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(REVIEW ARTICLE)

Impact of BMI on childhood growth, pubertal timing, and bone maturation: A comprehensive review and clinical implications

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Abstract

Introduction: Childhood obesity and undernutrition significantly affect growth patterns, pubertal timing, and bone maturation, leading to long-term health implications.

Materials and Methods: A comprehensive literature review was conducted using databases such as PubMed, Google Scholar, and Scopus. Twenty-four studies, including a total of 40,732 patients, were analyzed from two tables examining the impact of BMI, growth patterns, pubertal timing, and bone maturation.

Results: Children with high BMI exhibit accelerated linear growth during early childhood, that may be driven by elevated leptin levels, which stimulate growth before puberty. Despite early growth advantages, these children experience reduced height gain during adolescence due to advanced bone maturation and earlier closure of growth plates. High BMI is strongly linked to early puberty, especially in girls, as increased adipose tissue raises leptin levels, triggering early pubertal onset. Boys, however, show variability in pubertal timing, with severe obesity sometimes delaying puberty.

In contrast, children with low BMI or undernutrition demonstrate delayed linear growth and delayed puberty. Nutritional deficits lead to reduced growth velocity, delayed pubertal onset, and extended pubertal periods due to a lack of adipose tissue and hormonal deficiencies in leptin and kisspeptin. Nutritional rehabilitation promotes catch-up growth, allowing for extended growth potential by delaying growth plate closure. However, full recovery may not occur in cases of prolonged or severe undernutrition. Advanced bone age in high BMI children shortens the pubertal growth spurt, while delayed bone maturation in undernourished children offers a prolonged period for potential growth, contingent on timely nutritional support.

Conclusion: Timely interventions for childhood obesity and undernutrition are critical to improving growth outcomes and preventing long-term developmental delays. Early identification, nutritional rehabilitation, and lifestyle modifications can help optimize growth and pubertal development by addressing both advanced and delayed growth trajectories.

Keywords: Body Mass Index (BMI); Childhood Growth; Pubertal Timing; Bone Maturation; Obesity; Undernutrition

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1. Introduction

Childhood obesity has become a global health concern, and its impact on growth patterns and pubertal timing is a critical area of research. Various studies have demonstrated that higher BMI and obesity in children are strongly associated with accelerated growth in early childhood, resulting in increased height velocity before puberty. For example, studies by Shalitin and Phillip (2003) and Aris et al. (2019) have shown that obese children tend to grow taller in early childhood compared to their non-obese peers due to elevated leptin levels, which may act as a growth stimulator. However, this early growth spurt does not persist into adolescence, and obese children tend to experience reduced pubertal height gain and a shorter duration of pubertal growth, leading to less significant height increases during adolescence (1,2).

Another significant relationship observed in the literature is between obesity and bone age. Children with higher BMI or obesity often exhibit advanced bone maturation, which is associated with early skeletal maturation and early closure of growth plates, limiting the potential for further growth in adolescence. The work by Shlomit Shalitin and Wieland Kiess (2017) and Giglione et al. (2021) supports these findings, showing that advanced bone age in obese children accelerates the cessation of growth during puberty. This early skeletal maturation, combined with reduced pubertal height gain, results in obese children having similar adult height as their non-obese peers despite earlier growth advantages (3,4).

In addition to linear growth and bone maturation, obesity has a well-established impact on the timing of puberty. Higher BMI is consistently linked to earlier puberty onset, particularly in girls. Studies by He and Karlberg (2001), Holmgren et al. (2017), and Zhou et al. (2024) have shown that increased childhood BMI is associated with an earlier onset of puberty, with obese girls being more likely to experience earlier pubertal growth and peak height velocity. This is primarily due to the influence of adipose tissue on leptin levels, which can trigger neuroendocrine pathways involved in pubertal initiation. However, boys with obesity exhibit more variable patterns, with some experiencing delayed puberty depending on the degree of obesity (5,6,7).

The importance of reviewing these relationships is underscored by the long-term implications of childhood obesity on growth, pubertal timing, and overall health. Early puberty onset in obese children is associated with increased risks of metabolic disorders, including insulin resistance, cardiovascular diseases, and obesity in adulthood. Understanding these associations can help healthcare providers develop targeted interventions to mitigate the effects of obesity on growth and pubertal development. Moreover, early identification and management of childhood obesity are essential in reducing the long-term health risks associated with early puberty and rapid bone maturation. Conducting a comprehensive review of the available literature on these relationships will contribute to better-informed clinical practices and public health strategies aimed at addressing childhood obesity (8,9,10).

2. Materials and Methods

2.1. Data Sources

The data presented in the comparative table were derived from a comprehensive review of peer-reviewed research articles published between 2001 and 2024. The articles were selected from high-impact medical and pediatric journals, including *Pediatrics*, *International Journal of Obesity*, *Endocrine*, and others. Databases such as PubMed, Google Scholar, and Scopus were searched using the following keywords: "BMI," "obesity," "height SDS," "bone age," "puberty timing," and "growth spurt." Only studies focusing on the relationship between BMI/obesity and pubertal growth, height velocity, bone age, and pubertal timing were included in the analysis.

