbag weights >15% and faster paces (i.e., from walking to running), thus meaning that the same force on the lumbar spine can be exerted by a lighter load when more demanding tasks are performed (such as running to school if late).[36] Additional compensating mechanisms are represented by increased craniovertebral angle through neck flexion[37] and lumbar hyperlordosis to counterbalance the bag weight with the mass of the abdomen.[38] Both adaptations are strictly correlated to increasing fatigue, with the latter significantly impacting lumbosacral overstress.[39] This phenomenon may be even more pronounced in overweight children due to the association of altered sagittal balance and schoolbag-related increased loading on the lumbar spine. Interestingly, in a cross-sectional study from Spiteri *et al.*,[40] LBP due to heavy schoolbags was significantly associated with BMI in a logistic regression model. Despite the lack of reliable data, several institutions and societies recommend that schoolbag weight should not exceed 10–20% of a child's body weight. Such thresholds have been determined based on physiologic data investigating the combined effect of schoolbag loading on vital capacity, oxygen consumption, heart rate, minute ventilation, blood pressure, etc.[39]

Intervertebral disc degeneration

Intervertebral disc degeneration is a progressive, aging-related process, characterized by gradual nucleus pulposus dehydration due to increased extracellular matrix breakdown and loss of viable cells within the intervertebral disc.[41] This eventually leads to a decrease in disc height, loss of lumbar lordosis, and facet joint overload, which are collectively imputed to be the main trigger of nonspecific discogenic LBP.[42] Additional degenerative sequelae including disc herniation, degenerative spondylolisthesis, spinal stenosis, and segmental instability may occur. These conditions are often associated with neurological symptoms, including radiculopathy and myelopathy.[43] Although common, the pathophysiology of IDD is still unclear and has been associated with several risk factors, including genetic makeup, obesity,[6] type I and II diabetes,[44] mechanical overload,[43] and smoking.[45]

Once thought to occur primarily in adults, IDD has also been shown in children and adolescents and has been therefore termed juvenile IDD.[46] Differently from aging-related IDD, juvenile IDD is more frequently associated with spinal deformities and vertebral endplate changes that disrupt the biomechanical loads and stresses exerted on the intervertebral segment, thence increasing the risk of IDD.[47,48] In a population-based cross-sectional study by Samartzis *et al.*,[49] signs of juvenile IDD were retrieved in 35% of individuals aged between 13 and 20, with a higher prevalence of disc bulging, extrusion, and high-intensity zones. Furthermore, overweight and obesity were significantly associated with both the prevalence and severity of IDD, whereas IDD was correlated with a significantly higher prevalence of LBP and sciatica, as well as with diminished social interactions and greater physical disability. Therefore, overweight and obesity are prominent contributors to LBP in the pediatric population, with a three-fold higher risk of developing LBP compared to normal-weight children and adolescents.[12]

To date, the exact mechanisms fostering IDD in overweight and obese individuals are still not clearly understood. Mechanical overload has been traditionally imputed to be a major determinant of IDD. Indeed, previous preclinical studies have demonstrated that exerting supraphysiological compressive loads induces nucleus pulposus cell overstress due to increased mitochondrial damage and reactive oxygen species production, eventually leading to caspase-dependent apoptosis and necroptosis.[43] Static compressive loading has been correlated with increased cell death and matrix catabolism, hence leading to IDD. Diverse cross-sectional investigations have shown that a

higher BMI is associated with the occurrence of IDD, as well as with the number of levels involved, disc space narrowing and severity of degenerative changes.[50,51] It was also reported that persistent overweight was more strongly correlated with IDD, especially within subjects being obese at a young age,[51] particularly if affected by abdominal obesity.[52] A meta-analysis by Xu *et al.* showed that overweight was significantly associated with degenerative changes of the lumbar spine, with a higher impact than age and sex.[53]

Previous studies have also suggested the possible role of systemic inflammatory mediators released by the abundant and dysfunctional adipose tissues in individuals with overweight and obesity. Indeed, the adipose tissue has been renowned for its endocrine functions, which are exerted through the release of several adipokines, including leptin, resistin, and adiponectin, as well as pro-inflammatory cytokines such as interleukin (IL)-1β, IL-6, and tumor necrosis factor-α. All these molecules, which are secreted in particular by the dysfunctional adipose tissue that characterizes obesity, causing a low-grade inflammatory state, have been shown to exert detrimental effects on both nucleus pulposus and annulus fibrosus cells *in vitro*.[54] Interestingly, degenerated discs have displayed to express higher levels of IL-6, which were also positively associated with the severity of IDD.[55]

