SYSTEMATIC REVIEW

BMC Nutrition



Anthropometric status, body composition and timing of pubertal milestones in Sub-Saharan Africa: a systematic review



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Abstract

Background With the rise of the triple burden of malnutrition, the changing nutrition situation in Sub-Saharan Africa may be associated with changes in pubertal timing of adolescents. The purpose of this review was to summarize the association between nutritional status and pubertal milestones among children in SSA.

Method A search of publications was conducted in PubMed and Scopus on 1st April 2023. Observational studies with children aged 0 to 22 years, that reported nutritional status and association with pubertal milestones in SSA were selected for review. Risk of bias was assessed using the NOS and results were presented using the PRISMA.

Results Twenty-three studies published from 1992 to 2021 reporting data from nine countries and a total of 21,853 children were included in this review. Mean menarche age relative to nutritional status varied from 17.2 years in stunted adolescents in Senegal to 13.3 (in the underweight), 13.9 (in normal weight girls), and 14.1 (in overweight girls) years in adolescents in Ethiopia. Adolescents who were not stunted in Kenya and those with higher height-for-age z-scores (HAZ) and body mass index (BMI) in South Africa had more advanced breast development. Pubic hair development was positively associated with HAZ and BMI z-scores (BMIZ) at 5 years in South Africa and overweight and obesity in adolescents in Nigeria. Attainment of voice break in adolescent boys in Nigeria was associated with lower likelihood of stunting and underweight. In a study in Zambia, earlier onset and more rapid progression of genital development assessed by testicular volume in boys was associated with increased height and arm muscle.

Conclusions Higher BMI, height, weight, and triceps skinfolds are significantly associated with advanced pubertal development in SSA. In SSA, less than one-third of the countries have published any research studies on nutritional status and pubertal milestones. Future studies should focus on detailed assessment of pubertal development and associated nutritional factors in both male and female adolescents in SSA.

PROSPERO registration CRD42022350048.

Keywords Nutritional status, Pubertal milestones, Sub-Saharan-Africa, Adolescents

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Background

Puberty is a period of rapid linear growth with many physical changes that prepare the human body for reproduction [1, 2]. It is a result of complex biological processes that are mediated by genetic, hormonal, and environmental factors and that are characterized by the maturation of gametogenesis and secretion of gonadal hormones [2, 3]. Past reviews have shown variability in pubertal timing which has been linked to socioeconomic conditions, psychological factors, environmental toxicants, physical activity patterns and nutritional exposures [1, 4].

Sub-Saharan Africa (SSA) is faced with a triple (undernutrition, overnutrition, anaemia) burden of malnutrition which is of public health concern [5]. However, few studies have considered the role of nutritional status in pubertal timing in SSA. Considering that childhood nutritional status affects nutritional status in adulthood, and nutritional factors affect pubertal onset with implications on metabolic functions, investigating the relationship between nutritional status and pubertal milestones in African populations is necessary to identify normal and unusual patterns, to make suitable nutritional recommendations.

In a recent review of literature that included low- and middle-income countries (LMICs), the African region recorded relatively later age at menarche (13.82 years [y]), compared to other World Bank Regions that were represented; Eastern Mediterranean Region (12.21 y), South-East Asia Region (12.34 y) and Western Pacific Region (11.83 y). In the past years, countries in most part of the world have experienced a decline in menarcheal (onset of menses) age [4].

Increased adiposity and higher BMI have been linked to precocious puberty in girls which is defined as the onset of puberty before 8 years for females [6]. Several epidemiological and cross-sectional studies have shown that obese girls have earlier pubertal onset, which is characterized by the appearance of secondary sex characteristics, in particular the appearance of breast in females, testicular enlargement in males, and pubic/axillary hair in both sexes [7]. There are mixed findings of associations of obesity and pubertal onset in boys [8, 9]. These studies have also shown that menarche is advancing more quickly than thelarche (development of breast buds under areola) especially in girls with higher BMI [1]. On the other hand, undernutrition is associated with delayed puberty onset, particularly, reduced pubertal growth spurt and later age at menarche [2, 10, 11]. Nevertheless, it must be noted that a level of body fat mass needs to be achieved to attain reproductive capacity [12]. Nutrition has been suggested to explain about 25% of the variation in pubertal timing [2].

While nutrition may influence the timing of pubertal milestones [13], the evidence of this in SSA remains unclear. Even more fundamental, there is critical need to assess the extent of research conducted in this area within the region, and to identify future research directions. Therefore, the purpose of this systematic review was to summarize the evidence on the association between nutritional status and the achievement of pubertal milestones, as assessed by Tanner Staging [2] or the self-report Petersen Pubertal Development Scale [14] in SSA.

Methods

Data sources

An article search was conducted in PubMed and Scopus on 1st April 2023. Searches in other databases, including Embase, Web of Science, and Google Scholar, did not yield any additional papers. Search terms used were text words and medical subject headings (MeSH). Identified key words were used to finalize search terms (Supplementary File 1). The study protocol is registered as PROSPERO CRD42022350048.