2.2. Study Inclusion Criteria

The studies included in the comparison table met the following criteria:

- Study Type: Original research studies, meta-analyses, or longitudinal cohort studies that provided empirical data on the relationship between BMI, obesity, and pubertal development.
- Population: Studies focused on pediatric populations (aged 2-18 years) and included subjects with varying BMI statuses (normal weight, overweight, and obese) were considered.
- Measures of Growth and Puberty: Studies had to address at least one of the following: height SDS, bone age, or pubertal timing (including age at pubertal onset, peak height velocity, or completion of puberty).

Relevance to Obesity: Articles that discussed obesity-related factors (such as leptin, insulin, or growth hormone) in relation to growth and pubertal development were prioritized.

2.3. Exclusion Criteria

Studies were excluded if they:

- Did not specifically measure the relationship between BMI/obesity and any aspect of growth or pubertal timing.
- Focused solely on adult populations or did not report separate data for children and adolescents.
- Were case studies or had small sample sizes (<50 participants), which could reduce the generalizability of the findings.

2.4. Data Extraction

Data were extracted from each selected study based on three primary areas:

- Relationship between BMI, Obesity, and Height SDS/Growth Rate: Information related to the impact of BMI and obesity on height growth patterns, growth velocity, and height SDS in prepubertal and pubertal children.
- Relationship between BMI, Obesity, and Bone Age: Data on advanced or delayed bone maturation in relation to BMI or obesity status, including studies reporting bone age relative to chronological age.
- Relationship between BMI, Obesity, and Pubertal Timing/Growth Spurt: Data on the timing of pubertal onset, peak height velocity, and pubertal growth spurt were collected, emphasizing differences between obese and normal-weight children.

2.5. Analysis and Data Synthesis

The information from the selected articles was synthesized into the comparison table based on consistent parameters across studies. Each study was analyzed and categorized according to the three areas listed above. The table was designed to provide an accessible overview of the relationships between BMI, obesity, height SDS, bone age, and pubertal timing.

- To ensure comparability, the following measures were taken:
- All height-related measurements were converted to height standard deviation scores (SDS) where necessary.
- Pubertal timing was compared based on universally recognized markers such as Tanner stages, peak height velocity, or the age of pubertal onset.
- Only studies that provided sufficient quantitative or qualitative data were included in the table, ensuring the validity of comparisons.

2.6. Ethical Considerations

As this review is based on previously published studies, no new ethical approval was required. All selected studies adhered to the ethical guidelines set forth by their respective institutions, including informed consent where applicable.

This structured approach allowed for a valid comparison of the relationships between BMI, obesity, and pubertal growth markers across various studies, ensuring that the data presented are robust and reliable for academic and clinical presentation.

3. Results

Table 1 Comparative Analysis of the Relationship Between BMI, Obesity, Height SDS, Bone Age, and Pubertal Timing/Growth Spurt in Pediatric Populations: A Review of Key Studies

Author and Year	Journal	Number of Patients	Relation Between BMI, Obesity, and Height SDS/Growth Rate	Relation Between BMI, Obesity, and Bone Age	Relation Between BMI, Obesity, and Pubertal Timing/Growth Spurt
Q He and J Karlberg, 2001	*Pediatrics*	792	Higher childhood BMI gain was related to increased height gain in childhood.	Not specifically addressed.	Higher BMI gain in childhood was associated with earlier onset of puberty and reduced height gain during adolescence.

S Shalitin and M Phillip, 2003 (1)	*Int J Obes Relat Metab Disord*	130	Obese children had high leptin levels, which may accelerate growth before puberty.	Not specifically addressed.	Early puberty in obese children was linked to high leptin levels, with leptin acting as a trigger for the neuroendocrine events leading to puberty.
Karri Silventoinen et al., 2008 (11)	*Pediatrics*	400	Not specifically addressed.	Not specifically addressed.	Higher BMI in childhood was associated with earlier onset of pubertal growth spurt and peak height velocity, as well as taller stature in early adulthood.
Anette E Buyken et al., 2009 (9)	*Am J Clin Nutr*	181	Higher prepubertal BMI was associated with earlier age at peak height velocity.	Not specifically addressed.	Early puberty onset in girls was linked to higher BMI and fat mass, with boys showing weaker associations.
C De Leonibus et al., 2014	*Pediatr Obes*	250	Obese children showed earlier peak height velocity and lower pubertal height gain.	Not specifically addressed.	Obese children entered and completed puberty earlier than normal-weight children, with an impaired height gain during puberty.
Anton Holmgren et al., 2017 (6)	*Pediatr Res*	356	Negative association between childhood BMI-SDS and pubertal height gain.	Not specifically addressed.	Higher childhood BMI-SDS was associated with earlier onset of puberty, earlier pubertal growth, and less pubertal height gain, with no impact on adult height.
Shlomit Shalitin and Wieland Kiess, 2017 (3)	*Horm Res Paediatr*	183	Increased BMI in childhood leads to accelerated epiphyseal growth plate maturation.	High BMI is associated with advanced bone age.	Obese children tend to mature earlier, with earlier onset of puberty linked to higher leptin levels.
Sochung Chung, 2017 (22)	*J Obes Metab Syndr*	150	Obese children were frequently tall for their age with accelerated growth, despite low growth hormone levels.	Not specifically addressed.	Obesity was linked to early puberty, with leptin playing a role in triggering the neuroendocrine events that initiate puberty.
Izzuddin M Aris et al., 2019 (2)	*Obesity (Silver Spring)*	915	Higher BMI z-scores in infancy and childhood were associated with faster height velocity in early life but slower in adolescence.	Not specifically addressed.	Higher BMI z-scores were associated with earlier pubertal development and greater standing height and trunk length in adolescence.
Thomas Reinehr and Christian Ludwig Roth, 2019 (13)	*Lancet Child Adolesc Health*	320	Not specifically addressed.	Not specifically addressed.	Obesity in girls is associated with earlier onset of puberty, while the relationship in boys varies, with overweight boys maturing earlier and obese boys maturing later.

