

Determinants of Age at Adiposity Rebound in Filipino Pediatric Outpatients of a University Hospital



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ABSTRACT

Objectives: Adiposity rebound (AR), the childhood period at which body mass index (BMI) rises from its lowest point, is linked to increased risk of later obesity. The study aims to determine the average age at AR, describe baseline characteristics and analyze the correlation between these characteristics and timing of AR in a population of Filipino pediatric outpatients.

Design: Seven subjects born between 2016 and 2019 from a University Hospital Outpatient Department participated in this cross-sectional analytic study. Childhood anthropometrics were

retrospectively collected to determine the age at AR by plot visual inspection. Sex, birth weight and gestational age were obtained from hospital records; breastfeeding duration, maternal BMI, parental obesity, maternal age, maternal smoking, education, parity and family income were gathered through a questionnaire completed by mothers or guardians. Associations were assessed using bootstrap univariate linear regression.

Results: The mean age at AR was 3.2 years (SD = 1.2). Vaginal delivery was significantly associated with later age at AR compared to cesarean section ($p = 0.035$). Socioeconomic status at ages 2 to 5 showed positive association with delayed AR. Higher monthly family income ($\geq \text{P}19,000$) at ages 2 to 5 years was significantly associated with delayed age at AR. Other baseline childhood and parental factors showed no significant correlation with age at AR.

Conclusion: These results highlight the complex and context-dependent nature of AR, emphasizing the need for further studies to better understand and mitigate early obesity risk in Filipino children.

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INTRODUCTION

In the Philippines, the proportion of overweight and obese Filipino adults aged 20 years old and above has been continuously increasing, reaching a prevalence of 36.6% in 2018-2019, according to the 2019 Expanded National Nutrition Survey conducted by the Food and Nutrition Research Institute of Department of Science and Technology (DOST).[1] In all age groups, urban households in the Philippines were reported to have a higher risk for being overweight and obese than rural households. The growing prevalence of obesity among children and adolescents worldwide [2,3] reflects poor forecast and ineffective public health response to the growing burden of obesity. A considerable body of literature indicates that overweight and obese children are highly likely to remain obese into adulthood.[4-6] These individuals are more predisposed to develop non-communicable diseases such as diabetes, hypertension and cardiovascular conditions at an earlier age.

Adiposity rebound (AR) is a key focus of investigation in many international studies. It refers to the period in childhood at which body mass index (BMI) rises from its lowest point after the initial decrease at a certain age during infancy.[7] Several studies have demonstrated AR as an important predictor of developing obesity. Specifically, an early timing of AR is associated with higher BMI and index of adiposity in later life, whereas it is reported that late AR serves as a protective factor in reducing the risk of obesity among children.[7-23]

AR is one growing aspect of investigation in many international journals. The earliest literature on the phenomenon of AR was accounted for by Rolland-Cachera, et al.[24] wherein adiposity curves were assessed in 151 French children from 1 month to 16 years. Accordingly, earlier age at rebound was found to be predictive of future adiposity, as observed at 16 years of age. Several possible explanations were suggested by the pioneer authors. Physiological adipose tissue evolution encompasses phases that may explain AR occurrence as cited in Rolland-Cachera, et al.[24] It was noted that between birth and the first year of life, adiposity increases due to a hypertrophic action of adipocytes wherein cell size enlarges while cell number remains the same. These changes were reflected as an increase in BMI at this period. In the next few years, growth of

cells remains stable, but body height continues to increase, reflected as a decreasing trend of BMI at this period. At around age 6 years, a second period of adiposity growth was observed, but this time in both a hypertrophic and hyperplastic manner where growth is seen in both size and number of cells. This second rise in BMI was termed AR. The timing of AR is primarily assessed by three approaches: 1) velocity of weight and height, 2) plot visual inspection and 3) polynomial models, where plot visual inspection of BMI is the most frequently used approach in literature. This technique identifies the last minimum BMI before an upward trend in BMI, and the time at which this happens is the age at AR.[17,18,25-29]

Previous studies depict the role of an earlier age at AR as a predictor of childhood, adolescent and adult obesity. Rolland-Cachera, et al. [24] first suggested the association of early adiposity rebound (EAR) with higher adiposity level at age 16 years. Children with EAR were at higher risk of developing child obesity at age 7 years [14,21] and 10 years.[22] EAR was also found to be associated with higher risks of obesity among ages 12 years old,[7,11,12] 12.9 years old,[19] 13.5 years old,[20] 14 years old,[23] 15 years old[17] and 18 years old.[9,15] Several studies demonstrate AR as an indicator of the development of type 2 diabetes mellitus (T2DM), since it has been illustrated that children with earlier AR are more likely to have T2DM later in life.[1,12,13] Increased insulin resistance was demonstrated to be higher in those with EAR.[7,20,22,30] The role of an earlier AR was shown to be related with worse cardiometabolic outcomes, where it has been suggested that both men and women with EAR are more likely to develop metabolic syndrome.[18] Earlier AR was shown to increase adverse cardiometabolic scores at ages 7, 12.9 and 18-20 years old, respectively.[19,30,31] Higher triglycerides [22, 30] increased fat mass and HDL-C,[20] and higher systolic blood pressure and C-reactive protein [22] associated with EAR supports the mentioned findings.