Inclusion and exclusion criteria

Eligible studies included observational (cohort and cross-sectional) studies and involved children from SSA, not known to have any disease condition, and aged 0 to 22 years to cover the full age range of pubertal development. Studies were excluded if they did not report any association between nutritional status and pubertal milestones. Additionally, studies must have reported anthropometric measurements or body composition indices of children, before or at puberty, for assessing nutritional status. Pubertal milestones must have been assessed using the Tanner Stages [2] (adrenarche, gonadarche, pubarche, thelarche, spermarche, peak height velocity) or pubertal development scale as indicators for sexual maturity [14]. All publications included were human studies and without date restriction, up to the search date listed above.

Data extraction

We exported data into Endnote and Rayyan, from Pub-Med and Scopus. Relevant studies were extracted and presented using the PRISMA checklist (Fig. 1). Evidence from selected studies were summarized and synthesized by relationship between nutritional status and pubertal milestones. We selected publications that assessed pubertal milestones by clinical assessment/Tanner Staging [2] detailed in previous publication or self-report of pubertal development [14]. Data extracted included author, year of publication, study design, sample size, study population characteristics (prepubertal age of assessment, sex,

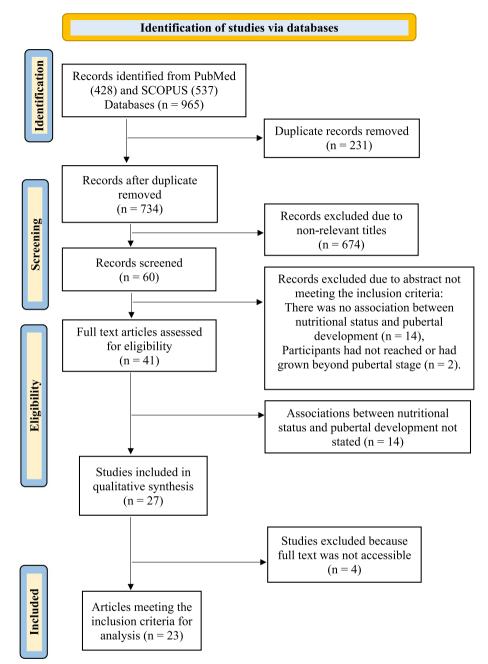


Fig. 1 PRISMA study selection diagram. This flow diagram illustrates the flow of the number of articles identified, excluded and included through different phases of the systematic review

country), nutritional status indices (BMIZ, HAZ, hip circumference (HC), waist circumference (WC), weight, height, triceps skinfold, upper arm muscle area, body composition), pubertal timings and pubertal milestones (thelarche, menarche, testicular volume, pubic hair development, genital development). Extracted data were summarized and synthesized.

Assessment of study quality

The study quality (risk of bias) was evaluated using the Newcastle Ottawa Scale (NOS) for cohort studies and an adapted form of the NOS cohort scale for cross-sectional studies [15, 16]. Two reviewers independently evaluated each study. Discrepancies in ratings were discussed and resolved through consensus, with the aim of minimizing subjective bias and enhancing the overall reliability of the study assessments.

Results

Search results

Two researchers identified 965 studies of which 231 were duplicates. Sixty records were screened after articles with non-relevant titles were removed.Twenty-three (23) studies conducted in nine (9) countries, which involved 21,853 children were included in this review. Selected studies included those that investigated associations between indicators for nutritional status and pubertal milestones. Summary of data extracted and synthesized have been shown in Table 1.

Pubertal milestones in girls

Breast development in girls

Eight (8) studies reported on nutritional status and breast development (Table 1); two (2) in South Africa [30, 31], one in Kenya [13], one in Zambia [21], one in Senegal [19] and three in Nigeria [3, 34]. In Nigeria, normal weight, overweight and obesity were significantly associated with more advanced breast development. That is, higher BMI was associated with earlier onset, more rapid progression, and earlier completion of breast maturation. Among South African girls, HAZ and BMIZ at 5 years were positively associated with earlier onset, more rapid progression, and earlier completion of breast development.

Pubic hair development in girls

Three (3) publications reported on pubic hair development (Table 1). In Nigeria, overweight, obesity and upper socioeconomic class were significantly and positively associated with more advanced pubic hair development [3, 33]. In South Africa, HAZ and BMIZ at 5 years were positively associated with more advanced pubic hair development [30].

Menarche

Seventeen (17) publications spanning from 1992 to 2021 investigated the relationship between nutritional status and menarche (Table 1): nine in Nigeria [3, 4, 17, 22, 23, 26, 32, 33, 36], two in Senegal [18, 19], one each from Uganda [27], Ghana [24] Kenya [13], Tanzania [28] South Africa [29] and Ethiopia [25]. In four studies, the mean age at menarche ranged from 17.2 years in stunted (<-2 HAZ) Senegalese [18] to 13.3 years in urban Ugandan [27] children whose mean height of 162.8 cm and HC of 93.5 cm suggested they were not stunted.

Generally, higher preschool and adolescent height, weight and BMI were significantly associated with earlier attainment of menarche [4], except in Senegal, where inconsistent observation was reported among adolescents [18, 19] (Table 1). Likewise, in Ghana, low birth weight babies attained menarche earlier than normal and high birth weight babies [24]. In Uganda, an inverse relationship was observed between age at menarche and HC but not BMI, height and WC. Urban dwelling girls had earlier menarche than rural dwelling girls [27]. In Nigeria, girls with high energy and protein intake attained menarche earlier than girls with low energy and protein intake [4]. Girls from upper socioeconomic families, private schools and Hausa tribe had early menarche [3].