Recently, a juvenile form of Modic changes has also been imputed as a cause of LBP in the pediatric age. Indeed, a study from Mallow *et al.*[56] reported that Modic changes were present in 14% of a cohort of adolescents with LBP. These alterations were also significantly associated with pain severity and the presence of additional degenerative changes, including Schmorl's nodes, disc space narrowing, and endplate damage. However, juvenile Modic changes were not associated with BMI in this study. The putative effects of obesity on IDD in overweight children and adolescents are summarized in Figure 1.

The role of vitamin D

Vitamin D plays a key role in the development and maintenance of the musculoskeletal system. Physical inactivity, adiposity, reduced sun exposure, insufficient dietary intake, or impairment in the metabolic activation of vitamin D are among the main causes of its deficiency.[57] Interestingly, vitamin D deficiency has been recently proposed as a predisposing factor of LBP in both adult and pediatric populations. This may be mediated by several mechanisms triggered by hypovitaminosis D, including upregulation of proinflammatory markers, lower paravertebral muscle strength, altered sensory neuron excitability, and reduced vertebral bone mineralization.[58,59]

Alghadir *et al.*[57] have demonstrated that LBP severity was associated with vitamin D deficiency and muscle fatigue biomarker increase in schoolchildren. Such associations were even stronger in females and individuals with obesity. On the other hand, a cross-sectional study on adolescents living in an area with a high prevalence of vitamin D deficiency showed that vitamin D was not a major determinant of LBP among children and adolescents.[60] Although several mechanisms may explain how low vitamin D levels may contribute to LBP, findings remain controversial.

Psychosocial aspects of LBP in overweight and obese pediatric subjects

According to previous data, childhood obesity is associated with a significantly higher risk to develop depression, anxiety, behavioral, and emotional disorders.[61] Children and adolescents with obesity are more likely to experience psychopathological distress compared to their normal-weight peers.[62] Several different pathomechanisms underlying this relationship have been described, including chronic systemic inflammation, alterations of the dopaminergic reward system, vitamin D deficiency, and disruption of neuroendocrine mechanisms involving the leptin-melanocortinergic-brain-derived neurotrophic factor signaling.[63] Furthermore, psychosocial contributors such as parental stress, poor-self regulation, weight-related stigma, bullying, and scarce participation in social activities, such as sports, also play significant roles.[64]

Recently, increased attention is being paid to the role of mental well-being in the development of chronic LBP.[65,66] According to the revised International Association for the Study of Pain definition of pain,[67] LBP may be triggered by an emotional experience influenced by other emotions (e.g., fear, sadness, and anxiety), even in the absence of any structural impairment. In this context, misinterpretation of pain as a sign of physical injury may lead to fear-avoidance behaviors and kinesiophobia, which cause refrainment from physical activity and promote self-isolation, ultimately