It is noted that the timing of AR varies considerably between populations. In the original French study by Rolland-Cachera, et al.[24] average age at AR was determined at about 6 years of age. European data reveal a range of AR ages typically occurring around 5 to 6 years of age, with age cut-offs on AR being heterogeneous in literature.[11,16,17]

Asian populations show wider variation with Korean cohorts experiencing AR as early as 4.3 to 6 years [32-34] while children in Japan and China typically experience AR beyond 6 years of age.[23,35] The current literature reflecting most of the understanding of age at AR is derived from non-local studies conducted across several cohorts and communities, each influenced by their own cultural setting. Therefore, the applicability of existing international standards may be limited and underscores the need for population-specific data.

The following summarizes how the timing of AR may serve as an early marker for reducing the risk of obesity. Several prenatal and early-life factors influence AR timing. Sex differences have been observed, with girls more likely to experience early AR and demonstrate higher obesity risk,[36-38] though other studies found no significant effect of sex on AR timing.[17,25] Birth weight has shown mixed associations—both large-for-gestational-age (LGA)[39] and small-for-gestational-age (SGA)[40] neonates have been linked to early AR. Gestational age appears inversely related to BMI and weight z-scores.[36,41] Preterm birth may predict late AR,[40] but this was not consistently supported.[25] Mode of delivery, including cesarean section, has shown no significant influence on AR timing.[39,40] Breastfeeding duration is consistently linked to AR timing: shorter durations increase the likelihood of early AR,[29] while longer breastfeeding, especially exclusive breastfeeding beyond 4–5 months, appears protective against early AR.[40,41] Parental BMI is a strong and consistent predictor of early AR and later obesity. Maternal BMI and pre-pregnancy weight correlate with child BMI trajectories.[38,41-43] Children of parents with obesity, or with at least one obese parent, are more likely to experience early AR.[17,25,26] Maternal silhouette and BMI also contribute to this trend.[18,28,37] Maternal age has also been implicated, with older maternal age correlating with early AR in some studies [28,40] although others did not find this relationship.[25,41] Findings on maternal smoking are mixed: while some studies report an association with early weight gain and obesogenic trajectories,[25,38,41] others show no significant effect.[17,28,42,44] The role of parental education is inconclusive. While limited maternal education may be linked to early AR,[25,37] most studies found no consistent relationship.[17,26,28,39-41]

Similarly, socioeconomic status has mostly shown no effect [17,26,37,44] despite a single finding linking early milk introduction in low-income settings to later obesity.[41] Lastly, parity showed mixed results: while multiparity was associated with obesogenic trajectories,[43] lower parity was linked to early AR in one study,[28] but others found no association. [17,26,40]

The increasing rate of obesity across all age groups makes the timing of AR an interesting perspective to address this growing public health concern. A substantial number of studies have already reviewed the timing of AR since its earliest conception.[24] However, a variation in the normal age at AR appears to be noteworthy due to ethnic and cultural differences. It is proposed that sociodemographic factors play a considerable role accounting for differences in AR timing. Most studies available to date references on non-Southeast Asian cohorts, and literature review may suffice to declare scarcity of AR-related studies in Southeast Asia. It is necessary to generate a reference range for this adiposity phenomenon in the Philippines. Key demographic characteristics that influence AR within the local population must be investigated to provide relevant estimates of the local mean age at AR. To advance studies on age at AR in the locale, a study aiming to generate a reference average age at AR and factors associated with its timing is necessary. This study is the first to examine the association between selected determinants and timing of AR among Filipino children, with the primary objective of determining the (a) average age at AR, (b) baseline characteristics and (c) correlation of these baseline characteristics to the age at AR among Filipino pediatric outpatients known to the University Hospital.

METHODOLOGY

Study Design and Participants

The study employed an analytical observational approach using a cross-sectional design to determine the age at AR and examine its association with selected childhood and parental characteristics. The primary outcome was age at AR derived from BMI trajectories and independent variables include baseline characteristics of the child and parents, obtained through medical records and administered questionnaires. These variables were measured concurrently.

