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Clinical and metabolic evolution study of SGA-born children from a historic cohort. Literature review and retrospective study of a historical cohort

Abstract

A newborn is considered as Small for Gestational Age (SGA) when their birth weight and/or length is below the 10th percentile. This condition, affecting 15-20% of births worldwide, is associated with higher risks of mortality and long-term complications. Fetal growth depends on a complex hormonal interaction, with factors such as maternal health, placental issues, and fetal conditions influencing outcomes. Long-term repercussions include postnatal growth, where 85-90% of SGA infants experience catch-up growth in the first two years. There is also an increased risk of precocious puberty, metabolic and cardiovascular diseases, and neurological alterations like ADHD. This study's hypothesis is that being born SGA generates a different clinical-metabolic profile. The main objectives are to provide a clinical-epidemiological description of a cohort of SGA children at 10 years and to determine the proportion with adequate weight and height upon reaching puberty.

Material and methods: A systematic literature review was performed. The study was approved by the Ethics Committee and analyzed an anonymized database of 103 SGA infants born between 2013-2015. Sixty variables were analyzed, including anthropometric, metabolic, and hormonal parameters, and comorbidities. Statistical analysis used tests like the t-test, Chi-square, and Pearson correlation, with a p-value < 0.05 considered significant.

Results: The initial cohort included 103 patients. Regarding catch-up growth, the percentage of children who did not achieve catch-up decreased with age, with only 7.61% for height and 4.44% for weight by 2 years. At 10 years old, 70.59% had normal height and 62.07% had normal weight, though 25.29% had high weight. No significant differences in height or weight were found between boys and girls. However, a significant association was found between female sex and precocious puberty, with all 7 cases occurring in girls. A positive correlation was found between weight and arm circumference at 3 months. However, no significant correlations were found between metabolic parameters (e.g., triglycerides, glucose) and abdominal circumference or weight at 2 years, nor between hormonal values (IGF-1, IGF-BP3, TSH) and catch-up growth or thyroid disorders. Early catch-up was not associated with a higher risk of precocious puberty. A high prevalence of comorbidities (57.61%) was found, the most frequent being ophthalmological disorders (30.43%), ADHD (11.95%), asthma (8.70%), and precocious puberty (7.61%).

Discussion: The catch-up growth rate in this cohort (92-95% by 2 years) aligns with literature (85-90%). The higher incidence of precocious puberty in girls supports existing knowledge, but its lack of association with early catch-up differs from some reports, possibly due to low statistical power. The proportion of overweight at 10 years (25%) was lower than in other studies, potentially due to better clinical follow-up. The inability to corroborate many metabolic and hormonal relationships may be due to the small sample size and lower overweight rate. The high comorbidity burden underscores the morbidity associated with SGA.

Conclusion: The main objectives were met. Most SGA children achieved catch-up by age 2 and had normal anthropometry at 10 years, though a quarter were overweight. A higher incidence of precocious puberty was confirmed in girls, but it was not linked to early catch-up. While a positive relationship between weight and arm circumference was confirmed, no significant links were found for various metabolic, hormonal, and puberty-related parameters. The high comorbidity prevalence highlights the need for multidisciplinary follow-up. Future research should increase sample sizes to better understand the long-term impact of being born SGA.

Keywords: SGA, catch-up growth, metabolic syndrome

Volume 12 Issue 2 - 2025

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Received: November 27, 2025 | **Published:** December 19, 2025

Introduction

Concept and epidemiology

A newborn is considered **Small for Gestational Age (SGA)** when their birth weight and/or length is below the 10th percentile (P10) for their gestational age and sex, according to population reference growth charts. A subgroup of these infants may present with **Intrauterine Growth Restriction (IUGR),** defined as a birth weight below the 3rd percentile (P3), which constitutes a pathological condition. Worldwide, between 15% and 20% of births are SGA, with

a prevalence that varies enormously between regions (from 7% in industrialized countries to 41.5% in South Asia). Being born SGA is associated with a higher risk of neonatal and infant mortality, as well as various long-term complications.

Pathophysiology and risk factors

Fetal growth[1] depends on a complex hormonal interaction, in which **Insulin-like Growth Factor I (IGF-1)** plays a crucial role. Factors that can alter this process are classified as follows:

I. **Maternal factors:** Extreme age, malnutrition, hypertension, smoking, infections, chronic diseases.

II. **Placental:** Placental insufficiency, infarcts, implantation defects.

III. **Fetal:** Chromosomal abnormalities, congenital malformations, multiple pregnancies.

Long-term repercussions

I. **Postnatal growth:** Approximately 85-90% of SGA infants experience **catch-up growth,**[2] primarily in the first 2 years of life. About 10% do not catch up and may be candidates for treatment with recombinant human growth hormone (rh -GH).