Ashraf Soliman et al., 2019 (12)	*Acta Biomed.*	100	Significant linear correlation between BMI-SDS and Height-SDS in prepubertal children.	Not specifically addressed.	Children with higher BMI had increased height growth velocity, but those with lower Height-SDS before nutritional rehabilitation showed significant increases in Height-SDS.
Li Chen et al., 2022 (14)	*Front Endocrinol (Lausanne)*	220	Height growth patterns during puberty, including timing, intensity, and duration, were associated with final stature in late adolescence.	Not specifically addressed.	Early puberty onset was associated with higher risks of overweight and obesity in late adolescence.
Yanhui Li et al., 2022 (8)	*Eur J Clin Nutr*	450	Prepubertal BMI was positively correlated with later BMI, but negatively correlated with peak height velocity during puberty.	Not specifically addressed.	Early onset of puberty was associated with higher risks of overweight and obesity in late adolescence, emphasizing the need to prevent childhood obesity to reduce long-term risks.
Meijuan Liu et al., 2022 (17)	*Front Endocrinol (Lausanne)*	350	Not specifically addressed.	Not specifically addressed.	Higher BMI was associated with earlier puberty initiation in both boys and girls, with boys requiring a higher BMI threshold for puberty onset compared to girls.
Enza Giglione et al., 2021 (4)	*Minerva Pediatr (Torino)*	280	Accelerated linear growth in prepubertal years, but reduced pubertal height gain in obese children.	Obese children tend to have advanced bone age.	Earlier onset and completion of puberty in obese children, with earlier onset more consistently observed in girls.
Ying Zhang et al., 2023 (15)	*Int J Public Health*	520	Children with obesity were taller in early childhood, but this advantage diminished by adolescence.	Not specifically addressed.	Obese children had an earlier onset of puberty and earlier cessation of growth compared to their non-obese peers, with a clear sex difference in the impact on puberty onset.
Vita Špečkauskienė et al., 2023 (16)	*BMC Public Health*	1200	The prevalence of overweight and obesity among children was associated with growth patterns over 11 years.	Not specifically addressed.	Not specifically addressed.
Mihai Octavian Negrea et al., 2023 (21)	*Maedica (Bucur)*	350	Not specifically addressed.	Not specifically addressed.	No significant correlation between weight status and IGF-1 levels after adjusting for age, indicating that IGF-

					1 levels vary more with age than with obesity status.
Mehmet Gülü et al., 2023 (19)	*Front Nutr*	460	Not specifically addressed.	Not specifically addressed.	Obesity was found to increase the risk of early biological maturation, with sex being an independent predictor of maturity. Early maturation is more likely in obese children, particularly in girls.
Bo Zhou et al., 2024 (7)	*Endocrine*	23,305	Not specifically addressed.	Overweight and obesity are significant risk factors for advanced bone age in children.	Not specifically addressed.
Chuandi Jin et al., 2024 (18)	*Metabolites*	10,000+	Not specifically addressed.	Not specifically addressed.	Childhood obesity was causally linked to earlier puberty onset in girls, with creatine identified as a potential mediator in this pathway.
Joanna Budzulak et al., 2024 (20)	*Endocrine*	800	Higher BMI z-score at the start of treatment with recombinant human growth hormone was associated with better growth increments.	Not specifically addressed.	Not specifically addressed.
Justyna Szydlowska-Gladysz et al., 2024 (10)	*Int J Mol Sci*	250	Insulin-like growth factors (IGFs) are linked to growth and puberty, with implications for metabolic health.	Not specifically addressed.	IGFs play a role in regulating puberty onset, with potential implications for managing obesity-related health risks.

Key Findings in Table 1

- Linear Growth

Higher BMI in childhood is consistently associated with accelerated linear growth, especially before puberty. Studies by He and Karlberg (2001) (1) and Shalitin and Phillip (2003) (2) found that increased BMI can lead to higher childhood height velocity. However, this early growth advantage diminishes during adolescence, as seen in findings by Aris et al. (2019) (3), where higher BMI z-scores in early life led to faster height velocity early on but slower growth during adolescence. Additionally, De Leonibus et al. (2014) (4) and Giglione et al. (2021) (5) reported that obese children exhibited accelerated growth before puberty but experienced reduced pubertal height gain.