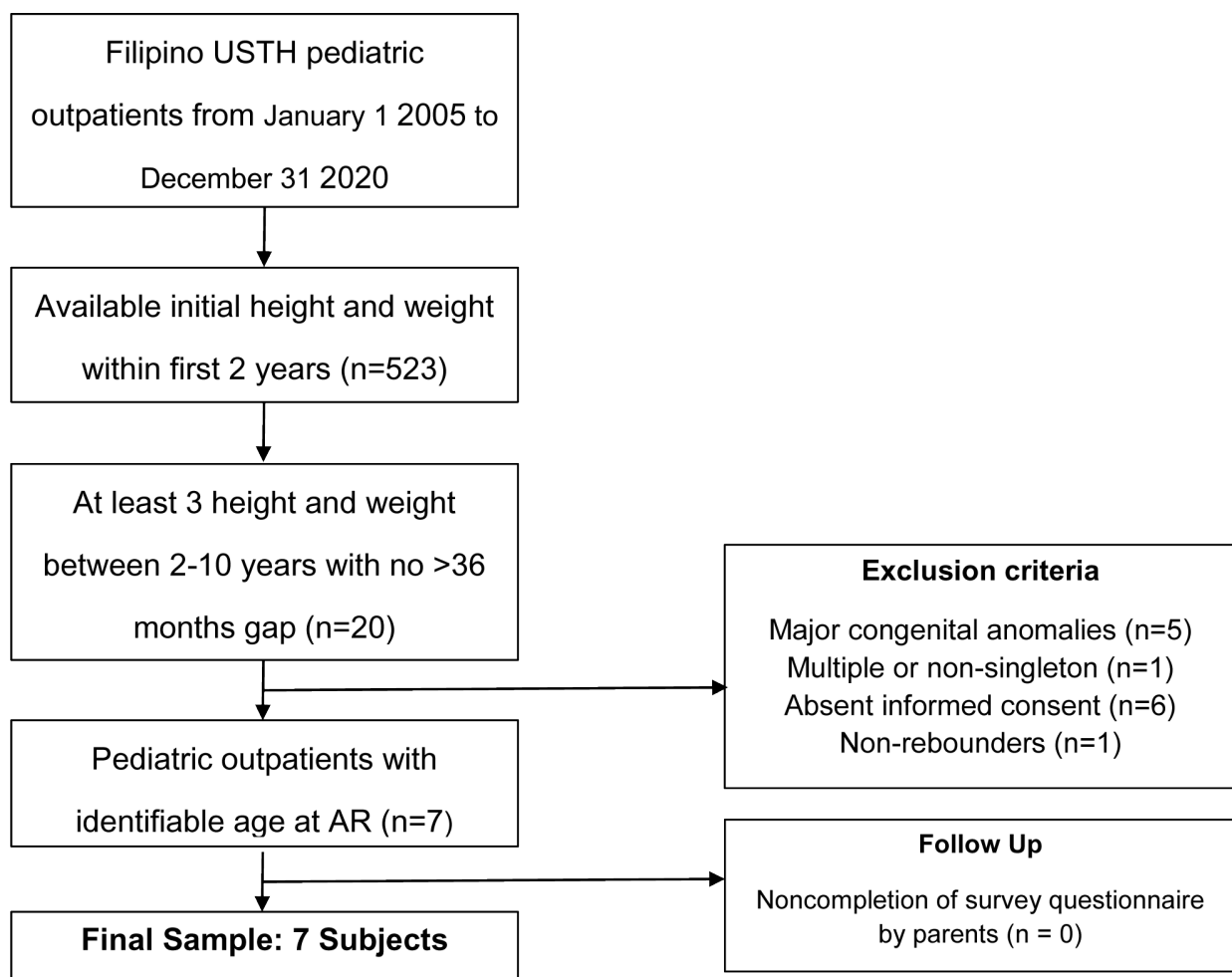


Figure 1: Study flow chart of participants

Inclusion and Exclusion Criteria

Eligible participants were Filipino pediatric outpatients at the UST Hospital, born between January 1, 2005 and December 31, 2020, with an identifiable age at AR. Inclusion criteria included initial length and weight measurements taken within the first two years of life to establish BMI baseline and at least three pediatric height and weight records between the ages of 2 and 10 years, with no gap exceeding 36 months between any two consecutive measurements. Participants with documented major congenital anomalies, multiple births, no parental or guardian consent, those with non-rebounder BMI trends or had inconclusive AR (no sustained rise of ≥ 0.01 kg/m² after BMI nadir and those whose parents or guardian failed to complete the required questionnaire were excluded from the study.

Sample Size Calculation

A minimum of eight children were required as samples for the study to achieve 80% power of

the test at two-sided 5% level of significance (Table 2). This sample size was based on the result of Estévez-González [27] that EAR was correlated ($r = -0.856$) with higher BMI at age 8. Sample size was calculated using the formula by Lachin (1981). [45] In the current study, only seven children ultimately met all inclusion criteria and were included in the final analytic sample due to limitations in data availability and stringent selection parameters.