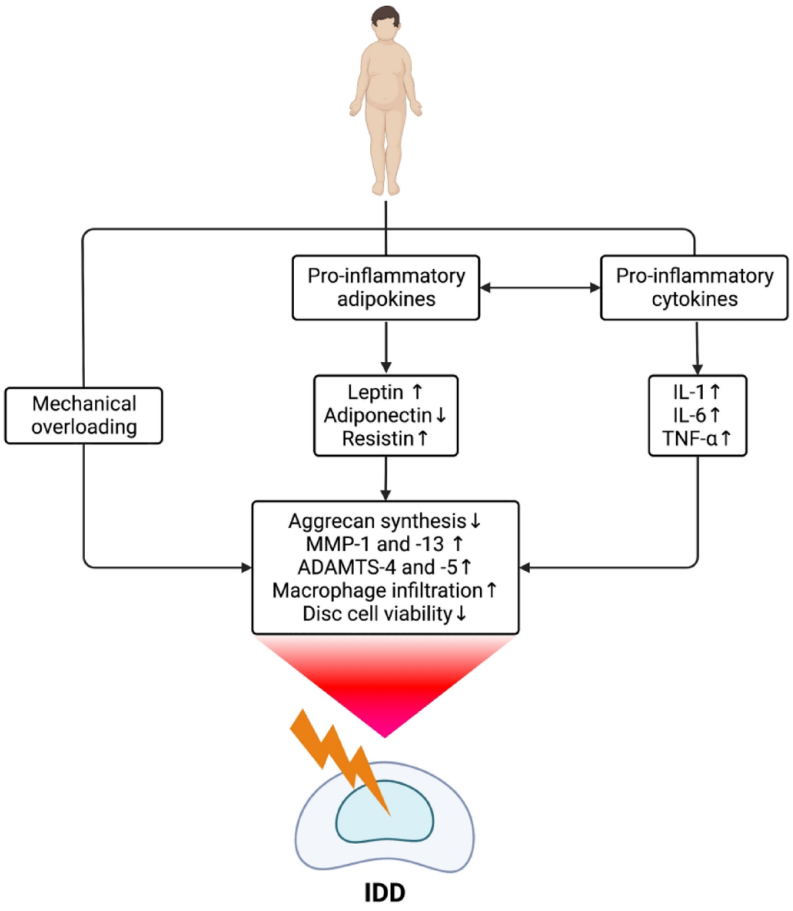

Figure 1. Putative mechanisms by which overweight and obesity are correlated with IDD. Adapted with permission from Cannata *et al*.[6] Created with BioRender.com. ADAMTS, a disintegrin and metalloproteinase with thrombospondin motifs; IDD, intervertebral disc degeneration; IL, interleukin; MMP, matrix metalloproteinase; TNF, tumor necrosis factor.

establishing a vicious cycle.[66] Previous reports have shown that LBP is consistently more common in children and adolescents affected by psychosocial distress. A cross-sectional study conducted on 3485 adolescents from the Netherlands showed that the prevalence of LBP was higher in participants not living with both of their parents, reporting depressive symptoms, and experiencing regular stress.[68] Notably, a recent systematic review and meta-analysis revealed that having suffered from childhood maltreatment was a significant risk factor for the development of LBP later in life.[69]

Prevention and treatment strategies

Identification of LBP risk factors may significantly reduce the prevalence of this disorder and its burden on the pediatric population.[70] Obesity appears to be a major risk factor for the development of LBP in childhood and adolescence.[71] Hence, planning adequate prevention and treatment strategies would be crucial to prevent the negative consequences of persisting LBP in adulthood, with associated functional limitations and financial losses.[72]

Obesity is a complex, multifactorial condition that has multiple determinants including genetic, physiological, socioeconomic, and environmental factors, demonstrating considerable heterogeneity in response to a given intervention.[73] Lifestyle modification therapy, mainly based on dietary adjustments and promotion of physical activity, is the necessary first step of obesity treatment, useful in improving many risk factors and obesity-related comorbidities, but often not successful in achieving clinically meaningful and long- lasting weight loss.[73] When lifestyle modifications fail, second- and third-level treatments, such as drugs and bariatric surgery, respectively, should be considered, although

potential risks have to be taken into account.[74] Recently, new drugs approved for use in adolescents >12 years old have been reported to be safe and effective in promoting significant weight loss (i.e., glucagon-like peptide [GLP]-1 receptor agonists). Maintaining a healthy weight from an early age and implementing all therapeutic interventions currently available with a personalized approach to the patient are the only strategies to reduce the prevalence of obesity and the risk of chronic complications, including LBP, in the adult age.[75]

