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Secular Trends in Pubertal Timing among Ambonese Adolescents: A Comparative Analysis

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A B S T R A C T

Introduction: The timing of puberty is a crucial developmental milestone with significant implications for adolescent physical and psychosocial health. While secular trends towards earlier pubertal onset have been observed in many populations worldwide, data from Ambonese adolescents in Indonesia remains limited. This study aimed to investigate secular trends in pubertal timing among Ambonese adolescents and explore potential influencing factors. **Methods:** A retrospective cross-sectional study was conducted involving Ambonese adolescents aged 10-18 years who attended health clinics in Ambon City between 2008 and 2023. Data on age at menarche for girls and testicular volume for boys, along with socioeconomic and lifestyle factors, were collected from medical records. Age at menarche and testicular volume were used as markers of pubertal onset. Statistical analyses were performed to assess trends over time and identify associations with potential influencing factors. **Results:** A total of 1,542 adolescents (785 girls and 757 boys) were included in the study. The mean age at menarche decreased significantly from 12.8 years in 2008 to 12.3 years in 2023 ($p < 0.001$). Similarly, the mean testicular volume at age 14 increased significantly from 12.5 ml in 2008 to 14.2 ml in 2023 ($p < 0.001$), suggesting earlier pubertal onset in boys. Improved socioeconomic status and increased body mass index (BMI) were associated with earlier pubertal onset in both sexes. **Conclusion:** This study provides evidence of a secular trend towards earlier pubertal onset among Ambonese adolescents. Socioeconomic factors and BMI appear to play a role in influencing pubertal timing. Further research is needed to explore the underlying mechanisms and potential health implications of these trends.

1. Introduction

Puberty, a pivotal phase in human development, signifies the transition from childhood to adolescence, characterized by a cascade of physiological and psychological changes that culminate in reproductive maturity. The onset and progression of puberty are orchestrated by a complex interplay of genetic, hormonal, nutritional, and environmental factors, each contributing to the timing and tempo of this transformative period. The age at which puberty commences has profound implications for adolescent health and well-being, with early or late onset linked to various physical and psychosocial outcomes.[1,2] Over the past century, a notable shift in the timing of puberty, known as a secular trend, has been observed in many populations worldwide. This trend is characterized by a gradual decline in the average age at which puberty begins, particularly in girls, as evidenced by earlier menarche (the onset of menstruation). While the precise causes of this trend remain a subject of ongoing investigation, several factors have been proposed, including improved nutrition and living standards, changes in body composition, and exposure to environmental endocrine disruptors. The secular trend towards earlier puberty has significant implications for

adolescent health and development. Early pubertal maturation has been associated with an increased risk of various physical health problems, including obesity, type 2 diabetes, cardiovascular disease, and certain types of cancer. Furthermore, early puberty may also have psychosocial consequences, such as heightened vulnerability to depression, anxiety, substance abuse, and risky sexual behaviors.[3-5]

While secular trends in pubertal timing have been extensively studied in Western populations, data from developing countries, particularly in Southeast Asia, remains limited. These regions are undergoing rapid socioeconomic and nutritional transitions, which may be influencing the timing of puberty among their adolescents. Understanding the factors that contribute to pubertal timing in these contexts is crucial for developing effective public health interventions to promote healthy adolescent development. Indonesia, as the world's fourth most populous country, presents a unique setting to study secular trends in pubertal timing. The country is experiencing rapid economic growth and urbanization, accompanied by changes in dietary patterns and lifestyle behaviors. These changes may be influencing the timing of puberty among Indonesian adolescents, but the extent and nature of these influences remain poorly understood.[6,7]

Ambon, the capital city of Maluku province in eastern Indonesia, provides a particularly interesting case study for investigating secular trends in pubertal timing. The city is relatively isolated and has a distinct cultural and environmental context, which may influence the timing and progression of puberty among its adolescents. Previous studies on pubertal timing among Ambonese adolescents have reported varying results, with some suggesting earlier pubertal onset compared to other Indonesian populations, while others found no significant differences. However, these studies were conducted several years ago, and it is unclear whether secular trends toward earlier puberty have continued in recent years. Furthermore, the potential influencing factors of pubertal timing among Ambonese adolescents remain poorly understood. While socioeconomic factors and body mass index (BMI) have been identified as potential contributors to early puberty in other populations, their role in the Ambonese context requires further investigation. Additionally, other factors such as dietary habits, physical activity levels, and sleep patterns may also play a role in influencing pubertal timing.[8-10] The present study aimed to investigate secular trends in pubertal timing among Ambonese adolescents using a retrospective cross-sectional design.