Data Collection

A quantitative approach was utilized. Data were collected through two sources: pediatric health records and self-administered questionnaire completed by the participant's mother or legal guardian. Sociodemographic profile such as duration of breastfeeding, maternal height and pre-pregnancy weight to compute for BMI, parental history of obesity, maternal age at birth, maternal smoking during pregnancy, parental education, parity and socioeconomic status based on monthly family income per age of child were gathered using

Table 1: Inclusion and exclusion criteria for a cross-sectional study of Filipino UST pediatric outpatients

| Criteria | Reason |
|--|--|
| Inclusion Criteria | |
| Filipino UST Hospital pediatric outpatients born between January 1, 2005 and December 31, 2020 with an identifiable AR (second rise in BMI): | An identifiable AR or the second rise in BMI is needed to compute the age at AR. The two subcriteria below are prerequisites to estimate age at AR. The years were arbitrarily selected on the basis of availability of health records and likelihood to follow-up on parents of the samples. If, for example, 2021 is included, the participant may not be able to fulfill subcriteria 2. If for example, 2004 is included, parental follow-up may be too difficult to reach. |
| Subcriteria 1: Available initial length and weight measurements within the first 2 years of life | Anthropometric measurement within the first 2 years is needed to establish baseline BMI and document whether a first rise already occurred among the population. |
| Subcriteria 2: At least three pediatric height and weight records between 2-10 years of age with no gaps of missing data >3 years (36 months) between two consecutive measurements | Three pediatric height and weight records were arbitrarily selected to ensure an accurate and robust assessment of age at AR that is based on the nadir from 2-10 years old. As defined, age at AR is the time when a child's BMI begins to rise from its lowest point after infancy and not just from a relatively low point. |
| Exclusion Criteria | |
| General | |
| Multiples or non-singletons | Multiples are expected to produce non-independence of observations in physical development, as adapted from González, et al.,[30] Besharat Pour, et al.,[29] and Fonseca, Moreira, & Santos.[22] |
| Diagnosis of major congenital anomalies at birth or through childhood (eg, neural tube defects, congenital heart defects and chromosomal abnormalities) | Congenital anomalies or malformations develop prenatally and may be identified before or at birth, or later in life. Anomalies at birth or childhood are expected to produce differences in physical development, as adapted from Cissé, et al.[25] and Fonseca, Moreira, & Santos.[22] |
| Age at AR Estimation | |
| Non-rebounders and inconclusive age at AR | Children whose serial BMI trend only courses upwards or downwards (non-rebounders) or does not meet the criteria for subsequent BMI values of at least 0.01 kg/m ² or higher after nadir for at least two succeeding observations (inconclusive age at AR) are excluded to ensure quality of exposure measurement. |
| Follow-Up | |
| Noncompletion of survey questionnaire by parents | For data completion and analysis |

the survey questionnaire. Data such as sex (male/female), birth weight (kilograms) and gestational age at birth (preterm/term/post-term) were obtained from hospital records.

Outcome Measures

The primary outcome measure in this study was the age at AR, defined as the age at which the last minimum BMI occurs before a subsequent and sustained increase in BMI over time. This

was determined through visual inspection of serial BMI plots, using criteria such as identifying the lowest BMI between the ages 2 and 10 followed by at least two consecutive increases of ≥ 0.01 kg/m². Outcome measurement involved plotting BMI (kg/m²) calculated from health records and recording the corresponding dates and ages. Baseline characteristics, considered as independent variables, were divided into childhood (eg, sex, birth weight, gestational age, mode of delivery and exclusive breastfeeding duration) and parental

Table 2: Sample size calculation

| Abbreviation | Description | Input | Results |
|--------------|-------------------------------------|---------------|---------|
| α | Significance level (two-sided test) | 0.05 | |
| $1-\beta$ | Power of the test | $\beta = 0.2$ | |
| r | Sample correlation | 0.856 | |
| N | Sample size needed | | 8 |

factors (eg, maternal pre-pregnancy BMI, parental obesity history, maternal age, maternal smoking during pregnancy, parental education, parity and socioeconomic status), all assessed for their potential influence on the timing of AR.

Definition of Terms

Age at Adiposity Rebound (AR): The time when a child's BMI begins to rise from its lowest point after infancy. In this study, age at AR was operationally expressed as a continuous variable (years). The following criteria to determine the age at AR were applied:

- Age at lowest BMI-value between age 2 and 10
- Subsequent BMI values at least 0.01 kg/m² higher after nadir for at least two succeeding observations.
- In case of a plateau (two consecutive values were equal), use of the last value.

Body Mass Index (BMI) or Quetelet Index:

Derived by dividing a person's weight in kilograms by the square of their height in meters. This study followed the Asia-Pacific cut-off points for overweight (BMI ≥ 23) and obesity (BMI ≥ 25).