Dietary adjustments

An unhealthy diet characterized by excessive calorie intake and a nutrient imbalance is the main risk factor associated with overweight and obesity in children and adolescents. According to the Academy of Nutrition and Dietetics, the Society for Nutrition Education and Behavior, and the School Nutrition Association, nutrition interventions should be implemented through school-based and multidisciplinary programs including nutrition education in classrooms, modifications of school policies, and especially methods for parental involvement. Entailing families and setting realistic goals appear to be imperative to achieve permanent changes in children eating habits and lifestyle that may last throughout adulthood. Educational programs should start with the assessment of children and family dietary habits (i.e., meal composition, frequency of food intake, size of portions, food preferences, and cooking methods).[73,76] Eating five meals per day, having an adequate breakfast, avoiding eating between meals as well as high-energy and low-nutrient density food, increasing the intake of fruit, vegetables, and fiber-rich food, and limiting portions have been considered the principal dietary recommendations, based on the cornerstones of the Mediterranean diet.[77] Therefore, a balanced and varied diet is strongly advised as a preventive and/or treatment strategy for the management of overweight/obesity and its complications in the pediatric age. Although no mechanistic relationship has been confirmed between hypovitaminosis D and LBP, it is recommended to maintain sufficient vitamin D levels during childhood and adolescence to ensure an adequate musculoskeletal and systemic development.[78]

Physical activity

The combination of diet and physical activity is crucial for the management of overweight and obesity in children and adolescents.[79] Previous studies revealed that physical activity is one of the most important factors to mitigate the risk of LBP in the pediatric age, as being physically active holds a prominent role in the prevention and management of LBP, due to its protective role on spinal structures.[79,80]

Although the ideal exercise protocol is still needed to be proven, previous reports demonstrated an inverse relationship between the intensity of physical activity and the severity of LBP.[79] Indeed, it has also been reported that low trunk muscle strength levels were more frequently associated with LBP in children.[81,82] The increase in physical activity levels can be achieved starting from the age of 2–3 years by active play, walking, swimming, and tricycle riding; at 5–6 years of age, gradual sports participation should be promoted two or three times a week. Exercise should primarily be focused on training aerobic capacity, as well as strength and flexibility while being adequate to the child's skills and stage of physical and psychomotor development.[73] Nonetheless, along with physical inactivity, extreme and unsupervised exercise should be also avoided as they may both cause LBP.[83]

According to a recent systematic review from Kędra *et al.*,[84] moderate evidence demonstrates that physical activity has a beneficial effect on LBP in children and adolescents. Interestingly, authors reported that both extreme levels of physical activity (i.e., sedentary lifestyle vs. strenuous exercise) are associated with LBP, following a U-shaped correlation. This highlights the significant importance of quality over quantity of exercise performed.

Conservative treatments for nonspecific LBP in the pediatric age

Considering that LBP in children and adolescents is usually nonspecific and transient, it is often treated conservatively. Treatment of LBP in the pediatric age is multidisciplinary and may include relative rest, exercise, physical therapy, and analgesic drugs (e.g., paracetamol, nonsteroidal analgesic drugs, etc.).[85]

The importance of physical exercise has been outlined above and confirmed in several studies. Interestingly, these papers pointed out the effectiveness of both supervised and free exercise in treating LBP, whereas passive educational

programs and ergonomic supports were not able to prevent LBP.[3,84,86] A recent systematic review and meta-analysis has demonstrated that physical therapy (including postural hygiene, specific exercises, etc.) was able to significantly enhance behavior, knowledge, trunk flexion endurance, trunk extension endurance, posture, and hamstring flexibility.[86] Therefore, pediatric patients should be actively involved in tailored programs mainly aimed at core strengthening and improvement of lower extremity flexibility.

Discussion

According to previous reports, overweight and obesity in the pediatric age appear among the main risk factors for the development of LBP.[71] Indeed, LBP is more frequent than conventionally expected in both children and adolescents and is associated with significant socioeconomic consequences,[15] apart from significantly increasing the risk of developing chronic LBP later in life.[4] Excluding specific and occasionally severe causes of LBP, most cases of LBP in overweight children and adolescents are nonspecific and have been associated with a wide range of possible causes, including anthropometric features,[2,22] posture misalignment,[24] physical inactivity,[26] hypovitaminosis D,[57] and lumbar spine overloading.[51] Collectively, these factors may contribute to the premature onset of IDD, which is the main cause of nonspecific LBP in adults,[66] while also being increasingly reported in the pediatric age.[46,56]