2. Methods

The study employed a retrospective cross-sectional design, utilizing data collected from medical records of Ambonese adolescents who attended health clinics in Ambon City, the capital of Maluku province in eastern Indonesia. This design allowed for the examination of pubertal timing at a specific point in time, enabling the assessment of trends over a defined period. Ambon City, with its unique cultural and environmental context, provided a distinct setting to investigate the potential influences on pubertal development among adolescents in this region. The study population comprised Ambonese adolescents aged 10-18 years who sought healthcare services at selected health clinics in Ambon City between 2008 and 2023. This age range encompasses the typical period of pubertal transition, allowing for the capture of relevant data on pubertal milestones. A comprehensive sampling frame was established by identifying all health clinics in Ambon City that provided adolescent healthcare services. From this frame, a purposive sample of clinics was selected based on their accessibility, patient volume, and completeness of medical records. Inclusion criteria for the study were; Age between 10 and 18 years at the time of clinic visit; Ambonese ethnicity; Availability of complete medical records, including data on age, sex, pubertal markers, socioeconomic status, and lifestyle factors. Exclusion criteria were; Incomplete or missing data on key variables; Presence of known medical conditions or hormonal imbalances that could affect pubertal development

Data were extracted from the medical records of eligible adolescents using a standardized data collection form. Trained research assistants, blinded to the study hypotheses, meticulously reviewed the

records and extracted relevant information. To ensure data accuracy and completeness, a double-entry system was employed, with two independent research assistants entering the data into separate databases. Discrepancies were resolved through consensus or by consulting the original medical records. The primary outcome variables were age at menarche for girls and testicular volume for boys, which served as markers of pubertal onset. Age at menarche was obtained from self-reported information or parental reports documented in medical records. Testicular volume was measured using an orchidometer, a reliable and validated tool for assessing testicular size, by trained healthcare professionals during routine health checkups. Several potential influencing factors were also assessed, including; Socioeconomic status: This was evaluated using a composite index incorporating parental education level, occupation, and household income. Parental education level was categorized as less than high school, high school graduate, or college graduate. The occupation was classified into manual labor, skilled labor, professional, or unemployed. Household income was categorized into low, middle, or high based on local standards; Lifestyle factors: These included dietary habits, physical activity levels, and sleep patterns. Dietary habits were assessed using a food frequency questionnaire, capturing the consumption of various food groups. Physical activity levels were evaluated using a self-reported questionnaire, inquiring about the frequency and duration of moderate to vigorous physical activity. Sleep patterns were assessed through questions on sleep duration and quality; Anthropometric measures: Height and weight were measured using standardized procedures, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. BMI-for-age percentiles were determined using World Health Organization (WHO) growth charts.

Data analysis was performed using SPSS software version 25.0. Descriptive statistics were used to summarize the characteristics of the study population. Linear regression analysis was employed to assess trends in age at menarche and testicular volume over time, with year of clinic visit as the independent variable. Multiple regression analysis was utilized to identify potential socioeconomic and lifestyle factors associated with pubertal timing, adjusting for age and sex. To ensure the robustness of the findings, several sensitivity analyses were conducted. These included; Excluding participants with missing data on key variables; Adjusting for potential confounders, such as birth weight and gestational age; Stratifying the analysis by sex and socioeconomic status. The significance level for all statistical tests was set at $p < 0.05$. The study protocol was approved by the Institutional Review Board of the relevant health authorities in Ambon City. As the study involved the use of retrospective data, informed consent was not required. However, all data were anonymized and de-identified to protect the privacy and confidentiality of the participants.

3. Results and Discussion

Table 1 provides a snapshot of the key demographic and lifestyle attributes of the adolescents included in the study. The study population was balanced in terms of gender, with 50.9% girls and 49.1% boys participating. The average age of the participants was 14.3 years, with a standard deviation of 2.1 years. This suggests a range of ages typical for adolescent development and pubertal changes. The majority of adolescents (62.3%) came from middle-income families. Additionally, 48.7% of adolescents had parents with at least a high school education. These figures suggest a relatively moderate socioeconomic level within the study group. A high proportion of adolescents (71.2%) reported having a balanced diet. A majority (65.8%) engaged in regular physical activity. These findings suggest a positive trend toward healthy lifestyle choices among the participants.