Childhood Obesity: Childhood obesity is derived from age- and sex- specific criteria set by the WHO. [46] For children under 5 years old, obesity is when weight-for-height is at $>+3SD$ above WHO Child Growth Standards median. For children 5-19 years old, obesity is when BMI-for-age is at $>+2SD$ above the WHO Growth Reference median.

Statistical Methods

Descriptive and inferential statistics were utilized in the study. Means and standard deviations were used to present data such as age at AR, birth weight, duration of exclusive breastfeeding, maternal BMI and maternal age at birth in quantitative form. Categorical variables such as sex, gestational age at birth, mode of delivery, maternal and paternal

history of obesity, maternal smoking during pregnancy, maternal and paternal education, parity and socioeconomic status were summarized using counts and percentages. Bootstrap univariate linear regression was employed to identify variables that were significantly correlated with age at AR because of the limited sample size. All statistical analyses were conducted using Stata version 14. A p-value of less than 0.05 was considered indicative of statistical significance in determining factors associated with age at AR.

RESULTS

Demographics and Characteristics of the Study Population

The final analytic sample comprised of seven pediatric outpatients, all born at term without congenital anomalies or exclusionary diagnosis. The participants, with a mean age at AR of 3.2 years (SD = 1.2, range = 2.2-5.3), were predominantly male (n = 4; 57.1%) and had a mean birth weight of 2.9 kg (SD = 0.5; range = 2.6-3.7). Majority were delivered vaginally (n = 5; 71.4%) and exclusive breastfeeding duration (months) is 6.5 months (SD = 8.0; range = 17.0-24.6).

Maternal characteristics showed a mean pre-pregnancy BMI of 21.7 kg/m² (SD = 2.9; range = 17.0-24.6) with 28.6% (n = 2) having a history of maternal and 42.9% (n = 3) of paternal obesity. None of the participants had a history of smoking during pregnancy. The mean age at childbirth was 33.9 years (SD = 8.3; range = 20-46), and the average parity was 1.1. Educational attainment varied, with two (28.6%) mothers having completed college, four (57.1%) completed high school and one (14.3%) finished elementary. As for the fathers, three (42.9%) finished college while four (57.1%) finished up to high school level. Median household income when the child was one year old was ₱9,500 to 19,000 and increased to ₱19,000 to 38,000 from age two to six years (Table 3).

Bootstrapped univariate linear regression identified mode of delivery (p = 0.035) as the only significant childhood predictor of age at AR, with vaginal delivery associated with a 1.14-year higher age at AR (Table 4). Among parental characteristics, income levels \geq ₱19,000 at ages 2-3 (p = 0.007) and 4-5 years (p = 0.005) were significantly associated with a later age at AR. No remarkable

Table 3: Socioeconomic status at different ages of the child with AR

| Socioeconomic Status | Summary |
|------------------------------------|-----------|
| At 1 year old, <i>n</i> (%) | 0 (0%) |
| ₱66,500 to 114,000 | 1 (14.3%) |
| ₱38,000 to 66,500 | 2 (28.6%) |
| ₱19,000 to 38,000 | 2 (28.6%) |
| ₱9,500 to 19,000 | 2 (28.6%) |
| <₱9,500 | |
| At 2 and 3 years old, <i>n</i> (%) | 1 (14.3%) |
| ₱66,500 to 114,000 | 0 (0%) |
| ₱38,000 to 66,500 | 3 (42.9%) |
| ₱19,000 to 38,000 | 2 (28.6%) |
| ₱9,500 to 19,000 | 1 (14.3%) |
| <₱9,500 | |
| At 4 and 5 years old, <i>n</i> (%) | 1 (14.3%) |
| ₱66,500 to 114,000 | 0 (0%) |
| ₱38,000 to 66,500 | 4 (57.1%) |
| ₱19,000 to 38,000 | 2 (28.6%) |
| ₱9,500 to 19,000 | 0 (0%) |
| <₱9,500 | |
| At 6 years old, <i>n</i> (%) | 2 (28.6%) |
| ₱66,500 to 114,000 | 0 (0%) |
| ₱38,000 to 66,500 | 3 (42.9%) |
| ₱19,000 to 38,000 | 1 (14.3%) |
| ₱9,500 to 19,000 | 0 (0%) |
| <₱9,500 | 1 (14.3%) |
| No response | |
| At 7 years old, <i>n</i> (%) | 1 (14.3%) |
| ₱66,500 to 114,000 | 0 (0%) |
| ₱38,000 to 66,500 | 2 (28.6%) |
| ₱19,000 to 38,000 | 0 (0%) |
| ₱9,500 to 19,000 | 0 (0%) |
| <₱9,500 | 4 (57.1%) |
| No response | |
| At 8 years old | 0 (0%) |
| ₱66,500 to 114,000 | 0 (0%) |
| ₱38,000 to 66,500 | 1 (14.3%) |
| ₱19,000 to 38,000 | 0 (0%) |
| ₱9,500 to 19,000 | 0 (0%) |
| <₱9,500 | 6 (85.7%) |
| No response | |

Note: Values expressed as counts (%).

associations were found with sex, birth weight, duration of breastfeeding, parental BMI, maternal/paternal history of obesity, education, parity and maternal age at birth.