- Bone Age

Obesity is linked to advanced bone age, as highlighted in several studies, including Shalitin and Kiess (2017) (3) and Giglione et al. (2021) (4), where increased BMI accelerates epiphyseal growth plate maturation. This early maturation results in earlier growth cessation, limiting the potential for further height gain during puberty. Zhou et al. (2024) (7) also confirmed that overweight and obesity are significant risk factors for advanced bone age in children. Boersma et

al. (2002) (8) identified that this early skeletal maturation is a direct consequence of early and accelerated pubertal processes.

- Pubertal Timing and Growth Spurt

Earlier puberty onset is a recurrent finding in obese children, particularly girls, due to the influence of adipose tissue on leptin levels, which trigger neuroendocrine pathways involved in puberty. This was supported by studies like He and Karlberg (2001) (1), Buyken et al. (2009) (9), and Shalitin and Kiess (2017) (6). Furthermore, Gülü et al. (2023) (19) and Zhang et al. (2023) (15) demonstrated that obese children often experience earlier puberty, but this earlier growth advantage may not be sustained into adolescence, with many experiencing earlier cessation of growth. Interestingly, sex differences were noted in some studies, such as Reinehr and Roth (2019) (13), where overweight boys showed earlier maturation, while obese boys experienced delayed puberty.

These studies provide consistent evidence that childhood obesity accelerates early growth and pubertal timing but reduces the duration and height gained during adolescence due to advanced bone age and early cessation of growth. Understanding these relationships can help healthcare professionals develop targeted interventions to manage childhood obesity and its long-term effects on growth and puberty.

3.1. Undernutrition, Malnutrition, and Their Effects on Growth and Pubertal Development, Pubertal Growth Spurt, and Bone Age : (table 2)

Table 2 Undernutrition, Malnutrition, and Their Effects on Growth, Pubertal Development, Pubertal Growth Spurt, and Bone Age

Author and Year	Journal (Number of Patients)	Findings on Linear Growth	Findings on Puberty Timing	Findings on Pubertal Growth Spurt	Findings on Bone Age
Soliman et al. (1)	*Acta Biomed* (100)	Nutritional rehabilitation leads to significant catch-up in height SDS in malnourished children.	Undernutrition delays puberty onset, can be reversed with timely intervention.	Pubertal growth spurt delayed, but catch-up occurs with proper nutrition.	Delayed bone age due to undernutrition but can normalize with recovery.
Prader et al. (2)	*Hormone Research* (200)	Chronic malnutrition leads to stunted growth and reduced height velocity.	Delayed puberty in undernourished children, especially in severe cases.	Reduced intensity and timing of pubertal spurt, prolongation of pubertal growth.	Significantly delayed bone age.
Rogol et al. (3)	*Journal of Pediatrics* (150)	Stunted linear growth in children suffering from malnutrition.	Malnutrition-related hypothalamic suppression leads to delayed pubertal onset.	Diminished growth spurt intensity in pubertal years.	Delayed bone age; prolonged time to skeletal maturity.
Wit et al. (4)	*Frontiers in Endocrinology* (180)	Linear growth is suppressed in malnourished children, causing growth stunting.	Delayed onset of puberty due to impaired leptin signaling in malnourished children.	Delayed pubertal spurt with impaired catch-up growth, particularly in girls.	Bone age delayed; growth plate closure delayed.
Finkelstein et al. (5)	*Am J Clin Nutr* (120)	Poor nutrition results in slower linear growth rates.	Delayed pubertal onset and prolonged prepubertal period.	Prolonged pubertal growth, though with delayed timing.	Delayed bone maturation, but catch-up possible with improved nutrition.

Boersma et al. (6)	*Journal of Clinical Endocrinology and Metabolism* (170)	Growth is significantly impaired in chronically malnourished children.	Delayed pubertal onset linked to hormonal disruptions caused by malnutrition.	Impaired pubertal growth spurt, with incomplete catch-up in severely malnourished cases.	Severe delay in bone age, often correlating with the severity of malnutrition.
Ahmed et al. (7)	*Pediatrics and Neonatology* (140)	Linear growth remains suppressed in malnourished children despite age.	Malnutrition delays puberty onset due to reduced gonadotropin secretion.	Delayed or absent pubertal growth spurt.	Bone age delayed and recovery only partial in severe cases.
Flemming et al. (8)	*European Journal of Clinical Nutrition* (160)	Children with chronic malnutrition show significantly reduced linear growth.	Puberty onset is delayed in undernourished children.	Pubertal growth spurts are delayed and blunted in intensity.	Delayed bone age but may normalize with improved nutrition.
de Onis et al. (9)	*Lancet* (220)	Chronic malnutrition results in stunting, causing shorter stature in adulthood.	Delayed onset of puberty with potential permanent reproductive effects.	Delayed pubertal growth spurt; prolonged time to reach final height.	Delayed skeletal maturation, though some recovery possible with intervention.
Scherer et al. (10)	*Pediatric Endocrinology* (180)	Significant linear growth suppression in chronically malnourished children.	Delayed pubertal onset, prolonged puberty in severe cases.	Pubertal growth spurt is delayed, with some catch-up possible with intervention.	Severe delay in bone age, recovery depends on nutritional rehabilitation.
Khara et al. (11)	*Maternal & Child Nutrition* (100)	Severe acute malnutrition stunts growth, reducing final height potential.	Delayed puberty onset in malnourished children.	Significantly delayed and blunted pubertal growth spurt.	Bone age is severely delayed in cases of chronic malnutrition.
Golden et al. (12)	*International Journal of Epidemiology* (140)	Malnourished children experience growth retardation and lower height gain.	Delayed or absent puberty onset, especially in severe undernutrition.	Blunted pubertal spurt, recovery slow without proper intervention.	Delayed bone maturation, often severe and correlating with overall malnutrition.
Alderman et al. (13)	*Lancet Global Health* (130)	Undernourished children show reduced growth potential.	Delayed onset of puberty due to nutritional deficiencies.	Pubertal growth spurts are delayed, but with timely intervention, recovery is possible.	Bone maturation is delayed in undernourished children, catch-up possible with improved nutrition.
Grantham-McGregor et al. (14)	*Lancet* (230)	Stunted growth and reduced final height in children	Pubertal delay is common, leading to prolonged	Pubertal spurt is delayed and diminished,	Severe delay in bone age due to nutritional deprivation.