Pubertal milestones in boys Growth spurt in boys

There was a significant increase in height, weight and BMI between Tanner Stages 2 and 3, between ages 11.6 and 13.1 years among South African boys [31].

Pubic hair development in boys

HAZ and BMIZ at 5 years were positively associated with more advanced pubic hair development in South African boys [22].

Genital development in boys

Four (4) studies reported on testicular growth [20, 30, 31, 35]. In South Africa, HAZ at 5 years was positively associated with more advanced genital development [30]. In Zambia [20] earlier testicular growth onset was positively associated with height and arm muscle area (Table 1). Urban boys had earlier testicular growth onset than rural boys [20]. Urban boys were taller, heavier, and completed testicular growth earlier than rural boys.

Voice break

One study conducted in Nigeria reported on voice break [22]. Earlier achievement of voice break was negatively associated with stunting and underweight.

Study quality

Out of cohort (Table 2) studies identified, 3 were judged to be at low risk of bias (LRB) and 3 at high risk of bias (HRB) according to the NOS score rating [37]. None was judged to be at very high risk of bias. Out of seventeen (17) cross-sectional studies (Table 3), 12%, 59%, 23%, and 6% (1 study) were very good (VG), good (G), satisfactory (SAT), and unsatisfactory (UNSAT).

Discussion

In our review of twenty-three studies comprising 18,543 girls and 3,310 boys, we found significant associations of anthropometric status and body composition, with pubertal development in most of the studies.

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| Authors/ Publication Year/ Country | Study Design | Participants | Nutritional Status Outcomes | Pubertal Development Assessment | Comments | Main Findings |
|---|--------------------------|--|---|---|--|---|
| Dare et al 1992 [17] Nigeria | Cross-sectional | Secondary school girls 10 – 22 years (n=1616) | BMI | Menarche | • Pre-menarche girls (<i>n</i> = 657) • Post-menarche girls (<i>n</i> = 959) | Pre-menarche girls (16.47 \pm 0.64 kg/m ²) had lower BMI (p <0.001) than post-menarche girls (19.45 \pm 0.28 kg/m ²) |
| Abioye et al 1997 [4] Nigeria | Cross-sectional | High school girls 9 –20 years (n=352) | Weight Height BMI | Achievement of menarche | Achieved menarche (n = 187) Not yet achieved menarche (n = 165) | Girls who achieved menarche were taller, heavier and had higher BMI (<i>p</i> <0.05) |
| Simondon et al 1998 [18] Senegal | Longitudinal | Rural girls 12 – 17 years with known HAZ at pre- school age 2 – 5 years (n= 1650) | HAZ measured at pre- school age | Age at menarche | • Had HAZ <-2 at preschool age ($n = 544$) • Had HAZ -2 to -1 at pre- school age ($n = 541$) • Had HAZ >-1 at preschool age ($n = 565$) | Estimated mean age at menarche: 17.2 years for <-2 HAZ, 16.5 years for -2 to -1 HAZ, and 15.6 years for >-1 HAZ (<i>p</i> <0.001) |
| Benefice et al 2001 [19] Senegal | Longitudi- nal cohort | Rural girls 0 —15 years (n= 406) | HAZ measured at infancy | Attainment of thelarche Attainment of menarche | Stunted at infancy (n=81) Non-stunted at infancy (n=286) | No differences in breast development and achieve- ment of menarche between rural girls who were stunted or not stunted at infancy |
| Campbell et al 2004 [20] Zambia | Cross-sectional | Rural and urban Tonga boys Height 5 – 19 years (<i>n</i> = 794) Upper a | Height Upper arm muscle area | Testicular volume | | Earlier onset and completion of testicular growth was posi- tively associated with height ($p < 0.001$) and arm muscle area ($p < 0.05$) |
| Gilette-Netting et al. 2004 [21] Zambia | Longitudinal | Rural and urban girls 6 –18 years (n=774) | Height Upper muscle area Triceps skinfold | •Tanner breast develop- ment | | • Greater height, upper mus- cle area and triceps skinfolds were significant predictors of pubertal onset and com- pletion ($p < 0.05$) |