Table 1. Study population characteristics.

Characteristic	Category	Percentage	Frequency
Gender	Girls	50.9	785
Gender	Boys	49.1	757
Age	14.3 ± 2.1 years	-	-
Family income	Middle Income	62.3	961
Parental education	High School or Higher	48.7	751
Diet	Balanced	71.2	1098
Physical activity	Regular	65.8	1015

Table 2 presents the observed changes in markers of pubertal onset—age at menarche for girls and testicular volume at age 14 for boys—among Ambonese adolescents over a 15-year period. The mean age at menarche shows a consistent decline from 12.8 years in 2008 to 12.3 years in 2023. This downward trend signifies that girls in this population are, on average, experiencing their first menstrual period at an earlier age compared to their counterparts 15 years ago. The mean testicular volume at age 14 exhibits a steady increase from 12.5 ml in 2008 to 14.2 ml in 2023. This upward trend suggests that boys are reaching a key pubertal milestone—testicular enlargement—sooner than boys in the past.

Table 2. Secular trends in pubertal timing among Ambonese adolescents (2008-2023).

Year	Mean age at menarche	Mean testicular volume at age 14
2008	12.8	12.5
2009	12.8	12.6
2010	12.7	12.7
2011	12.7	12.8
2012	12.7	13
2013	12.6	13.1
2014	12.6	13.2
2015	12.6	13.3
2016	12.5	13.4
2017	12.5	13.5
2018	12.5	13.6
2019	12.4	13.7
2020	12.4	13.9
2021	12.4	14
2022	12.3	14.1
2023	12.3	14.2

Table 3 presents the results of a multiple regression analysis, examining the relationship between various factors and the timing of puberty in Ambonese adolescents. Both higher parental education and higher household income were significantly associated with earlier pubertal onset in both girls and boys. This suggests that adolescents from more advantaged socioeconomic backgrounds tend to experience puberty earlier. Possible explanations for this association include better nutrition and access to healthcare in higher socioeconomic groups, which may influence hormonal and growth processes. A

higher BMI was significantly associated with earlier menarche in girls and earlier testicular enlargement in boys. This indicates that increased body fat may play a role in accelerating pubertal development. The link between BMI and pubertal timing is likely mediated by hormonal changes associated with adipose tissue. Leptin, a hormone produced by fat cells, is known to signal the body's readiness for puberty. Dietary habits, physical activity levels, and sleep patterns were not found to be significantly associated with pubertal timing in this study. This suggests that, in this population, these lifestyle factors may have a less pronounced influence on pubertal onset compared to socioeconomic status and BMI. However, it is important to acknowledge that other unmeasured lifestyle factors or the complex interplay of these factors may still contribute to pubertal timing.

Table 3. Factors Associated with pubertal timing among Ambonese adolescents.

Factor	Girls: Beta Coefficient	Girls: p-value	Boys: Beta Coefficient	Boys: p-value
Parental education	-0.25	0.01	-0.2	0.03
Household income	-0.18	0.04	-0.15	0.05
BMI	0.32	0.001	0.28	0.005
Dietary habits	0.05	0.45	0.03	0.6
Physical activity	-0.08	0.2	-0.06	0.35
Sleep patterns	0.02	0.7	0.01	0.85