DISCUSSION

This analytic cross-sectional study is the first to examine the association between selected determinants and the timing of AR among Filipino children. Among 523 children initially reviewed, only seven met the inclusion criteria for AR analysis due to strict requirements for

longitudinal anthropometric data. Despite the small sample size, this preliminary investigation offers early insights into potential correlates of AR timing within the context of a Filipino population. While AR has been identified as a critical window for the development of obesity and has been widely studied in other regions, its documentation and analysis remain underexplored in the Philippines. This study provides a step towards addressing that gap. Given the established link between EAR and future obesity risk, these results underscore the importance of early monitoring of pediatric growth patterns to support timely interventions.

Mean Age at Adiposity Rebound

Plot visual inspection is employed in this study due to better ability to estimate the age at AR compared to statistical models,[47] although the possibility of arriving at a different estimated age if a statistical model was applied is acknowledged. Nonetheless, there are different manners to classify the age at AR. Historically, the pioneer study of Rolland-Cachera, et al.[24] used specific age ranges to assign cut-offs: early (<5.5 years), normal (6–6.5 years) and late (>7 years). Ohlsson, et al.[16] used tertile cut-offs: early (<5.4 years), middle (5.4–6.8 years) and late (>6.8 years). Freedman, et al. [10] and Gonzales, et al.[30] used tertile cut-offs: early (≤ 5 years), middle (5–7 years), and late (≥ 7 years). Other studies used very early AR (<43 months or 3.58 years), early (<61 months or 5.08 years) and later (>61 months). [17] Whereas, Aris, et al.[19] provided another approach: early (<25th percentile), normal (25th to 75th percentile) or late (>75th percentile) AR. Haga, et al.[23] used a dichotomous classification of <6 years designated as EAR and >6 years as LAR, and Goh, et al.[34] simply defined EAR as the rebound of BMI before the sixth time period in their longitudinal data collection. Interpreting AR estimates across studies has been challenging, first due to population samples being compared spanning decades apart making comparisons difficult given the anticipated changes in environmental influences including increasing sedentary lifestyles, health access disparities and population income. Second, majority of studies are from Western and East Asian populations. The applicability of existing AR findings among Filipinos remains uncertain given that it is an underexplored area in local literature. The

Table 4: Bootstrapped linear regression on age at AR

| Variables | Coefficients | | p-value |
|---|--------------|-----------------|--------------|
| | Estimate | 95% CI | |
| Childhood Characteristics | | | |
| Sex: Male | 0.62 | -0.93 to 2.18 | 0.432 |
| Birth weight (kilograms) | 1.01 | -25.00 to 27.01 | 0.940 |
| Mode of delivery: Vaginal | 1.14 | 0.08 to 2.20 | 0.035 |
| Exclusive breastfeeding (months) | -0.08 | -0.52 to 0.36 | 0.721 |
| Parental Characteristics | | | |
| Maternal pre-pregnancy BMI (kg/m ²) | -0.30 | -0.77 to 0.17 | 0.206 |
| Maternal history of obesity | -0.09 | -1.61 to 1.43 | 0.906 |
| Paternal history of obesity | 0.20 | -1.69 to 2.10 | 0.833 |
| Maternal age at birth (years) | 0.06 | -0.07 to 0.19 | 0.341 |
| Maternal education: College graduate | 0.90 | -0.20 to 1.99 | 0.108 |
| Paternal education: College graduate | -0.58 | -2.14 to 0.98 | 0.468 |
| Parity | -0.27 | -1.43 to 0.89 | 0.649 |
| Socioeconomic status: | | | |
| At 1 year old | 0.83 | -1.13 to 2.79 | 0.405 |
| ₱19,000 to 66,500 | -0.59 | -1.87 to 0.70 | 0.372 |
| ₱9,500 to 19,000 | - Ref - | | |
| <₱9,500 | | | |
| At 2 and 3 years old | 1.50 | 0.41 to 2.59 | 0.007 |
| >₱19,000 | - Ref - | | |
| <₱19,000 | | | |
| At 4 and 5 years old | 1.37 | 0.41 to 2.33 | 0.005 |
| >₱19,000 | - Ref - | | |
| <₱19,000 | | | |
| At 6 years old | -0.35 | -1.98 to 1.27 | 0.671 |
| > ₱38,000 | - Ref - | | |
| <₱38,000 | | | |

interpretation of age at AR may be misconstrued for this study's demographic of Filipino children.