| Table 1 (continued) | | | | | | |
|--|-----------------|--|--------------------------------|---|---|---|
| Authors/ Publication Year/ Country | Study Design | Participants | Nutritional Status Outcomes | Pubertal Development Assessment | Comments | Main Findings |
| Leenstra et al 2005 [13] Kenya | Cross-sectional | Adolescent girls in two poor rural settings 12 – 18 years (<i>n</i> = 928) | HAZ BMIZ | Achievement of menarche Tanner breast develop- ment | Thin pre-menarche girls $(n = 126)$ • Stunted pre-menarche girls $(n = 126)$ • Thin post-menarche girls $(n = 105)$ • Thin post-menarche girls $(n = 12)$ • Stunted post-menarche girls $(n = 5)$ • Thin girls at Tanner Stage 1 $(n = 53)$ • Stunted girls at Tanner Stage 2 $(n = 45)$ • Thin girls at Tanner Stage 2 $(n = 45)$ • Stage 2 $(n = 41)$ | Post-menarche girls were 85% (95% CI: 0.07 – 0.30) less likely to be thin, and 90% (95% CI: 0.03 – 0.25) less likely to be stunted compared with pre-menarche girls Girls at Tanner Stage 2 for breast development (B2) were at 60% (95% CI: 0.22 – 0.75) reduced risk of stunting than girls at Tanner Stage 1 (B1) |
| Omigbodun et al 2010 [22] Nigeria | Cross-sectional | Adolescent school boys (<i>n</i> =924) and girls (<i>n</i> =875) 10 - 19 years | HAZ BMI | Achievement of menarche Voice break | Adolescents who had achieved menarche or voice break (n = 1004) Adolescents who had not achieved menarche or voice break (n = 795) | • Adolescents were ~ thrice odds of stunting ($\rho < 0.01$), and twice odds of underweight ($\rho < 0.01$) if they had not had menarche or voice break |
| Goon et al 2010 [23] Nigeria | Cross-sectional | Rural girls 12–18 years $(n=722)$ | BMI | Attainment of menarche | • Girls who had attained menarche ($n = 674$) Girls who had not attained menarche ($n = 48$) | Post-menarcheal girls had higher BMI than pre-menar- cheal girls (p < 0.01) |
| Aryeetey et al 2011 [24] Ghana | Cross-sectional | School girls 9 – 18 years (n= 529) | BMI Birth weight | Achievement of menarche | Girls: • Underweight ($n = 228$) • Normal weight ($n = 243$) • Overweight ($n = 58$) Girls with birth weight: • $2.5 + 4$ kg ($n = 63$) • $2.5 - 4$ kg ($n = 138$) • $2.5 + 4$ kg ($n = 44$) | • Overweight (11.21 ± 1.1.5 kg/m ²) adolescents attained menarche earlier compared to normal (12.72±0.74 kg/ m ²) and underweight (13.43 ± 1.10 kg/m ³) girls ($p < 0.01$) • Girls who had low birth weight experienced earlier menarche compared with girls who had normal and high birth weight ($p < 0.01$) |

| Table 1 (continued) | | |
|-------------------------------|--------------|---------|
| Authors/ Publication Year/ | Study Design | Partici |

| Authors/ Publication Year/ Country | Study Design | Participants | Nutritional Status Outcomes | Pubertal Development Assessment | Comments | Main Findings |
|---|--------------------|---|---|--|--|--|
| Ayele & Berhan 2013 [25] Ethiopia | Cross-sectional | Adolescent girls 10 – 19 years in public schools (n=660) | Ĩ | Achievement of menarche | Underweight adolescents (n = 54) Normal weight adoles- cents (n = 561) Overweight adolescents (n = 45) | Mean ages (years) at menarche: 13.3 for under- weight, 13.9 for normal weight and 14.1 for over- weight A unit increase in BMI was associated with 1.4 times odds of earlier menarche (p < 0.01) |
| Onyiriuka & Egbagbe 2013 [26] Nigeria | Cross-sectional | Adolescent schoolgirls 0—15 years ($n = 2150$) | BMI | Attainment of menarche | • Girls who had attained menarche (<i>n</i> = 1640) Girls who had not attained menarche (<i>n</i> =510) | • Schoolgirls who attained menarche had higher BMI compared to pre-menar- cheal schoolgirls (18.8 \pm 1.6 versus 16.4 \pm 1.9, P < 0.001;) |
| Mpora et al 2014 [27] Uganda | Cross-sectional | Urban and rural secondary school girls 12 – 18 years (n= 274) | BMI Height WC HC | Age at menarche | | Hip circumference was significantly and negatively correlated with age at menarche (<i>t</i>=-0.109; <i>p</i>=0.036) No significant association was found between age at menarche and BMI, height, or waist circumference |
| Rebacz-Maron 2015 [28] Tanzania | Cross-sectional | Female students 12 – 22 years (n = 97) | Height Weight % body water Body fat % body muscle | Attainment of menarche | | -Z statistic for Mann–Whit- ney U test between pre- and post- menarche girls amounted to -2822 ($p = 0.005$), -3.164 ($p = 0.002$), 3.221 ($p = 0.001$) and $2.989(p = 0.003) respectively for:body height, body weight, %water and % muscle$ |
| Salgin et al 2015 [29] South Africa | Prospective cohort | Girls 9 – 10 years (n = 1 201) Weight gain | Weight gain | • Menarche | | • Catchup weight gain during infancy was asso- ciated with younger age at menarche in girls ($p < 0.001$) |