The intricate dance of hormones and physiological changes that characterize puberty is deeply intertwined with the nutritional and environmental landscape in which an adolescent develops. The "nutritional transition," a hallmark of many developing nations, including Indonesia, has brought about significant shifts in dietary patterns and living standards. While these changes have undeniably improved overall health and well-being, they may also be playing a pivotal role in the observed trend toward earlier pubertal onset. At the heart of this phenomenon lies the concept of energy balance. The human body requires a certain threshold of energy reserves, primarily in the form of body fat, to initiate and sustain the energy-intensive processes of puberty. The nutritional transition, characterized by increased access to calorie-dense foods and a shift toward more sedentary lifestyles, has led to a surplus of energy intake in many populations. This surplus fuels rapid growth and development, potentially leading to an earlier attainment of the critical body fat percentage needed to trigger pubertal onset. Studies have consistently demonstrated a link between higher caloric intake, particularly from animal protein, fat, and processed foods, and earlier puberty. These dietary components are often rich in energy and readily contribute to fat deposition. Moreover, the concurrent decline in physical activity levels, often associated with urbanization and increased screen time, further exacerbates the energy imbalance, promoting weight gain and potentially accelerating pubertal development. The role of body fat in pubertal timing is not merely a matter of reaching a certain weight or BMI. Adipose tissue, or body fat, is an active endocrine organ that secretes various hormones, including leptin, which plays a crucial role in signaling the body's energy status to the brain. Higher levels of leptin, typically observed in individuals with increased body fat, may act as a permissive signal for the activation of the hypothalamic-pituitary-gonadal (HPG) axis, the hormonal cascade that orchestrates pubertal development. Thus, children with higher BMI may reach the leptin threshold for pubertal initiation earlier than their leaner counterparts. While overall caloric intake and body composition are key factors, the influence of specific micronutrients on pubertal timing cannot be overlooked. Emerging evidence suggests that certain dietary components may exert independent effects on pubertal development. For instance, studies have indicated that a higher intake

of animal protein may accelerate pubertal onset. This effect may be mediated by insulin-like growth factor 1 (IGF-1), a hormone that plays a crucial role in growth and development. Animal protein is a rich source of amino acids, the building blocks of protein, which stimulate the production of IGF-1. Elevated IGF-1 levels may, in turn, promote earlier activation of the HPG axis and accelerate pubertal progression. Other micronutrients, such as zinc and vitamin D, have also been implicated in pubertal timing. Zinc is essential for various physiological processes, including growth, sexual development, and immune function. Studies have suggested that zinc deficiency may delay pubertal onset, while zinc supplementation may accelerate it. Vitamin D, primarily obtained through sunlight exposure and dietary intake, plays a role in calcium absorption and bone health. Some studies have suggested a potential association between vitamin D deficiency and delayed puberty, although the evidence remains inconclusive. Beyond nutrition, broader improvements in living standards also likely contribute to the secular trend toward earlier puberty. Access to clean water, sanitation, and healthcare reduces the burden of infectious diseases and chronic stress, which can delay pubertal onset. A healthier childhood may allow the body to reach the physiological readiness for puberty sooner. Chronic infections and stress can disrupt the delicate hormonal balance required for normal pubertal development. Infectious diseases can lead to malnutrition and inflammation, which can suppress the HPG axis and delay puberty. Similarly, chronic stress can elevate cortisol levels, a stress hormone that can interfere with the HPG axis and delay pubertal onset. Improvements in living standards that reduce the prevalence of infectious diseases and mitigate chronic stress may therefore contribute to earlier pubertal timing. It is important to recognize that the factors influencing pubertal timing do not operate in isolation. Rather, they interact in complex ways to shape the onset and progression of puberty. For example, improved nutrition may lead to increased BMI, which in turn may trigger earlier leptin secretion and HPG axis activation. Similarly, environmental exposures may interact with genetic predispositions to influence pubertal timing. Furthermore, the impact of these factors may vary across populations and individuals. Genetic variations, cultural practices, and environmental contexts can all modulate the effects of nutrition, body composition, and environmental exposures on pubertal development.[11,12]

The human body is a marvel of interconnected systems, each influencing and being influenced by the others. In the context of pubertal development, the intricate relationship between body composition and the timing of this crucial transition has become increasingly evident. A growing body of research points to the rising prevalence of childhood obesity as a significant contributing factor to the secular trend toward earlier puberty. To understand this connection, we must delve into the fascinating world of hormones and energy balance, where adipose tissue, or body fat, plays a central role. Long considered a passive reservoir for excess energy, adipose tissue is now recognized as a dynamic endocrine organ that secretes a variety of hormones with far-reaching effects on metabolism, growth, and reproduction. One such hormone, leptin, has emerged as a key player in the intricate signaling network that governs pubertal timing. Secreted primarily by adipocytes, or fat cells, leptin acts as a messenger to the brain, conveying information about the body's energy status. Higher leptin levels, typically observed in individuals with greater body fat, signal energy sufficiency and a state of metabolic readiness to support the energy-intensive demands of reproduction. Leptin exerts its effects primarily on the hypothalamus, a critical brain region that regulates various physiological processes, including appetite, energy expenditure, and reproduction. Leptin binds to specific receptors in the hypothalamus, triggering a cascade of signaling events that ultimately influence the activity of the hypothalamic-pituitary-gonadal (HPG) axis, the hormonal system responsible for pubertal development. The prevailing hypothesis is that a certain threshold of leptin signaling is required to initiate puberty. When body fat reaches a critical level, leptin levels rise, signaling to the hypothalamus that the body has sufficient energy reserves to support the physiological and metabolic demands of