II. **Pubertal development:** There is an increased risk of **adrenarche and precocious puberty,** especially in girls with a rapid and exaggerated weight "catch-up", which may later be associated with Polycystic Ovary Syndrome (PCOS).

III. **Metabolic and cardiovascular risk:** They have an increased risk of developing insulin resistance, type 2 diabetes, metabolic syndrome, and cardiovascular diseases in adulthood.

IV. **Neurological development:** Alterations in brain development may be observed, leading to a smaller head circumference, impaired intelligence quotient (IQ), a higher incidence of Attention Deficit Hyperactivity Disorder (ADHD), and learning difficulties.

Hypothesis and objectives

Hypothesis: Being born SGA generates a different clinical-metabolic profile compared to children with appropriate weight for gestational age (AGA).

Main objectives

I. Perform a clinical-epidemiological description at 10 years of a cohort of SGA children followed from birth.

II. To determine the proportion of SGA children who, upon reaching puberty, have an adequate weight and height.

Secondary objectives: These include studying the association with precocious puberty, sex differences, the relationship between anthropometric and metabolic parameters, and the prevalence of comorbidities.

Material and methods

Bibliographic search strategy

To obtain general bibliographic information on SGA and FGR infants, an advanced search was carried out in the "PubMed" section of the National Library of Medicine website (pubmed.ncbi.nlm.nih.gov). The following search terms were entered: (((sga[Title]) OR (small for gestational age[Title])) OR (fetal growth restriction[Title])) OR (fgr[Title]), yielding 5,777 results. The search was then narrowed using the filters "last 5 years," "free full text," "systematic review," and "humans," resulting in 37 articles.

From these, the following publications were selected: *"Early-onset fetal growth restriction: A systematic review on mortality and morbidity,"*[6] *"Catch-up growth in full-term small for gestational age infants: A systematic review,"*[2] *"Breastfeeding supports growth in small for gestational age infants: A systematic review and meta-analysis,"*[13] and *"Fetal growth restriction."*[1] The remaining articles were excluded due to being overly specific.

To gather information on the prevalence of SGA infants, a search was conducted on the World Health Organization website (who.int/es) using the term "SGA." The first result, titled *"Newborn Health – World Health Organization (WHO),"* was selected. This source refers to a Guideline Development Group meeting dedicated to updating WHO recommendations on the care of preterm or low birth weight infants.[3]

For global prevalence data, a search was performed on the Nature website (nature.com). Using the "Advanced Search" option, the terms (prevalence AND world) in "that contain these terms," and (small for gestational age) in "where the title contains" were used. This search produced 344 results. Filters were applied to limit results to "Scientific Reports" (Journal), "Medical research" (Subject), and articles published within the past 5 years, resulting in 34 publications. From these, the article *"Prevalence of small for gestational age infants in 21 cities in China, 2014–2019"*[4] was selected, while the remaining articles were excluded because they focused on adult diseases related to SGA birth or maternal pregnancy-associated factors.[5–9]

To explore the consequences and clinical implications of being born SGA, information was obtained from the Spanish Society of Pediatric Endocrinology (seep.es). Through the "Publicaciones Grupos de Trabajo SEEP" section, the Clinical Practice Guideline for the follow-up of SGA children was accessed,[10] which served as a primary bibliographic source for this work. A review of this guideline's references revealed that many originated from *Anales de Pediatría* (analesdepediatria.org).[11] An advanced search using the term "small for gestational age" in the title, limited to the last 5 years, yielded three publications. From these, the articles "*Small for gestational age newborn: concept, diagnosis and neonatal characterization, follow-up and recommendations*"[5] and "*Differences in the thyroid function of small for gestational age and those of adequate weight. Is the thyroid function of small for gestational age newborns normal?"*[14] were selected. The remaining article was excluded for focusing exclusively on prenatal evaluation.

To analyze comorbidities in SGA children, another PubMed search was conducted using the query: ((sga[Title]) OR (small for gestational age[Title])) AND (comorbidities[Title]), which yielded a single result.[12]

Additional bibliographic support was provided through material from Dr. Díez's university lectures (Table 1).