| Table 1 (continued) | | | | | | |
|--|--------------------|---|--------------------------------|---|--|--|
| Authors/ Publication Year/ Country | Study Design | Participants | Nutritional Status Outcomes | Pubertal Development Assessment | Comments | Main Findings |
| Lundeen et al 2016 [30] South Affica | Prospective cohort | Boys (<i>n</i> = 1060) and girls (<i>n</i> = 1135) 0 - 20 years | Height BMI | Genital development (SMS) Breast development Pubic hair development | | Girls: HAZ and BMIZ at 5 years were positively associated with public hair and breast development (p < 0.05) Boys: HAZ and BMIZ associated with public hair development (p < 0.01). HAZ at 5 years was associated with gental development |
| Mao & Dalvie 2016 [31] South Affrica | Cross-sectional | Rural school boys 5 – 19 years (n=269) | Height Weight BMI | Tanner Stages (sexual development) | | • Significant increase in height (p < 0.001), weight (p < 0.001) and BMI (p < 0.001) between Tanner stages 2 and 3; between ages 11.6 to 13.1 years |
| Nwokocha et al 2016 [32] Nigeria | Cross-sectional | Female teenagers 9–18 years (<i>n</i> = 897) | BMI | • Menarche | Pre-menarche girls (n = 204) Post-menarche girls (n = 693) | Girls with higher BMI achieved menarche earlier at ages 8 and 9 than girls with lower BMI (p < 0.001) |
| Anyanwu et al 2016 [33] Nigeria | Cross-sectional | School girls 6 – 18 years (n= 1155) | BMI | Pubic hair development Breast development Menarche | Prepubertal overweight/ obese girls < 7 years (89%) Pubertal overweight/ obese girls < 7 years (11%) | Obese girls had lower mean ages of 6.80 ± 0.45 and 6.67 ± 0.52 years at P2 and B2, respectively, compared to nonobese girls (P < 0.05), and lower AAM (p < 0.001) |
| Ugege et al 2017 [34] Nigeria | Cross-sectional | Primary school girls from private and public schools, 8 – 13 years (n = 158) | BMI | •B2 | Underweight girls (n = 0) Normal weight girls (n = 154) Overweight girls (n = 2) Obese girls (n = 12) | Overweight/obese girls were more likely to have early-normal pubertal onset compared with normal weight girls (odds ratio [OR]; 1.259, p = 0.001) |

| Authors/ Publication Year/ Country | Study Design | Participants | Nutritional Status Outcomes | Pubertal Development Assessment | Comments | Main Findings |
|---|-----------------|---|--------------------------------|---|--|--|
| 2018 [3] Nigeria | Cross-sectional | Girls from private and pub- lic schools 6 – 15 years (n = 800) | IM | Breast development Pubic hair development Achievement of menarche | Underweight girls (n = 66) Normal weight girls (n = 634) Overweight girls (n = 64) Obese girls (n = 36) | BMI was a determinant of achievement of menarche (p = 0.001), pubic (p < 0.001) maturation Breast development was significantly associated with normal nutritional status (AOR; 4.5, p < 0.001), and obesity (AOR; 15.4, 2, p < 0.001), and obesity (AOR; 15.4, 2, p < 0.001) with reference to underweight (AOR; 4.7, p < 0.001) with reference to underweight (AOR; 15.7, p < 0.001) with reference to underweight (AOR; 15.7, p < 0.001) with reference to underweight (AOR; 0.1, p = 0.005), obesity (AOR; 0.1, p = 0.005), with reference to underweight (AOR; 0.1, p < 0.009) with reference to underweight (AOR; 0.1, p < 0.009) with reference to underweight (AOR; 0.1, p < 0.009) with reference to underweight (AOR; 0.1, p < 0.009) with reference |
| Chidumwa et al 2020 [35] South Africa | Longitudinal | Girls (<i>n</i> = 280) and boys (<i>n</i> = 263) in urban settings 0 – 17 years (<i>n</i> = 543) | BMIZ | • Tanner breast develop- ment (for girls) • Tanner genital develop- ment (for boys) | Stunted girls (n = 50) Non-stunted girls (n = 230) Stunted boys (n = 75) Non-stunted boys (n = 188) | Stunting was associated with lower relative risk of faster pubertal trajectories (p < 0.001) |
| Garenne et al 2021 [36] Nigeria | Cross-sectional | Girls 15 – 19 years (n =1884) BMI |) BMI | • Menarche | | • Taller and heavier girls reached menarche earlier compared to shorter and lichter cirls (n < 00001) |

| Author Year | Study Selection | | | | Study Comparability | Study Outcome | | | Score | Score Study Quality |
|--|---|--|------------------------------------|--|---|--------------------------------|--|--|-------|---------------------|
| Country | 1) Representativeness of the exposed cohort | 2) Selection of the non-exposed cohort | 3) Ascertainment of exposure | 4) Demonstration that outcome of interest was not present at start of study | Comparability of cohorts based on the design or analysis | 1) Assessment of outcome | 2) Was follow-up long enough for outcomes to occur | 3) Adequacy of follow up of cohorts | | |
| Simondon et al 1998 [18] Senegal | - | | | - | 0 | 0 | - | 0 | 2 | HRB |
| Benefice et al 2001 [19] Senegal | - | - | - | - | 0 | - | - | - | ~ | LRB |
| Gilette-Netting et al 2004 [21] Zambia | - | F | - | | 0 | - | - | 0 | Q | HRB |
| Salgin et al 2015 [29] South Africa | F | - | - | - | 2 | 0 | - | 0 | ~ | LRB |
| Lundeen et al 2016 [<u>30]</u> South Africa | - | 0 | - | 2 | - | 7 | - | 0 | ø | LRB |
| Chidumwa et al 2020 [35] South Africa | - | - | - | | - | 0 | - | 0 | Q | HRB |
| The NOS for cohort s | The NOS for cohort studies presents the following categories for rating: low risk of bias (LRB): 7 - 9 points, high risk of bias (HRB): 4 - 6 points, and very high risk of bias (VHRB): 0 - 3 points [1] | ving categories for ratin | ng: low risk of bias (l | LRB): 7 - 9 points, high | risk of bias (HRB): 4 - 6 | points, and very | high risk of bias (VHRB | 3): 0 - 3 points [1] | | |