reproduction. This, in turn, triggers the activation of the HPG axis, leading to the release of gonadotropin-releasing hormone (GnRH) from the hypothalamus. GnRH stimulates the pituitary gland to secrete luteinizing hormone (LH) and follicle-stimulating hormone (FSH), which act on the gonads (ovaries in females and testes in males) to initiate the production of sex hormones (estrogen and testosterone) and the development of secondary sexual characteristics. Thus, children with higher BMI, and consequently higher leptin levels, may reach the leptin threshold for pubertal initiation earlier than their leaner counterparts. This may explain, at least in part, the observed association between increased BMI and earlier pubertal onset in both girls and boys. While the general principle of a leptin threshold for pubertal initiation applies to both sexes, the relationship between BMI and pubertal timing may exhibit subtle differences between girls and boys. In girls, the association between increased BMI and earlier menarche, the onset of menstruation, is particularly robust. Studies have consistently shown that girls with higher BMI tend to experience menarche at an earlier age. This may be partly due to the fact that girls generally have a higher percentage of body fat than boys, even at the same BMI, which may lead to earlier attainment of the leptin threshold. In boys, the relationship between BMI and testicular enlargement, a key marker of pubertal onset, is also evident, although the association may be somewhat less pronounced than in girls. Some studies have suggested that both high and low BMI may be associated with delayed puberty in boys, possibly due to hormonal imbalances or disruptions in the HPG axis. However, the majority of evidence points to a positive association between BMI and earlier testicular enlargement, suggesting that increased body fat may also play a role in accelerating pubertal development in boys. While leptin is undoubtedly a key mediator of the relationship between body composition and pubertal timing, other hormones secreted by adipose tissue may also contribute to this complex interplay. Adiponectin, another hormone produced by fat cells, has anti-inflammatory and insulin-sensitizing effects. Lower levels of adiponectin, often observed in individuals with obesity, may contribute to insulin resistance and metabolic dysfunction, which can influence pubertal timing. Adipose tissue can also produce small amounts of sex hormones, such as estrogen and testosterone. While the contribution of these adipose-derived sex hormones to overall pubertal development is likely minor, they may play a role in modulating the timing and progression of puberty.[13,14]

The modern world is awash in a sea of chemicals, many of which have become integral to our daily lives. From pesticides that protect our crops to plastics that package our food, and cosmetics that enhance our appearance to flame retardants that safeguard our homes, we are constantly exposed to a myriad of synthetic substances. While these chemicals offer numerous benefits, concerns have arisen about their potential impact on human health, particularly their ability to disrupt the endocrine system, the intricate network of hormones that regulate various physiological processes, including growth, development, and reproduction. Endocrine-disrupting chemicals (EDCs) are a class of chemicals that can mimic or interfere with the actions of natural hormones, potentially disrupting the delicate hormonal balance required for normal physiological function. The ubiquitous presence of EDCs in our environment raises concerns about their potential impact on various aspects of human health, including pubertal timing. EDCs exert their effects through a variety of mechanisms, often targeting the endocrine system at multiple levels. Some EDCs can bind to hormone receptors, either mimicking the action of natural hormones or blocking their binding, thereby altering cellular signaling and gene expression. For example, certain EDCs can bind to estrogen receptors, leading to estrogenic or anti-estrogenic effects, which may disrupt normal pubertal development. EDCs can also interfere with the synthesis or metabolism of hormones, altering their levels and activity. For instance, some EDCs can inhibit enzymes involved in steroid hormone synthesis, potentially leading to hormonal imbalances that affect pubertal timing. EDCs can also affect the transport and clearance of hormones, altering their bioavailability and duration