Ethical considerations

The study was authorized by the Clinical Research Ethics Committee (CEIC) of the Araba University Hospital (File 2012-050). The student worked with an anonymized, coded, and non-traceable database, provided by the project directors, containing data on SGA infants born between June 2013 and May 2015.

Table 1 Summary of bibliographic search results

Search source	Search terms / filters	Total results	Filters applied	Final selected articles	Reason for exclusion of others
PubMed – General information on SGA/FGR	(((sga[Title]) OR (small for gestational age[Title])) OR (fetal growth restriction[Title])) OR (fgr[Title])	5,777	Last 5 years; Free full text; Systematic review; Humans → 37 results	*Early-onset fetal growth restriction…Catch-up growth in full-term SGA infants… Breastfeeding supports growth in SGA infants…Fetal growth restriction*	Articles were too specific
WHO Website – Prevalence	"SGA"	Not specified (general search)	Manual selection	*Newborn Health – WHO*	Others not relevant to prevalence or neonatal guidelines
Nature – Global prevalence	(prevalence AND world) in text; (small for gestational age) in title	344	Scientific Reports; Medical research; Last 5 years → 34 results	*Prevalence of small for gestational age infants in 21 cities in China, 2014–2019*	Focused on adult outcomes or maternal factors
SEEP Website – Clinical consequences	Internal guideline references	3 (from Anales de Pediatría)	Last 5 years	*Recién nacido pequeño para la edad gestacional…Diferencias en la función tiroidea…*	One article focused only on prenatal evaluation
PubMed – Comorbidities	((sga[Title]) OR (small for gestational age[Title])) AND (comorbidities[Title])	1	None	The single result obtained	—

Variables and statistical analysis

Sixty variables from 103 patients were analyzed, including anthropometric data (height, weight, mid-upper arm circumference [MAPrq], abdominal circumference [AC] at different ages), catch-up measurements, metabolic parameters (blood glucose, insulin, LDL, HDL, triglycerides [TG]), hormonal parameters (IGF-1, IGF-BP3, TSH), and comorbidities (ADHD, asthma, precocious puberty, thyroid disorders, etc.). The analysis was performed using Microsoft Excel and the *Statisfy* tool, employing statistical tests such as:

I. **Binomial Test:** For descriptive analysis of nominal variables (e.g., catch-up testing).

II. **One-sample t-test:** To compare the proportions of height and weight at age 10 with population references.

III. **Chi-square test and Fisher's exact test:** To evaluate associations between categorical variables (e.g., sex and precocious puberty).

IV. **Student's t-test for independent samples:** To compare means between two groups (e.g., height at 10 years by sex).

V. **Pearson correlation:** To evaluate relationships between quantitative variables (e.g., weight and mid-upper arm circumference). A **p-value < 0.05** was considered significant.

Results

The initial cohort consisted of 103 patients (58 men, 45 women). 11 were lost to follow-up and 2 died.

Catch-up growth

I. **Size:** The percentage of children who **did NOT** achieve the "catch-up" decreased with age: 38.75% at 3 months, 35.23% at 6 months, 30.77% at one year, 25.56% at one and a half years and **only 7.61% at 2 years.**

II. **Weight:** The evolution was similar: 35.90% at 3 months, 35.29% at 6 months, 30.68% at one year, 26.14% at one and a half years and **4.44% at 2 years.**

Anthropometric situation at 10 years old

I. **Height:** 70.59% had normal height, 14.12% short height, and 15.29% tall height. The t-test showed a significant difference with the reference population ($p < 0.001$).

II. **Weight:** 62.07% had normal weight, 12.64% had low weight, and 25.29% had high weight. This distribution was also significantly different ($p = 0.02$).

Analysis by sex

I. There were no significant differences in **height at 10 years** (p=0.198) or weight **at 10 years** (p=0.387) between boys and girls.

II. **Statistically significant** association was found between **female sex and precocious puberty** (Chi^2=9.49, p=0.002). All cases of precocious puberty[7] occurred in girls.

III. There were no differences by sex in the performance of the **weight "catch-up" at 6 months** (p=0.271).

Anthropometric and metabolic relationships

I. **Positive and significant correlation** was found between **weight at 3 months and arm circumference at 3 months** (r=0.79, p<0.001).

II. **No significant correlations were found** between:

a. Abdominal circumference at 2 years and TG values (p=0.893) or glycemia (p=0.191).

b. Weight at 2 years and insulinemia values (p=0.084), LDL (p=0.498) or HDL (p=0.289) (Figure 1).