 Table 2
 Assessment of study quality—cohort studies

Reference [<mark>37</mark>]

| Author Year | Study Selection | | | | Study Comparability | Study Outcome | | Score | Study Quality |
|--|---|--|---|---------------------------|-------------------------------------|--------------------------------|---------------------------|-------|------------------|
| Country | 1) Representativeness of the sample | 2) Selection of the control group | 3) Ascertainment of the exposure (disease) | 4) Non- respondents | 1) Study controls for factors | 1) Assessment of outcome | 2) Statistical test | | |
| Dare et al 1992 [17] Nigeria | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 6 | SAT |
| Abioye et al 1997 [4] Nigeria | 1 | 0 | 1 | 1 | 0 | 1 | 0 | 4 | UNSAT |
| Campbell et al 2004 [20] Zambia | 0 | 1 | 1 | 2 | 1 | 2 | 1 | 8 | G |
| Leenstra et al 2005 [13] Kenya | 1 | 1 | 1 | 2 | 1 | 1 | 1 | 8 | G |
| Omigbodun et al 2010 [22] Nigeria | 1 | 1 | 0 | 2 | 1 | 1 | 1 | 7 | G |
| Goon et al 2010 [23] Nigeria | 1 | 1 | 1 | 2 | 1 | 1 | 0 | 7 | G |
| Aryeetey et al 2011 [24] Ghana | 1 | 1 | 0 | 2 | 1 | 1 | 1 | 7 | G |
| Onyiriuka & Egbagbe 2013 [26] Nigeria | 1 | 1 | 1 | 2 | 0 | 1 | 1 | 7 | G |
| Ayele & Berhan 2013 [25] Ethiopia | 1 | 1 | 1 | 2 | 1 | 1 | 1 | 8 | G |
| Mpora et al 2014 [27] Uganda | 1 | 1 | 1 | 0 | 0 | 1 | 1 | 5 | SAT |
| Rebacz- Maron 2015 [28] Tanzania | 0 | 0 | 1 | 2 | 0 | 1 | 1 | 5 | SAT |
| Mao & Dalvie 2016 [31] South Africa | 1 | 1 | 1 | 1 | 2 | 1 | 1 | 8 | VG |
| Nwokocha et al 2016 [32] Nigeria | 1 | 1 | 1 | 0 | 0 | 1 | 1 | 5 | SAT |
| Anyanwu et al 2016 [33] Nigeria | 1 | 1 | 1 | 2 | 0 | 2 | 1 | 8 | G |
| Ugege et al 2017 [34] Nigeria | 1 | 1 | 1 | 2 | 0 | 2 | 1 | 8 | G |

Table 3 Assessment of study quality—cross sectional studies

Table 3 (continued)

| Author Year | Study Selection | | | | Study Comparability | Study Outcome | | Score | Study Quality |
|---|---|--|---|---------------------------|-------------------------------------|--------------------------------|---------------------------|-------|------------------|
| Country | 1) Representativeness of the sample | 2) Selection of the control group | 3) Ascertainment of the exposure (disease) | 4) Non- respondents | 1) Study controls for factors | 1) Assessment of outcome | 2) Statistical test | | |
| lrewole et al 2018 [3] Nigeria | 1 | 1 | 1 | 2 | 1 | 2 | 1 | 9 | VG |
| Garenne et al 2021 [<mark>36</mark>] Nigeria | 1 | 1 | 1 | 0 | 0 | 1 | 1 | 5 | SAT |

The adapted NOS for cross-sectional studies presents the following categories for rating: very good (VG): 9 - 10 points, good (G): 7 - 8 points, satisfactory (SAT): 5 - 6 points, and unsatisfactory (UNSAT): 0 - 4 points [1]

Reference [16]

Differences in pubertal development by nutritional status in SSA

In the current review, studies reported inconsistencies in the relationship between nutritional status and pubertal development both within and across different countries. For example, in a study in Senegal, no differences were observed in breast development between adolescent girls stunted and not stunted at infancy [19]. However, achievement of breast development in girls was associated with lower likelihood of stunting in Kenya [13], and higher HAZ and BMI in South Africa [30]. Moreover, while a study in Ethiopia reported a negative association between menarche age and BMI [25], a study in Ghana reported a positive association between these variables [24], and there was no association between menarche age and BMI in Uganda [27]. These differences may be attributed to ethnic or racial differences which are unclear in the various studies. It is speculated that ethnicity is a determining factor in physique characteristics and fat patterns [27]. Also, epigenetics may be considered as a role-player in these discrepancies, changing the trend of pubertal development in same and different countries, with changing nutrition and related lifestyle behaviours.

In Zambia, rural boys showed later onset, slower progression and later completion of testicular growth compared to their counterparts in urban settings [20]. The investigators observed that, completion of testicular growth in the slowly growing rural population of adolescents in Zambia, was linked to greater triceps skinfold suggesting that testicular growth is associated with energetic availability even under poor nutritional conditions. The researchers attributed this to the poorer somatic growth of rural adolescents, which reflects in their height and upper arm muscle. They concluded that variation in pubertal onset, progression and maturation in boys is related to energetic status despite genetic factors.