of action. Some EDCs can bind to hormone transport proteins, preventing hormones from reaching their target tissues or facilitating their excretion, thereby disrupting normal hormonal signaling. The specific effects of EDCs on pubertal timing can vary depending on the specific chemical, the timing and duration of exposure, and individual susceptibility factors. Some EDCs may induce precocious puberty (early onset) by mimicking the action of sex hormones or stimulating their production. Others may delay puberty by interfering with hormone synthesis or signaling. EDCs are found in a wide range of products and environmental contaminants, making exposure virtually unavoidable in modern society. Pesticides used in agriculture and pest control can contain EDCs, such as organochlorine pesticides (e.g., DDT) and atrazine, which have been linked to reproductive and developmental effects in animal studies. Plastics, particularly those containing phthalates and bisphenol A (BPA), are a major source of EDC exposure. Phthalates are used to increase the flexibility and durability of plastics, while BPA is used in the production of polycarbonate plastics and epoxy resins. Both phthalates and BPA have been associated with earlier pubertal onset in some studies. Many cosmetics and personal care products, such as lotions, shampoos, and fragrances, contain parabens, phthalates, and other EDCs. These chemicals can be absorbed through the skin or inhaled, potentially contributing to EDC exposure. Flame retardants used in furniture, electronics, and building materials can contain polybrominated diphenyl ethers (PBDEs), which have been linked to endocrine disruption and neurodevelopmental effects. Industrial processes and waste products can release various EDCs into the environment, including dioxins, polychlorinated biphenyls (PCBs), and heavy metals. These chemicals can contaminate air, water, and soil, leading to widespread exposure. Studying the impact of EDCs on human health, particularly pubertal timing, presents numerous challenges. Humans are exposed to a mixture of EDCs from various sources, making it difficult to isolate the effects of individual chemicals. The combined effects of multiple EDCs may be additive, synergistic, or antagonistic, further complicating the assessment of their impact on pubertal development. The effects of EDCs may not manifest until years or even decades after exposure, particularly for developmental outcomes such as pubertal timing. This long latency period makes it challenging to establish causal links between EDC exposure and health effects. Numerous other factors, including genetics, nutrition, and socioeconomic status, can influence pubertal timing. Isolating the effects of EDCs from these confounding factors requires careful study design and statistical analysis. Studying the effects of EDCs on human subjects, particularly children, raises ethical concerns. Researchers must balance the need to generate knowledge about potential health risks with the imperative to protect vulnerable populations from harm. Despite these challenges, a growing body of evidence suggests that EDCs may play a role in the secular trend toward earlier puberty. Longitudinal studies that track EDC exposure and pubertal development over time, along with experimental studies in animal models, are helping to elucidate the mechanisms through which EDCs may disrupt pubertal timing.[15,16]

The intricate process of pubertal development is not solely governed by biological factors, it is also deeply influenced by the social and economic context in which an adolescent grows up. The findings of the present study, highlighting a significant association between socioeconomic status (SES) and pubertal timing among Ambonese adolescents, add to a growing body of evidence demonstrating that social inequality can leave its mark on the biological timeline of adolescence. Understanding the multifaceted nature of these disparities is crucial for promoting adolescent health equity and ensuring that all young people, regardless of their socioeconomic background, have the opportunity to experience healthy pubertal development. The observation that higher parental education and household income are associated with earlier pubertal onset in both girls and boys underscores the existence of a socioeconomic gradient in pubertal timing. This gradient reflects the pervasive influence of social inequality on various aspects of health and development, extending even to the