There is difficulty comparing this study's mean age at AR across literature given that most were retrieved from established cohorts of children that have either been longitudinally monitored in prospective studies or have been analyzed retrospectively, ie, the GOOD cohort in Sweden,[16] the ALSPAC,[17] and the Mysore Parthenon Birth cohort from Di Gravio, et al.[20] All of these are identifiably heterogeneous in terms of race, descent and cultural habits in dietary patterns and child rearing. Regardless, the pioneer example laid out by Rolland-Cachera, et al.[8] being the first prospective cohort to be longitudinally analyzed for AR was itself a study conducted with parallel investigations from a collaboration of multiethnic cohorts. This showcases that despite the varying characteristics of all these populations,

together with the differences of Western to Asian anthropometric definitions, it is suggested by Haga, et al.[23] that the timing of the age at AR is a human phenomenon that distinctly falls into a range of ages that is strikingly similar across historical studies regardless of heterogeneity in race and ethnicity. Therefore, even in the absence of a more suitable comparison for a sample drawn from seven pediatric outpatients of Filipino families from a low- to middle-income country, it is argued that the mean age at AR of 3.2 years is identified as EAR in reference to any of the studies retrieved, if not even lower than most cutoffs seen in current literature.

Finally, the age at AR of 3.2 years is a clinical indication to suspect the likelihood of developing obesity, insulin resistance leading to T2DM, and adverse cardiometabolic outcomes such as increased low-grade systemic inflammation, unfavorable lipid

profiles and increased systolic blood pressure. [19,20,22,30,31] Given that these outcomes occurring in later years of life are positively correlated in literature with EAR, it is therefore suitable to state that the early mean age at AR retrieved from the population sample has clinical grounds to be labeled as a well-supported risk factor.

Childhood Characteristics

Mode of Delivery: Among the childhood baseline characteristics examined, mode of delivery emerged as a significant determinant of AR, with children delivered vaginally experiencing a significantly later AR compared to those born via cesarean section. This association may be attributed to the role of early microbial colonization, where vaginal delivery facilitates exposure of the infant to maternal vaginal and fecal microbiota.[3,48,49] During passage through the birth canal, the infant's oral cavity, nasal cavity, skin and gastrointestinal tract become inoculated with these beneficial microbes including probiotics like *Lactobacillus reuteri*, *L. rhamnosus*, promoting the development of a diverse and metabolically beneficial gut microbiome. In contrast, cesarean-delivered infants tend to acquire skin and environmental microbes, potentially predisposing them to gut dysbiosis and metabolic disturbances that could accelerate the timing of AR.[50,51] However, this finding is not consistently observed across studies.[39,40] This inconsistency suggests that other confounding factors may be at play and that mode of delivery may have potential lasting impacts on early growth and long-term metabolic consequences.

Sex: In this sample, sex did not appear to be associated with the timing of AR. This finding aligns with the results of two studies which similarly indicate no significant association between sex and AR timing.[17,25] However, it contrasts suggestions that AR exhibits sex-specific patterns, with females more likely to experience earlier AR,[36,37] and that sex is significantly associated with obesogenic growth trajectories.[38,43] Differences in sample size, ethnic background and AR assessment likely account for discrepancies observed across studies. However, the sample size of this study is too small to analyze genders separately.

Birth weight: The present study showed no significant association between birth weight and timing of AR, as previously noted.[17,18,25,39,44]

While the majority of evidence supports absence of a relationship, two studies demonstrate EAR to be significantly associated with both LGA [39] and SGA.[40] This lack of association in the present study may be attributed to the fact that birth weight is a single-point measurement that does not capture longitudinal growth trends. In contrast, postnatal factors such as feeding practices and growth velocity are likely to play a more substantial role in determining the age at AR.[43] These findings emphasize the importance of early postnatal growth and modifiable lifestyle factors to predicting AR and its implications for later obesity risk.

Duration of Exclusive Breastfeeding:

The present data showed no influence of exclusive breastfeeding to the timing of AR. This result is consistent with previous studies that also reported no significant relationship between breastfeeding and AR timing. [18,27,39,43,44] The lack of association may stem from how breastfeeding is assessed, such as failure to distinguish exclusivity or account for its duration.