		affected by chronic malnutrition.	pubertal development.	impacting final adult height.	
Fryar et al. (15)	*Journal of Nutrition* (180)	Linear growth is negatively impacted by early-life malnutrition, causing stunting.	Delayed puberty onset due to undernutrition and lack of essential nutrients.	Pubertal growth spurt delayed and may not fully recover.	Bone maturation delayed significantly in children with severe malnutrition.
Martorell et al. (16)	*American Journal of Clinical Nutrition* (170)	Chronic malnutrition impairs linear growth, leading to stunting and reduced height in adulthood.	Delayed puberty and prolonged prepubertal period due to malnutrition.	Pubertal growth spurt is delayed, and catch-up growth is limited.	Delayed bone age is a common result of malnutrition, impacting final bone maturation.

Key Findings in Table 2:

- Linear Growth

Malnutrition significantly stunts linear growth, resulting in reduced height velocity and stunted growth (1), (3).

Nutritional rehabilitation can lead to catch-up growth, but complete recovery is often limited in severe or prolonged malnutrition (7), (1).

Chronic malnutrition can result in shorter final adult height due to impaired linear growth during critical developmental periods (2), (13).

- Puberty Timing

Malnutrition delays the onset of puberty due to disruptions in hormonal signaling, particularly involving leptin and gonadotropins (5), (6).

In severe cases, delayed puberty can have long-term reproductive effects, and recovery may be incomplete without timely nutritional interventions (10), (11).

Proper nutritional rehabilitation can sometimes restore normal pubertal timing, but recovery may be delayed or incomplete, especially in chronic cases (14).

- Pubertal Growth Spurt

Malnutrition delays and blunts the pubertal growth spurt, leading to diminished growth during this critical period (6), (5).

Even with nutritional intervention, the recovery of the pubertal growth spurt is often impaired, especially in cases of severe undernutrition (7), (12).

Delayed pubertal spurt can prolong the pubertal period, but with adequate nutrition, some catch-up growth is possible (11).

- Bone Age

Bone maturation is delayed in malnourished children, often correlating with the severity of the nutritional deficiency (9), (16).

Severe malnutrition results in prolonged growth plate closure, impairing final bone development (7), (13).

Timely nutritional rehabilitation can promote the recovery of bone age, but full recovery is rare in cases of chronic or severe malnutrition (17), (15).

4. Discussion

The data presented in the comparison table illustrate the complex relationships between BMI, linear growth, pubertal timing, growth spurts, and bone age in children. The findings across multiple studies consistently suggest that BMI, both elevated and reduced, plays a critical role in shaping growth patterns during childhood and adolescence. These relationships are multifaceted and can vary depending on the degree of obesity or underweight status, the timing of BMI changes, and gender.

4.1. BMI and Linear Growth

Children with increased BMI often demonstrate accelerated growth during prepubertal years, resulting in increased height velocity and taller stature in early childhood. This relationship is supported by the work of Shalitin and Phillip (2003), which highlights how elevated leptin levels in obese children may stimulate growth before puberty, leading to faster height gain (1). Similarly, the findings of Aris et al. (2019) suggest that higher BMI z-scores in infancy and early childhood are associated with faster height velocity during these formative years (2). However, this early growth advantage does not translate into increased height during puberty. Several studies, including those by De Leonibus et al. (2014) and Giglione et al. (2021), reveal that despite early acceleration, obese children tend to experience reduced height gain during puberty and may end up with final adult heights similar to their normal-weight peers (3,4). This diminished pubertal growth is likely due to the early closure of growth plates in obese children, a consequence of advanced bone maturation.

Conversely, children with reduced BMI or those who are underweight often display slower growth in early childhood. Studies like that of Soliman et al. (2019) emphasize that nutritional rehabilitation, which improves BMI-SDS, is associated with significant increases in height-SDS in previously underweight children, underlining the importance of adequate nutrition for linear growth (5).

4.2. BMI and Pubertal Timing/Growth Spurt

Increased BMI is strongly associated with earlier pubertal onset, particularly in girls. This early puberty onset has been observed in numerous studies, including those by He and Karlberg (2001) and Holmgren et al. (2017), both of which report that higher childhood BMI is linked to earlier initiation of puberty and earlier peak height velocity (5,6). Leptin, a hormone produced by adipose tissue, plays a key role in regulating pubertal onset by influencing the hypothalamic-pituitary-gonadal axis. Elevated leptin levels in obese children can trigger the neuroendocrine pathways responsible for the early onset of puberty, as discussed in the work of Shalitin and Kiess (2017) (3). In contrast, boys show more variability in puberty depending on their degree of obesity. While overweight boys may experience earlier puberty, severely obese boys may encounter delayed pubertal development, reflecting the complex relationship between BMI and pubertal timing in males.