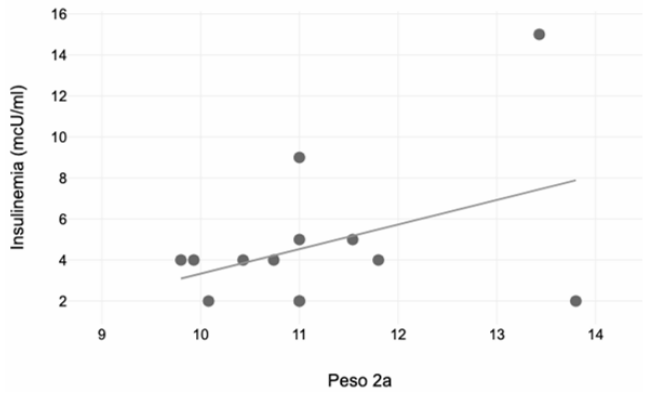

Figure 1 Representative relationship between insulin and weight SGA population. Logarithmic regression line.

Hormonal evaluation

I. **No significant differences were found** in the values of **IGF-1** (p=0.076) or **IGF-BP3** (p=0.582) between children who performed the height "catch-up" at 3 months and those who did not.

II. **No significant relationship was found** between having thyroid disorders and TSH values (p=0.076) (Figure 2,3).

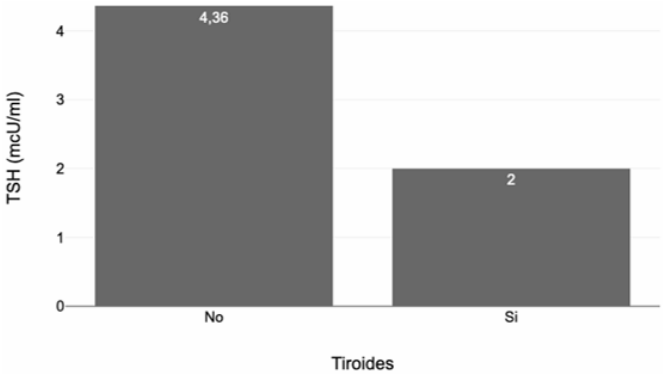

Figure 2 Differences between TSH level and pathology (No/Yes) at SGA population.

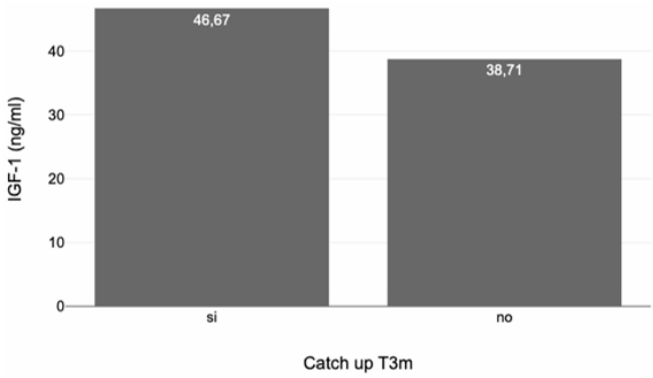

Figure 3 Differences between SGA with and without catch up at 3 months on relationship with IGF-1 levels.

Early "Catch-up" and precocious puberty

I. **No increased likelihood** of precocious puberty was observed in children who performed the "catch-up" of height (p=0.334) or weight (p=0.467) at 3 months.

Comorbidities

I. 57.61 **%** of the cohort presented at least one comorbidity.

II. The most frequent were: **ophthalmological disorders (30.43%), ADHD (11.95%), asthma (8.70%), thyroid disorders (7.61%) and precocious puberty (7.61%).**

Discussion

The results of this cohort are compared with the existing scientific literature, revealing points of agreement and disagreement.

I. **Catch-up growth:** The cohort data (92-95% "catch-up" at 2 years) are consistent with published data (85-90%), confirming that most SGA infants normalize their growth in the first years of life.

II. **Weight-arm circumference relationship:** Evidence is corroborated indicating a direct relationship between weight and fat and muscle deposits measured by arm circumference.

III. **Precocious puberty and sex: The results partially support the literature. A higher incidence of precocious puberty in girls** is confirmed, as described by several authors. However, unlike what has been reported, in this cohort girls **did not** show a significant trend toward earlier weight catch-up, and early catch-up was **not generally** associated with a higher risk of precocious puberty. The low prevalence of precocious puberty (N=7) may have limited the statistical power to detect this association.