Comparing current review with previous studies

Previous reviews that investigated nutritional status and pubertal development have not included many studies in SSA. In a recent review of 27 studies globally [8], six (6) studies were in SSA. Two (2) of these studies did not report associations of nutritional status with pubertal development [38, 39]. One (1) study compared pubertal development of HIV and non-HIV infected girls. Therefore, only three (3) studies [27, 28, 34] from the previous review that reported associations of pubertal development with nutritional status overlap with the current review of anthropometric status, body composition and pubertal development in SSA.

The previous review included 27 studies that described healthy adolescents born in and after 1998, with the latest publication in 2018 [8]. Researchers identified studies that described age at menarche (AAM) and/or Tanner Stages 2 to 5. There was a similar trend of advanced pubertal development with higher BMI. The meta-analysis showed earlier onset of menarche, B2 and gonadal development stage 2 by Tanner Staging (G2) for overweight compared to thin and normal children, classified by BMI [8]. Likewise, adolescents with normal BMI from the African region experienced later age at menarche compared with adolescents from other WHO regions (Eastern Mediterranean Region, Region of the Americas, South-East Asia Region, Western Pacific Region, and the European Region) [8]. The reviewers suggested that AAM and puberty vary among races and countries. Moreover, the meta-analysis suggested a potential association between overweight/obesity and earlier onset of female puberty,

although the relationship was not significant, unlike twenty-one (21) studies in the current review (Table 1), which found significant associations.

In the current review, although the sample in Uganda found no significant association between menarche age and BMI or WC, HC was inversely correlated with menarche age [27]. Likewise, cross-sectional data from the third NHANES III showed that among 10–14 years old females, unit increases in HC is associated with 24% odds of attaining menarche; nevertheless, increases in WC and triceps are associated with 7% and 9% lower odds of menarche, respectively [27]. It is suggested that even though HC is correlated with BMI, HC supports protective metabolic functions.

It is important to note that each of the 23 studies investigated some pubertal milestones. Therefore, comparing pubertal development across the 9 countries was short of complete and accurate comparison for pubertal milestones of boys and girls. For example, only one study investigated the association of HAZ and BMIZ at 5 years, with pubic hair development in adolescents in South Africa. Also, most studies assessed breast development (8 studies) and menstruation (17 studies) for females, while 4 studies assessed genital development for males. There was a relatively smaller proportion of boys, compared to girls, assessed for pubertal development. This reduces the extent of investigations of pubertal development in boys as most studies do not assess all pubertal milestones. Furthermore, there were differences in pubertal assessment methodologies like self (pubertal development scale) and clinical (Tanner staging) assessment of various pubertal milestones, across the studies which challenged comparison across studies.

Possible mechanism for observed associations

Pubertal development is stimulated by synergistic effects of increased secretion of gonadal sex steroids, growth hormones (GH) and insulin-like growth factor 1 (IGF-1) [40]. Although the mechanism for metabolic control of puberty is unclear, recent findings have shown relevance of novel neurohormonal and molecular mechanisms that include key cellular energy sensors [41]. Leptin and estrogen levels produced in fat cells have been suggested to play a vital role in stimulation of pubertal development [30]. At the skeletal level, increased IGF-1, with increased insulin, lean body mass/muscle mass/fat mass stimulate chondrocyte maturation and proliferation, and bone mineral accretion increasing bone maturation [42]. This process stimulates pubertal growth spurt. Between the ages of 6 and 8 y, rises in adrenal hormones (like cortisol and dehydroepiandrosterone) and gonadal hormones (like testosterone, estradiol and progesterone) stimulate observable physical changes like body hair [43].

In a state of increased weight, there is increased levels of leptin, insulin, IGF-1 [42] and cortisol [44]. Increased insulin and IGF-1 levels promote increased lean body mass, muscle mass and fat mass, stimulating growth spurt. Increased Leptin and IGF-1 contribute to skeletal development by stimulating chondrocyte maturation and proliferation, bone mineral accretion and increased bone maturation. These developments ultimately lead to the achievement of pubertal growth spurt. Increased Leptin stimulates the hypothalamus to generate luteinizing hormone releasing hormone, the pituitary to secrete gonadotropin, adrenals to secrete adrenal steroids and gonads to secrete sex steroids [42]. The secretion of gonadotropin and sex steroids expedite pubertal development [42]. The sex steroids and skeletal maturation contribute to growth spurt [42]. A serine/threonine protein kinase, mTOR, activated in energy excess, contributes to mediation in the leptin effects of expediting puberty through modulation of the hypothalamic expression of kisspeptin 1 gene, which acts as regulator of reproductive functions [41]. Available evidence suggests that there is significant increase in dehydroepiandrosterone at the time when a child's BMI is highest compared to when it was lower [42]. This implies that increased body fat may significantly contribute to the activation of adrenal androgen production and the onset of adrenarche.

Moreover, the gluteofemoral fat (lower-body fat) has been inversely associated with AAM, blood glucose, blood pressure and lipid levels [27]. It has also been found to be associated with reduced risk of cardiovascular diseases, all-cause and cardiovascular mortality [27]. While leptin and adiponectin levels have been positively associated with gluteofemoral fat, inflammatory cytokine levels have been inversely linked [45]. It is speculated that gluteo-femoral fat traps excess fatty acids and prevents chronic exposure to elevated lipid levels [27].