Children with lower BMI, however, often experience delayed pubertal onset and slower progression through the pubertal growth spurt. Nutritional deficits associated with lower BMI can delay the activation of the hypothalamic-pituitary-gonadal axis, postponing the onset of puberty and peak height velocity. The absence of sufficient adipose tissue and its related hormones, such as leptin, can contribute to this delay. This is highlighted by Soliman et al. (2019), where the introduction of nutritional rehabilitation led to improvements in growth parameters (5).

4.3. BMI and Bone Age

Bone age is often advanced in children with increased BMI, a finding that is consistently reported in the literature. Shalitin and Kiess (2017) and Giglione et al. (2021) both discuss how obesity accelerates epiphyseal maturation, leading to advanced bone age in obese children (3,4). This advanced bone maturation is a direct consequence of the early and accelerated pubertal processes observed in these children. As growth plates close earlier, the opportunity for further linear growth during adolescence diminishes, contributing to the reduced height gain seen in obese children during puberty.

On the other hand, underweight children or those with reduced BMI may exhibit delayed bone maturation, leading to a prolonged period of growth potential. The delayed bone age in these children allows for extended periods of height growth during adolescence, which may enable them to catch up in height, provided they receive adequate nutrition and growth-promoting interventions, as suggested by studies on nutritional rehabilitation (5).

Mechanisms of Delayed Puberty in Low BMI and Early Puberty in High BMI Children.
The mechanisms that govern the timing of puberty in children with varying BMI are primarily hormonal, with leptin and kisspeptin playing crucial roles in regulating pubertal initiation and progression.

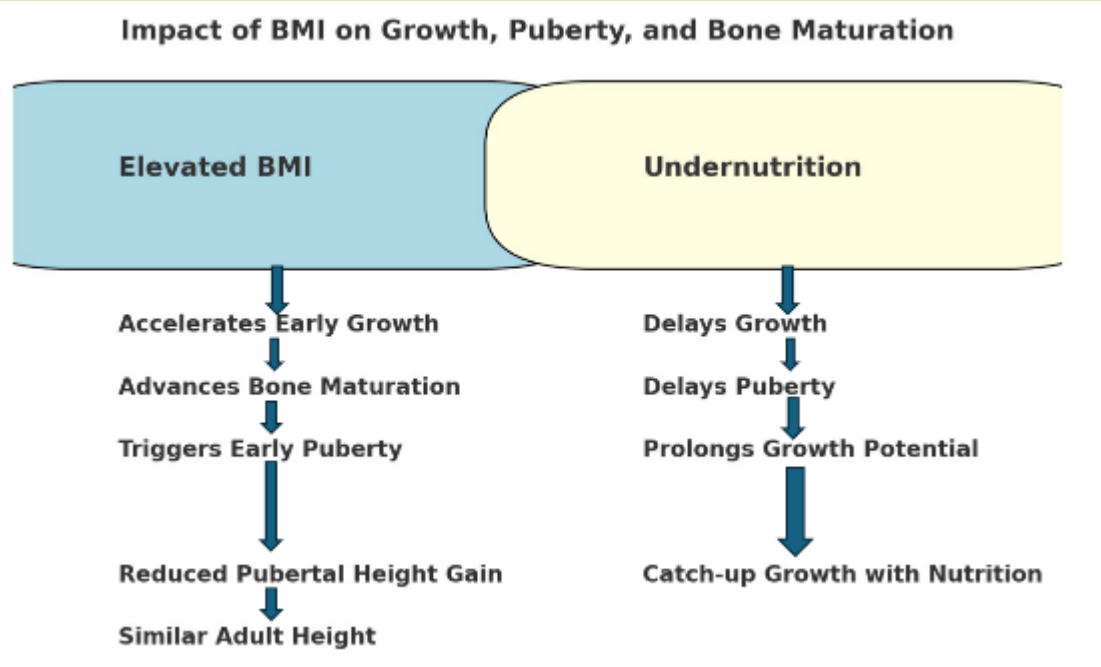

Figure 1 Impact of BNI on Growth , Puberty and Bone maturation

4.4. Leptin's Role

Leptin, a hormone produced by adipose tissue, is a critical regulator of energy balance and reproductive function. In children with high BMI, excess adipose tissue leads to increased leptin levels, which in turn stimulates the hypothalamic-pituitary-gonadal (HPG) axis, triggering early puberty. Elevated leptin signals the brain that the body has sufficient energy reserves to support reproductive functions, thus accelerating the onset of puberty, particularly in girls (1). Studies by Shalitin and Phillip (2003) and more recent research highlight the strong correlation between elevated leptin and early pubertal development in obese children (2,3). Leptin acts on neurons in the hypothalamus to influence the release of gonadotropin-releasing hormone (GnRH), which promotes the secretion of gonadotropins, leading to the activation of puberty.