IV. **Sex distribution at birth:** In this cohort, more SGA males were born, which contrasts with some studies that report a higher prevalence in females, possibly due to selection bias.

V. **Weight at 10 Years:** It is encouraging that only 25% of the cohort was overweight or obese at 10 years of age, a lower proportion than the 40% reported in other studies. This could be attributed to better clinical follow-up and pediatric monitoring in the studied cohort.

VI. **Metabolic and hormonal parameters:** The relationships described in the literature between waist circumference/weight and markers of metabolic syndrome (TGs, glucose, insulin, LDL, HDL) or between early catch-up and IGF-1/IGF-BP3 levels could not be corroborated. Low statistical power (small sample size, N<30 in these analyses) and the lower rate of overweight in the cohort are factors that may explain these discrepancies.

VII. **Comorbidities:** The high percentage of comorbidities (57.61%) highlights the morbidity associated with being born small for gestational age (SGA). The high frequency of ophthalmological abnormalities (30.43%) is a notable finding that warrants further investigation. The incidence of ADHD (11.95%) was higher than the general estimate for SGA infants (5–7%), supporting the known association. The lack of differences in TSH levels in children with thyroid abnormalities contradicts expectations, but again, low statistical power is a key limitation.

But, we could disccusion after lecture differents originals than Hales and Barker's[15] formulation of the *thrifty phenotype*

hypothesis establishes the conceptual cornerstone for interpreting SGA. They propose that when the fetus is exposed to undernutrition or placental insufficiency, it adapts by prioritizing vital organ development—particularly the brain—while reducing investment in metabolic tissues such as pancreatic β-cells and skeletal muscle. These adaptations, while beneficial for immediate survival, set the stage for metabolic vulnerability after birth. If the postnatal environment is nutritionally abundant, these "thrifty" adaptations become maladaptive, predisposing individuals to insulin resistance, type 2 diabetes, hypertension, and cardiovascular disease. Barker's complementary work on the fetal origins of adult disease reinforces the epidemiological evidence: populations exposed to low birth weight consistently show higher rates of non-communicable diseases later in life, supporting the idea that SGA is not a benign condition but a marker of altered developmental programming.

Gluckman and Hanson[16] extend this model through an evolutionary-developmental lens. They argue that fetal programming represents a predictive adaptive response, whereby the fetus attempts to anticipate the future environment based on intrauterine cues. In the context of SGA, this prediction favors energy-efficient physiology and fat storage. Their work also highlights the role of hormonal pathways—including cortisol, IGF-1, and leptin—in shaping organ development and metabolic set points. Importantly, they show that the mismatch between predicted and actual postnatal environments is central to the emergence of obesity and metabolic disease. This mismatch model helps explain why many SGA infants who experience rapid catch-up growth develop disproportionate adiposity ("catch-up fat") and cardiometabolic complications in childhood and adulthood.

Conradt and colleagues[17] contribute a molecular perspective by emphasizing the importance of epigenetic mechanisms—such as DNA methylation, histone modifications, and microRNA regulation—in mediating fetal programming. Their review demonstrates that environmental stressors during pregnancy, including malnutrition and hypoxia, can leave stable epigenetic marks in genes related to metabolism, stress response, and growth. These modifications may persist throughout life and transmit risk across generations. Their work underscores that SGA-related programming is not only structural or hormonal but also deeply rooted in changes in gene regulation.

Finally, Heijmans and Mill[18,19] offer further evidence of the epigenetic underpinnings of developmental programming. Their studies, including work on cohorts exposed to historical famines, illustrate how prenatal adversity produces lasting alterations in metabolic gene expression. They show that individuals with lower birth weight often exhibit differential methylation patterns in genes involved in glucose metabolism and cardiovascular regulation. These findings provide a mechanistic bridge between the thrifty phenotype hypothesis and observed adult disease patterns, reinforcing the notion that SGA represents a biological imprint of early-life adaptation.

Taken together, these last five articles converge on the idea that SGA[15–19] is best understood through the framework of fetal programming: a process in which the fetus adapts to suboptimal conditions by adjusting growth, physiology, and gene expression. Although these adaptations promote short-term survival, they also increase vulnerability to metabolic and cardiovascular diseases when the postnatal environment does not match fetal expectations. The integration of epidemiological, physiological, evolutionary, and epigenetic evidence offers a robust explanation for the lifelong implications of being born small for gestational age.