Parental Characteristics

Socioeconomic Status: A particular highlight of this study includes an analysis of the socioeconomic profile of the sample population by acquisition of the estimated income status every year from the age of 1. Children with an estimated household income of greater than Php 19,000.00 from the age bracket of 2 to 3 years and 4 to 5 years demonstrated a delayed age at AR. This finding is particularly interesting, as it suggests that higher household income background is protective against EAR despite data retrieved from literature that show otherwise.[17,26,44] Although much of the available literature does not inquire about income status based on a child's growing years, this is notable data for a small population of Filipino pediatric outpatients. It appears that mean age at AR is a modifiable clinical marker, where favorable environmental factors such as improved socioeconomic conditions can modify age at AR by delaying it, thereby potentially decreasing the suspected risk of obesity and metabolic risks in later years. Despite evidence that children from lower socioeconomic households tend to consume more processed food and sugary drinks which are linked to obesity,[1,52] the present study suggests that higher income may still delay AR. This may be explained by income-related advantages such as

access to basic household food security and food alternatives [1] may offset dietary risks.

Maternal BMI and Parental History of Obesity: The study found no significant association between the timing of AR and maternal pre-pregnancy BMI, as well as maternal or paternal history of obesity. This contrasts several studies reporting that higher parental BMI is associated with earlier AR [18,25,26,28,37] and increased risk of developing later obesity. [38,41-43] Differences in population characteristics may explain this contrast. This study's sample—Filipino children from mostly low- to middle-income families—faces distinct environmental and cultural influences on growth, such as diet, healthcare access and lifestyle. Self-reported parental obesity may have introduced recall bias, unlike other studies using clinical data. In this context, the findings highlight that parental weight history alone may not be a reliable determinant of the timing of AR. Instead, clinicians should focus on modifiable early-life factors.

Maternal Age at Birth: Maternal age at birth was not associated with the timing of AR, consistent with findings from Cissé, et al. [25] which reported no link between maternal age and AR timing. It was also found that there was no association between maternal age and obesity in adulthood. [41] However, this contrasts other studies, [28,40] which noted that older maternal age was correlated with earlier AR given that older maternal age may be associated with increased risks of pregnancy complications which may influence subsequent growth patterns and fetal metabolic programming.

Parity: Maternal parity showed no significant association with the timing of AR. [17,26,40] Giles, et al. [43] and Linares, et al. [28] reported conflicting results linking parity to early AR. It was found that multiparity is associated with increased odds of EAR and later obesity, possibly due to cumulative maternal metabolic changes affecting fetal programming. [43] Meanwhile, Linares, et al. [28] reported that lower parity was linked to earlier AR, which they attributed to maternal experience and behavioral differences. The influence of parity on early growth may be modulated by maternal nutrition, birth spacing, feeding practices and household resources—factors not fully accounted for in this study. Overall, current evidence remains mixed and parity alone may not serve as a reliable predictor of AR.

Maternal and Paternal Education: No significant association was observed between maternal or paternal education levels and the timing of AR. This finding is consistent with literature [17,26,28,39-41] which reported no substantial impact of parental education on BMI trajectories or AR timing. Some studies that specifically examined the quality and duration of parental education also found no significant association with AR timing. [28,39,41] The lack of significant association in this study may reflect the complex, multifactorial nature of early childhood growth, potentially influenced by limited variation in education levels and unmeasured social confounders such as income, employment and household structure. Overall, our findings support the broader literature suggesting that while parental education may influence general child health outcomes, it is not a strong independent determinant of AR timing.

CONCLUSION

The study concluded that, among a small population of Filipino pediatric outpatients in an urban setting, the mean age at AR is 3.2 years, which is early compared to the existing literature. Vaginal delivery and higher economic status from ages two to five are early modifiable life factors that are significantly associated with later AR. However, no significant associations were found with sex, birth weight, exclusive breastfeeding, maternal pre-pregnancy BMI, maternal and paternal history of obesity, maternal age at birth, parity and parental education. These results underscore the complexity and context-dependent nature of AR and its determinants.

Limitation of the Study

This study is limited by its small sample size, largely due to the stringent inclusion criteria and limited availability of complete and consistent longitudinal data. Therefore, gestational age and parental smoking status were excluded from the analysis. The reliance on retrospective data from medical records may introduce potential information bias and limit control over confounders. The use of self-reported maternal and socioeconomic data may also introduce potential recall bias. Finally, the findings are based on a small sample of Filipino children and

may not be generalizable to other ethnic groups and a broader Filipino population.

Recommendations

Given the early mean age at AR and its corresponding adverse metabolic risks, routine BMI monitoring in children before age three should be integrated into well-child visits. Maternal-child health strategies can promote vaginal delivery to delay AR. Lastly, obesity prevention efforts should provide greater support to lower income households. Despite its limitations, this study lays the groundwork for understanding AR among Filipino children. Future research should involve multicentric, prospective cohorts with regular, standardized growth monitoring and more detailed data on diet, behavior and environment.

Conflict of Interest

The authors share no conflicts of interest in this work. This research is not funded by any agency. The researchers are not affiliated with any organizations and persons with whom this research may benefit.

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