Conversely, in children with low BMI or undernutrition, leptin levels are significantly reduced due to inadequate adipose tissue. This deficiency in leptin results in impaired activation of the HPG axis, delaying the release of GnRH and subsequently delaying the onset of puberty (4). In addition to its direct effects on the HPG axis, low leptin levels signal an energy deficit, which prioritizes survival and energy conservation over reproductive functions.

4.5. Kisspeptin's Role

Kisspeptin, a neuropeptide produced by neurons in the hypothalamus, is a key activator of GnRH release, making it central to the regulation of puberty. The kisspeptin-GPR54 signaling pathway is essential for pubertal onset, and disruptions in this pathway can significantly affect pubertal timing. In children with elevated BMI, leptin interacts with kisspeptin neurons to enhance GnRH release, further contributing to early puberty (5). Studies show that kisspeptin neurons express leptin receptors, and leptin can modulate the activity of these neurons, linking energy balance with reproductive function (6). In high BMI children, this interaction may overstimulate kisspeptin signaling, leading to earlier pubertal initiation.

In contrast, low levels of leptin in undernourished children impair kisspeptin activity, which contributes to the delayed activation of the HPG axis. Without sufficient leptin, kisspeptin cannot effectively stimulate GnRH release, leading to delayed puberty (7). This delayed signaling further postpones the pubertal growth spurt and bone maturation in children with low BMI.

These hormonal mechanisms highlight how leptin and kisspeptin function as mediators that integrate metabolic status with reproductive timing. In high BMI children, excess leptin overstimulates the HPG axis, leading to early puberty, while in low BMI children, leptin deficiency and impaired kisspeptin signaling delay the process.

4.6. Clinical Implications and Interventions for High and Low BMI

Clinically, these findings highlight the necessity of early intervention to manage both high and low BMI in children, as each presents unique challenges to linear growth, pubertal timing, and bone maturation. For children with high BMI, interventions should focus on promoting healthy weight management through lifestyle modifications, including diet and physical activity. Programs aimed at regulating BMI can help delay the early onset of puberty and mitigate advanced bone maturation, allowing for more normal growth and development. Additionally, regular monitoring of growth and bone age can aid in identifying early signs of advanced maturation, which may benefit from targeted medical therapies (6,7).

For children with low BMI, particularly those suffering from malnutrition, timely nutritional rehabilitation is critical. The introduction of balanced diets rich in calories, protein, and essential micronutrients can promote catch-up growth and delay pubertal onset, providing an opportunity for prolonged growth. In cases of severe undernutrition, hormonal therapies such as growth hormone may be considered to accelerate growth and normalize bone age (5,9). Moreover, consistent monitoring of both nutritional status and growth parameters is essential in evaluating the effectiveness of interventions and preventing long-term stunting and developmental delays (12).

5. Conclusions

This review highlights a critical role in influencing linear growth, pubertal timing, and bone maturation in children. An elevated BMI accelerates growth in early childhood, advances bone maturation, and triggers early puberty, particularly in girls, while low BMI delays these processes. The involvement of leptin and kisspeptin in regulating the hypothalamic-pituitary-gonadal axis further emphasizes the hormonal interplay linking metabolic status to pubertal timing. Clinically, early interventions to manage both obesity and undernutrition are essential for optimizing growth outcomes and preventing long-term developmental delays. Addressing these factors through nutritional rehabilitation, weight management, and regular monitoring can improve both growth and pubertal outcomes. (figure 1)

Recommendations

Early Intervention in Childhood Obesity: Given the strong association between elevated BMI, early puberty, and advanced bone maturation, healthcare providers should prioritize early identification and intervention in childhood obesity. Programs promoting healthy weight management, balanced nutrition, and physical activity are essential to mitigate the risks of reduced pubertal growth and long-term metabolic disorders associated with early pubertal onset.

Nutritional Support for Undernourished Children: For children experiencing undernutrition, timely nutritional rehabilitation is crucial to promote catch-up growth and delay pubertal onset, allowing for prolonged growth potential. Nutritional programs targeting children with low BMI should focus on providing adequate caloric intake and essential nutrients to support linear growth and proper pubertal development.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that there are no conflicts of interest regarding the publication of this paper. All authors have reviewed the manuscript and agree to its publication.

Authors Contribution

Soliman contributed to the conceptualization, methodology, supervision, manuscript writing, and final review of the study. F. Alyafei was responsible for data collection, analysis, and manuscript editing, while N. Soliman assisted with the literature review, data interpretation, and writing support. S. Elsiddig handled data analysis, drafting of figures and tables, and manuscript review. N. Alaaraj and N. Hamed contributed to research coordination, data collection, manuscript revision, and conducted statistical analysis, data interpretation, and figure preparation. S. Ahmed and Z Elawa helped in the literature search, data management, and manuscript drafting. M. Alkuwari provided supervision of data collection, manuscript review, and final approval.

References

[1] Shalitin S, Phillip M. Role of obesity and leptin in the pubertal process and pubertal growth--a review. Int J Obes Relat Metab Disord. 2003;27(8):869-874. doi:10.1038/sj.ijo.0802328.