The failure to achieve adequate catch-up growth in children born small for gestational age (SGA)[20,21] can be partly explained by a complex interplay between growth hormone (GH) resistance, reduced insulin-like growth factor 1 (IGF-1) bioavailability, and early alterations in insulin sensitivity. These endocrine disruptions reflect deeper mechanisms of fetal programming, in which intrauterine undernutrition leads to long-lasting changes in growth-regulatory pathways.

Finally, at the neonatal and infant period, many SGA infants show *relative GH resistance*. Although GH concentrations are often normal or elevated, hepatic responsiveness to GH is reduced. This impairment is linked to decreased expression of GH receptors and downregulation of post-receptor signaling elements such as JAK2 and STAT5. As a consequence, the liver produces lower levels of IGF-1 and IGFBP-3, leading to insufficient anabolic signaling.[22]

Low IGF-1 levels in early life are critical because IGF-1 is the primary mediator of postnatal linear growth. Inadequate IGF-1 action compromises chondrocyte proliferation in the growth plate, limiting longitudinal bone growth and contributing to *impaired early catch-up growth*. At the same time, reduced IGF-1 bioactivity may reflect an adaptive mechanism resulting from fetal programming—prioritizing energy conservation over growth during periods of resource scarcity.[23]

Insulin is a key growth-promoting hormone during fetal and early postnatal life. Insulin resistance, which is commonly observed in SGA infants—even before the onset of adiposity—further worsens defective catch-up growth. Early insulin resistance reduces glucose uptake at the growth plate and affects both chondrocyte activity and hepatic IGF-1 production.

Because insulin and IGF-1 signaling share post-receptor pathways (PI3K–Akt), resistance to one often reinforces resistance to the other. Thus, early insulin resistance amplifies IGF-1 resistance, creating a hormonal environment that supports survival but restricts somatic growth.

In later childhood and adolescence, some SGA individuals continue to experience GH and IGF-1 inefficiency, particularly those who fail to exhibit spontaneous catch-up. Chronic GH resistance and low IGF-1 levels reflect structural and epigenetic programming of the GH receptor and IGF-1 gene regulatory regions. Importantly, insulin resistance often worsens with age—especially in individuals who undergo rapid weight gain or develop "catch-up fat," a disproportionate accumulation of adipose tissue.[20–22]

This evolving insulin resistance contributes to a physiological paradox:

I. **IGF-1 remains low → growth remains suboptimal**, while

II. **Insulin levels rise → promoting adiposity instead of linear growth**.

This divergence reflects the failure of the GH–IGF-1 axis to normalize despite increased metabolic demand. Ultimately, the combination of GH resistance, low IGF-1, and ongoing insulin resistance perpetuates a state of *growth failure with metabolic risk*, characteristic of a thrifty phenotype.[24]

Implications for growth and long-term outcomes

Together, these mechanisms explain why a subset of SGA children fail to achieve appropriate catch-up growth despite adequate nutrition. The endocrine environment shaped by fetal programming favors energy storage rather than growth, maintaining a phenotype suited for scarcity but maladaptive in nutrient-rich environments. Over time, the persistent GH–IGF-1 dysfunction and insulin resistance not only

impair growth but also increase susceptibility to metabolic syndrome, type 2 diabetes, and cardiovascular disease.

Conclusion

Following the cohort analysis, the following conclusions were reached:

I. **Main objectives** were met: the cohort was described clinically and epidemiologically and it was determined that at 10 years, the majority have normal height (70.59%) and weight (62.07%), with 25.29% being overweight.

II. It is confirmed that between **85-90% of PEG children achieve the "catch-up"** in weight and height by age 2.

III. There is a **higher incidence of precocious puberty in SGA girls,** but this was not associated with an early "catch-up" of weight or height.

IV. **No differences in height or weight were found** at age 10 between boys and girls.

V. **Positive relationship** between **weight and arm circumference** is confirmed at 3 months.

VI. **No significant relationships were found** between:

a. Metabolic parameters (TGs, glycemia, insulinemia, LDL, HDL) and abdominal perimeter or weight at 2 years.

b. TSH values and the presence of thyroid abnormalities.

c. IGF-1/IGF-BP3 values and the realization of an early "catch-up".

d. The realization of an early "catch-up" and the development of precocious puberty.

I. **High prevalence of comorbidities** was identified (57.61%), the most frequent being ophthalmological disorders, ADHD and asthma.

II. In short, this study supports some established findings in the literature, but also reveals the need to increase the sample size and standardize variables in future research to more accurately understand the long-term impact of being born SGA.

Acknowledgments

None.

Conflicts of interest

The author declares that there are no conflicts of interest.

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