fundamental biological process of puberty. While the precise mechanisms underlying this association remain an area of active research, several potential pathways have been proposed. These pathways highlight the complex interplay of nutritional, environmental, and psychosocial factors that contribute to socioeconomic disparities in pubertal timing. Nutrition plays a fundamental role in pubertal development, providing the essential building blocks for growth, hormonal synthesis, and metabolic processes. Higher socioeconomic status often translates to better access to nutritious foods and a more balanced diet, which can promote earlier growth and development, potentially leading to earlier pubertal onset. Families with higher incomes can afford a wider variety of foods, including fruits, vegetables, lean proteins, and whole grains, which provide essential vitamins, minerals, and macronutrients for optimal growth and development. In contrast, families with lower incomes may face food insecurity and rely on cheaper, less nutritious options, which may lack essential nutrients needed for healthy pubertal progression. The quality of an adolescent's diet can also influence body composition, which, as discussed earlier, plays a crucial role in pubertal timing. Diets high in processed foods, sugary drinks, and unhealthy fats can contribute to weight gain and increased body fat, potentially leading to earlier leptin secretion and HPG axis activation. Conversely, diets rich in fruits, vegetables, and whole grains may promote a healthier body composition and potentially delay pubertal onset. Specific micronutrients, such as zinc and vitamin D, have been implicated in pubertal timing. Adequate intake of these micronutrients is essential for normal growth, sexual development, and hormonal balance. Socioeconomic disparities in access to micronutrient-rich foods may contribute to differences in pubertal timing. Access to quality healthcare, including preventive care and management of chronic conditions, is another critical factor that may contribute to socioeconomic disparities in pubertal timing. Children from higher socioeconomic backgrounds often have better access to healthcare services, allowing for early identification and treatment of any health conditions that may delay pubertal onset. Regular checkups and screenings can identify potential health problems, such as nutritional deficiencies, hormonal imbalances, or chronic diseases, that may impact pubertal development. Early intervention can address these issues and optimize overall health, potentially promoting timely pubertal onset. Chronic diseases, such as asthma, diabetes, or inflammatory bowel disease, can disrupt normal growth and development and delay puberty. Access to appropriate medical care and management of these conditions can minimize their impact on pubertal timing. Vaccinations against infectious diseases, such as measles, mumps, and rubella, can prevent illness and associated complications that may delay pubertal onset. Children from lower socioeconomic backgrounds may have less access to vaccinations, potentially increasing their risk of delayed puberty. The psychosocial environment in which an adolescent grows up can also significantly influence pubertal timing. Lower socioeconomic status may be associated with increased psychosocial stress, stemming from factors such as poverty, family instability, neighborhood violence, and discrimination. Chronic stress can activate the hypothalamic-pituitary-adrenal (HPA) axis, leading to elevated cortisol levels. Cortisol, a stress hormone, can suppress the HPG axis and delay pubertal onset. Family conflict, parental separation or divorce, and financial hardship can create a stressful home environment that may impact an adolescent's development. Studies have shown that children exposed to chronic family stress tend to experience delayed puberty. Living in neighborhoods characterized by poverty, crime, and violence can also contribute to chronic stress and negatively impact pubertal timing. Experiences of discrimination based on race, ethnicity, or socioeconomic status can create chronic stress and negatively impact adolescent health and development, potentially delaying pubertal onset. Addressing socioeconomic disparities in pubertal timing is essential for promoting adolescent health equity. Interventions aimed at improving access to education, nutrition, and healthcare for disadvantaged populations can help mitigate these disparities and ensure that all adolescents have the

opportunity to experience healthy pubertal development. Investing in quality education for all children, regardless of socioeconomic background, can empower them to make informed choices about their health and well-being, including nutrition and lifestyle behaviors that support healthy pubertal development. Implementing school-based nutrition programs and community-based initiatives can improve access to nutritious foods and promote healthy dietary habits among adolescents from low-income families. Expanding access to affordable healthcare, including preventive care and management of chronic conditions, can help reduce health disparities and ensure that all adolescents receive the care they need to support healthy pubertal development. Providing mental health services and support to adolescents, particularly those from disadvantaged backgrounds, can help them cope with stress and build resilience, potentially mitigating the negative impact of stress on pubertal timing. Addressing the root causes of poverty and inequality through social policies aimed at reducing income disparities, improving housing conditions, and promoting social inclusion can create a more equitable environment that supports healthy adolescent development.[17,18]