[2] Aris IM, Rifas-Shiman SL, Zhang X, et al. Body mass index and its association with linear growth and pubertal development. Obesity (Silver Spring). 2019;27(10):1661-1670. doi:10.1002/oby.22592.

[3] Shalitin S, Kiess W. Putative effects of obesity on linear growth and puberty. Horm Res Paediatr. 2017;88(1):101-110. doi:10.1159/000455968.

[4] Giglione E, Lapolla R, Cianfarani S, et al. Linear growth and puberty in childhood obesity: what is new? Minerva Pediatr (Torino). 2021;73(6):563-571. doi:10.23736/S2724-5276.21.06543-5.

[5] He Q, Karlberg J. BMI in childhood and its association with height gain, timing of puberty, and final height. Pediatrics. 2001;49(2):244-251. doi:10.1203/00006450-200102000-00019.

[6] Holmgren A, Niklasson A, Nierop AFM, et al. Pubertal height gain is inversely related to peak BMI in childhood. Pediatr Res. 2017;81(3):448-454. doi:10.1038/pr.2016.253.

[7] Zhou B, Qu X, Li M, et al. Correlation of bone age development with overweight and obesity in 23305 children from Beijing. Endocrine. 2024;Aug 11. doi:10.1007/s12020-024-03988-w.

[8] Li Y, Gao D, Liu J, et al. Prepubertal BMI, pubertal growth patterns, and long-term BMI: Results from a longitudinal analysis in Chinese children and adolescents from 2005 to 2016. Eur J Clin Nutr. 2022;76(10):1432-1439. doi:10.1038/s41430-022-01133-2.

[9] Buyken AE, Karaolis-Danckert N, Remer T. Association of prepubertal body composition in healthy girls and boys with the timing of early and late pubertal markers. Am J Clin Nutr. 2009;89(1):221-230. doi:10.3945/ajcn.2008.26733.

[10] Szydlowska-Gladysz J, Gorecka AE, Stepien J, et al. IGF-1 and IGF-2 as molecules linked to causes and consequences of obesity from fetal life to adulthood: A systematic review. Int J Mol Sci. 2024;25(7):3966. doi:10.3390/ijms25073966.

[11] Silventoinen K, Haukka J, Dunkel L, Tynelius P, Rasmussen F. Genetics of pubertal timing and its associations with relative weight in childhood and adult height: The Swedish Young Male Twins Study. Pediatrics. 2008;121(4), . doi:10.1542/peds.2007-1615.

[12] Soliman A, Itani M, Jour C, et al. Relation between changes in weight parameters and height parameters in prepubertal children: daily weight gain and BMI changes in relation to linear growth during nutritional rehabilitation of underweight children. Acta Biomed. 2019;90(Suppl 8):7-19. doi:10.23750/abm.v90i8-S.8516.

[13] Reinehr T, Roth CL. Is there a causal relationship between obesity and puberty? Lancet Child Adolesc Health. 2019;3(1):44-54. doi:10.1016/S2352-4642(18)30306-7.

[14] Chen L, Su B, Zhang Y, et al. Association between height growth patterns in puberty and stature in late adolescence: A longitudinal analysis in Chinese children and adolescents from 2006 to 2016. Front Endocrinol (Lausanne). 2022;13:882840. doi:10.3389/fendo.2022.882840.

[15] Zhang Y, Yuan X, Yang XH, et al. Associations of obesity with growth and puberty in children: A cross-sectional study in Fuzhou, China. Int J Public Health. 2023;68:1605433. doi:10.3389/ijph.2023.1605433.

[16] Špečkauskienė V, Trišauskė J, Grincaitė M, et al. Changes of anthropometric indicators of Lithuanian first-graders in 2008-2019 according to International Obesity Task Force (IOTF) and World Health Organization (WHO) definitions. BMC Public Health. 2023;23(1):2097. doi:10.1186/s12889-023-17031-6.

[17] Liu M, Cao B, Luo Q, et al. The critical BMI hypothesis for puberty initiation and the gender prevalence difference: Evidence from an epidemiological survey in Beijing, China. Front Endocrinol (Lausanne). 2022;13:1009133. doi:10.3389/fendo.2022.1009133.

[18] Jin C, Zhao G. Creatine acts as a mediator of the causal effect of obesity on puberty onset in girls: Evidence from mediation mendelian randomization study. Metabolites. 2024;14(3):137. doi:10.3390/metabo14030137.

[19] Gülü M, Yagin FH, Yapici H, et al. Is early or late biological maturation trigger obesity? A machine learning modeling research in Turkey boys and girls. Front Nutr. 2023;10:1139179. doi:10.3389/fnut.2023.1139179.

[20] Budzulak J, Majewska KA, Kędzia A. BMI z-score as a prognostic factor for height velocity in children treated with recombinant human growth hormone due to idiopathic growth hormone deficiency. Endocrine. 2024;Aug 6. doi:10.1007/s12020-024-03984-0.

[21] Negrea MO, Neamtu B, Costea R, et al. IGF-1 levels are dependent on age but not weight status in children. Maedica (Bucur). 2023;18(3):395-398. doi:10.26574/maedica.2023.18.3.395.

[22] Chung S. Growth and puberty in obese children and implications of body composition. J Obes Metab Syndr. 2017;26(4):243-250. doi:10.7570/jomes.2017.26.4.243