On the other hand, in a state of malnutrition, decreased BMI, and reduced fat mass, there are increased levels of cortisol and GH, but reduced levels of leptin, insulin and IGF-1 [42]. Increased cortisol, GH and decreased insulin are associated with decreased lean body mass, muscle mass and fat mass, delaying growth spurt [42]. Moreover, decreased leptin and IGF-1 delay chondrocyte maturation and proliferation, decrease bone mineral accretion and delay bone maturation delaying growth spurt [42]. Consequently, in the hypothalamus, there is inhibition of luteinizing hormone releasing hormone generation and delay in gonadotropin secretion in the pituitary, as a result of decreased leptin levels, delaying pubertal development [42]. In the adrenals and gonads, there is delay in sex steroid secretion delaying the pubertal growth spurt and pubertal development. Leptin deficiency can lead to absence of puberty [42]. Adenosine monophosphate activated protein kinase, a heterotrimeric serine/threonine kinase, interplays with kisspeptin neurons in the metabolic control of puberty, mediating the repressive effects of nutritional deficiency. Sirtuin 1, an energy sensor, represses kisspeptin 1 expression and delays puberty during energy restriction through the epigenetic regulation of arcuate nucleus kisspeptin 1 neurons.

Strengths and limitations

The 6 longitudinal cohort studies allowed for assessment of precise pubertal timing and development. Nevertheless, the cross-sectional nature of 17 studies limited the capture of precise timing of pubertal milestones. Consequently, some reported pubertal timings may have been overestimated or underestimated due to the time of measurement and the possibility of incorrect recall. Specifically, adolescents tend to overestimate as much as underestimate pubertal development [46].

While the NOS provided a structured approach to evaluating the quality of studies included in the review, potential biases in these studies may have influenced our findings. Such biases include those related to subject selection, outcome measurement, and inadequate control for confounders, which could affect the observed associations between nutritional status and pubertal milestones. For example, failure to control for socioeconomic factors may confound results, making it difficult to isolate the specific effects of nutrition. Additionally, publication bias could have skewed our findings toward significant associations. Given these considerations, we have interpreted our results cautiously and recommend that future studies use rigorous designs to minimize bias.

It is noteworthy that the variability in data across studies, particularly due to differences in methodology (e.g., self-report versus clinical assessment), may affect the generalizability of our findings. Self-reported measures could introduce recall bias, while clinical assessments may vary depending on the resources and training available. Similarly, differences in participant characteristics across studies (e.g., age ranges, socio-economic backgrounds, and nutritional contexts) could limit the applicability of our findings to broader populations in SSA. Therefore, our findings should be interpreted with caution.

The review may be biased from the varying quality of the studies in SSA. This distribution suggests potential bias in the review due to the inclusion of lower-quality studies, particularly cross-sectional studies, which are more prone to confounding and other biases. Consequently, the presence of unsatisfactory and high risk of bias studies could weaken the strength of the summarized evidence. Notably, the review assessed associations between different anthropometric and body composition indices versus assessing puberty by different methods across diverse populations. Therefore, the findings may be generalizable to populations in SSA with similar sample characteristics.

Conclusions

Some studies have shown differences (South Africa, Zambia) while others have shown no significant associations (Kenya). Population variation in timing of reproductive maturation is poorly understood. Subsequent studies should focus on all Tanner Stages or adapt the Peterson pubertal development scale score for detailed assessment of pubertal development in a cohort study. Moreover, considering that ~15% of the review population was male, there must be a conscious effort to include same number of boys as girls to help bridge the gap in pubertal assessment of males. Finally, this review serves as baseline for future monitoring of trends in association between nutritional status and pubertal development in SSA. This adds to the body of knowledge of understanding racial and country differences in nutritional status and pubertal milestones. Furthermore, findings serve as a point of reference for further literature and clinical application.

Staging

Abbreviations

| Age at menarche |
|--------------------------------------|
| Breast development stage 2 by Tanner |
| BMI z-scores |
| Good |
| Height for age z score |
| Hip circumference |
| High risk of bias |
| Insulin-like growth factor 1 |
| Low to middle income countries |
| Low risk of bias |
| Medical subject headings |
| Newcastle Ottawa Scale |
| Waist circumference |
| Satisfactory |
| Sub Saharan Africa |
| Unsatisfactory |
| Very good |
| Very high risk of bias |
| |

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s40795-024-00951-w.

Supplementary Material 1.

Acknowledgements

Not applicable.

Authors' contributions

HN: conceptualized the systematic review; HN and SAA: designed the systematic review; HN and LMDA: identified the studies; HN: conducted the data extraction and reviewed the papers; HN and LMDA: conducted quality

assessment of selected studies; HN: wrote the manuscript; EP, SAA and BMO: reviewed the draft manuscript; HN and SAA: had primary responsibility for final content. All co-authors: read and approved the final manuscript. Due to her death during the preparation of this manuscript co-author ELP was unable to read and approve the final manuscript.

Funding

This review was funded by the NIH [as part of PhD Scholarship]. The findings, recommendations and conclusions in the article are those of the authors; these are not influenced by the funders.

Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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Received: 17 April 2024 Accepted: 14 October 2024 Published online: 24 October 2024

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