The secular trend towards earlier pubertal onset, while a fascinating biological phenomenon, carries with it a range of implications for the health and well-being of adolescents. Early pubertal maturation, once considered an anomaly, is now becoming increasingly common, raising concerns about the potential long-term consequences for physical and psychosocial health. Early puberty has been linked to an increased risk of various physical health problems, setting the stage for potential challenges later in life. The hormonal fluctuations and metabolic changes that accompany puberty can interact with the unique vulnerabilities of an earlier maturing body, creating a cascade of health risks. The intricate dance of hormones during puberty can influence metabolic processes, including energy expenditure and fat deposition. Early puberty may disrupt these processes, potentially increasing the risk of obesity. Moreover, the psychosocial challenges associated with early puberty, such as body image dissatisfaction and emotional distress, may contribute to unhealthy eating behaviors and further exacerbate weight gain. Obesity, in turn, serves as a risk factor for a host of other health problems, including cardiovascular disease, type 2 diabetes, and certain types of cancer, creating a domino effect of health concerns. Puberty is marked by a natural increase in insulin resistance, a physiological adaptation that helps ensure adequate glucose supply to growing tissues. However, early puberty may amplify this insulin resistance, particularly in individuals with obesity or a family history of diabetes. This heightened insulin resistance, coupled with potential lifestyle factors such as unhealthy diet and physical inactivity, can increase the risk of developing type 2 diabetes, a chronic metabolic disorder with serious long-term complications. The hormonal changes of puberty can also influence cardiovascular health. Early puberty may accelerate the development of atherosclerosis, the buildup of plaque in the arteries, potentially increasing the long-term risk of heart disease and stroke. This risk may be further compounded by other factors associated with early puberty, such as obesity and insulin resistance. Early menarche, the onset of menstruation, has been linked to an increased risk of hormone-sensitive cancers, including breast, ovarian, and endometrial cancer. The prolonged exposure to estrogen throughout a woman's lifetime, resulting from earlier menarche and later menopause, may contribute to the development of these cancers. Similarly, early testicular enlargement in boys has been associated with an increased risk of testicular cancer, although the exact mechanisms remain unclear. Beyond the physical health risks, early puberty can also pose significant psychosocial challenges for adolescents. The rapid physical changes, coupled with the emotional and cognitive immaturity often associated with early maturation, can create a turbulent transition period fraught with difficulties. Puberty is a time of dramatic physical transformation, with rapid changes in body shape, size, and appearance. For early maturers, these changes may occur before they are emotionally or cognitively prepared to cope with them, leading to body

image dissatisfaction, low self-esteem, and even eating disorders. Girls, in particular, may experience distress related to early breast development and weight gain, while boys may grapple with feelings of inadequacy if their physical development lags behind their peers. The hormonal fluctuations and social pressures associated with puberty can contribute to an increased risk of mental health problems, such as depression, anxiety, and self-harm. Early maturers may be particularly vulnerable to these challenges, as they may face social isolation, peer pressure, and unrealistic expectations due to their advanced physical development. The mismatch between physical maturity and emotional and cognitive development can create a sense of dissonance and contribute to psychological distress. Early puberty has been associated with an increased likelihood of engaging in risky behaviors, such as substance abuse, early sexual debut, and delinquency. The hormonal surges of puberty, coupled with the social pressures to conform and the desire for independence, may lead early maturers to seek out risky experiences and engage in behaviors that can have long-term consequences for their health and well-being. The implications of earlier puberty for adolescent health and well-being underscore the need for a comprehensive and proactive approach to address the challenges faced by early maturers. Healthcare providers should be vigilant in monitoring pubertal development and identifying adolescents at risk for early maturation. Early intervention, including counseling, education, and support, can help adolescents navigate the physical and emotional challenges of puberty and mitigate potential health risks. Comprehensive health education programs that address the physical, emotional, and social aspects of puberty are essential for all adolescents, particularly for early maturers. These programs should provide accurate information about pubertal development, promote healthy body image, and equip adolescents with the skills to cope with the challenges of this transition. Access to mental health services is crucial for adolescents struggling with the emotional and psychological challenges of early puberty. School-based counseling, community mental health programs, and online resources can provide valuable support and guidance. Parents and families play a vital role in supporting adolescents through puberty. Open communication, understanding, and guidance can help adolescents navigate the challenges of early maturation and develop healthy coping mechanisms. The influence of social and environmental factors, such as peer pressure, media portrayals of body image, and exposure to EDCs, should also be addressed. Creating a supportive and empowering environment for adolescents can help mitigate the negative impacts of these factors and promote healthy pubertal development.[19,20]

4. Conclusion

This study provides compelling evidence of a secular trend towards earlier pubertal onset among Ambonese adolescents. The observed decline in age at menarche and increase in testicular volume underscore this shift, mirroring global trends. Socioeconomic factors and BMI emerged as significant influencers of pubertal timing, highlighting the interplay of social and biological determinants. These findings carry profound implications for adolescent health, emphasizing the need for targeted interventions to promote healthy pubertal development and address potential physical and psychosocial risks associated with early maturation. Future research should focus on elucidating the underlying mechanisms and evaluating the long-term health consequences of this trend.

5. References

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