Considering the risk of propagating LBP through adulthood, it is essential to plan effective prevention and treatment strategies to reduce the burden of this condition on the pediatric population. Opportunely, most strategies are conservative and essentially consist in lifestyle changes that share the main goals of achieving a healthy weight[75] and maintaining adequate physical fitness.[84] Nutrition education with appropriate dietary adjustments and active involvement in sports activities is fundamental. Noteworthily, dietary plans should ensure an adequate amount of both macro- and micronutrients according to the individual's age and needs.[77] As the role of schoolbag weight in causing LBP is still not well understood, it may be reasonable to keep it under 10–15% of the child's body weight, as suggested by most guidelines.[87] In case of acute LBP seeking medical attention, after excluding specific causes, a conservative treatment consisting of a short course of analgesics and physical therapy may be initiated.[85] However, as the effectiveness of most treatments has been evaluated in adults, it should be considered that the spine of a child and adolescent is physiologically different from the adult spine (ligamentous laxity, bone architecture, muscle mass).[3] Therefore, the development of dedicated treatment guidelines to tackle LBP in the pediatric age is urgently needed.

Particular attention should be paid to the mental well-being of overweight/obese children and adolescents with LBP. Psychosocial factors such as depression, social isolation, and anxiety are well-known risk factors for LBP[88] as well as more commonly described in the young overweight and obese compared to normal-weight peers.[89,90] In this subpopulation, LBP may contribute to further promoting physical inactivity through fear avoidance, pain catastrophizing, and kinesiophobia mechanisms.[88] Eventually, this may create a vicious cycle in which the lack of physical exercise leads to reduced social participation, lower calorie expenditure, poorer physical fitness, and ultimately worsening LBP with the risk of additional weight gain. This subset of patients should be promptly recognized to plan a personalized treatment strategy including cognitive behavioral therapy, dietary advice, and supervised physical exercise.[88,91] In this regard, a recent study showed that the implementation of a regular physical exercise program ameliorated emotional well-being, self-perception, and self-concept in children with obesity and concurrent anxiety and depression.[92] Involving families and introducing appropriately defined school education programs would be of great value in improving the care of LBP. Interestingly, previous studies have demonstrated that Back School and postural education programs in school-aged children and adolescents resulted in healthier backpack use,[93] better ergonomic knowledge,[94] and lower self-reported LBP rates.[95]

This study has some limitations. First, the narrative nature of this review is not able to provide an analytical evaluation of mentioned studies, although yielding a comprehensive outlook on the topic, which was missing in the literature. While systematic and scoping reviews are inherently characterized by a stronger methodology and reliability when performing our search, we realized that the topic was significantly wide, underexplored, and populated with low-evidence studies. In this regard, considering the variety of different

aspects involved and the low level of the available evidence, we preferred to perform a narrative review to preliminarily scrutinize the state of the evidence. Systematic reviews on the diverse domains explored in this preliminary work have been planned by our team and will be performed in the future. Second, the majority of presented data are derived from retrospective cohort studies or cross-sectional studies, which inherently lack adequate statistical power and unbiased interpretability, therefore limiting the reliability of their results.

Conclusions

LBP is a common condition in the pediatric age, especially in overweight and obese subjects. Considering the risk of persisting LBP into adulthood, as well as the detrimental consequences of excessive body weight, it is essential to plan adequate prevention and treatment strategies as early as during childhood. These mainly entail weight loss through diet optimization, promotion of an active lifestyle, and preservation of mental well-being. Although several factors are involved in the pathophysiology of pediatric nonspecific LBP, the available evidence is fragmentary and mainly composed of nonrandomized cohort studies and cross-sectional analyses, which significantly limit the interpretability and generalizability of current knowledge. Therefore, future studies and randomized clinical trials should be oriented toward the implementation of specific treatments and preventive strategies to reduce the burden of LBP in pediatric individuals with overweight and obesity.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Author contributions

Luca Ambrosio: Conceptualization; Data curation; Investigation; Methodology; Writing – original draft; Writing – review & editing.

Giorgia Mazzuca: Conceptualization; Formal analysis; Investigation; Methodology; Writing – original draft; Writing – review & editing.

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Fabrizio Russo: Investigation; Visualization; Writing – review & editing.

Francesca Cannata: Investigation; Visualization; Writing – review & editing.

Gianluca Vadalà: Investigation; Visualization; Writing – review & editing.

Claudio Maffeis: Conceptualization; Supervision; Visualization; Writing – review & editing.

Rocco Papalia: Investigation; Supervision; Visualization; Writing – review & editing.

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