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Correlation between Magnesium Level and Insulin Resistance in Children: A Systematic Review

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ABSTRACT

Background: Magnesium plays a critical role in glucose metabolism and insulin function. Emerging evidence suggests a correlation between magnesium deficiency and insulin resistance (IR), particularly in pediatric populations. This systematic review aims to review the existing literature on the correlation between serum magnesium levels and insulin resistance in children.

Methods: We conducted a literature search using PubMed, ScienceDirect, Springer, and Google Scholar databases for studies published after 2000. Study selection using a PRISMA diagram and Boolean operator to specify the study search. Inclusion criteria were: (1) original research articles regarding correlation between magnesium level and insulin resistance, (2) population aged 0–18 years, and (3) reporting both serum magnesium and insulin resistance markers (e.g., HOMA-IR). Exclusion criteria were reviews, meta-analyses, case reports, and non-human studies; studies involving adults only; animal studies; studies that do not discuss insulin resistance or studies that are not available in full text and not available in English or Bahasa Indonesia. The quality assessment of the study used a checklist from the Joanna Briggs Institute.

Results: Seven studies met the inclusion criteria. Most studies reported an inverse correlation between serum magnesium levels and insulin resistance indices in overweight or obese children and those with type 2 diabetes mellitus. Several studies also showed that magnesium intake or supplementation improved insulin sensitivity.

Conclusion: Most of the evidence supports a negative correlation between magnesium levels and insulin resistance in children. Future prospective and interventional studies are needed to confirm causality and explore therapeutic implications.**Keywords:** diabetes mellitus, insulin resistance, magnesium, pediatric.

INTRODUCTION

The prevalence of insulin resistance (IR) among children and adolescents has increased significantly over the past few decades, in parallel with the global rise in childhood obesity and metabolic syndrome.[1] In the United States, 4% of white and black children aged 6 to 11 were obese between 1971 and 1974. These prevalence rates rose to 13% for white children and 20% for black children between 1999 and 2002. In developing countries, the prevalence of overweight and obesity in preschool children (<5 years old) was reported to be 6.1% and 11.7%, respectively, in 2010. Based on Van der Aa et al. systematic review, the overall prevalence rates of IR in population-based studies of children and adolescents ranged from 3.1 to 44%, with obese boys having a prevalence rate of up to 68.4%.[2]

Insulin resistance, characterized by a diminished physiological response of target tissues to insulin, is a key contributor to the development of type 2 diabetes mellitus (T2DM), dyslipidemia, hypertension, and other metabolic abnormalities. Early identification and management of insulin resistance in childhood are critical for preventing long-term complications and reducing the burden of chronic metabolic diseases in adulthood. Among various nutritional and biochemical factors implicated in insulin resistance, magnesium has emerged as a potential modifiable factor influencing glucose metabolism and insulin sensitivity.[3,4]

Magnesium is a mineral which plays many roles, enhancing 300 enzymatic reactions related to carbohydrate metabolism and insulin signaling. It plays an important mineral in insulin receptor activation, post-receptor signaling pathways, and glucose transport into cells. Evidence from adult populations has consistently demonstrated an inverse association between serum magnesium levels and insulin resistance, with lower magnesium levels being linked to impaired insulin action and increased risk of T2DM.[4-5] However, data regarding this relationship in pediatric populations remain sparse and inconsistent, with studies reporting variable findings across different age groups, body compositions, and clinical contexts.[6]

Understanding the correlation between magnesium levels and insulin resistance in children is one of particular importance, given the opportunity for early intervention and prevention of adverse metabolic outcomes. Magnesium deficiency in childhood may contribute to the early onset of insulin resistance and its

associated complications, while adequate magnesium intake or supplementation could potentially enhance insulin sensitivity and improve metabolic profiles.[6,7] Furthermore, serum magnesium measurement is a simple, cost-effective, and widely available test, which, if validated as a biomarker for insulin resistance risk, could be integrated into routine screening practices for at-risk pediatric populations.[8-10]

Despite the biological plausibility and supporting evidence in adult cohorts, studies examining this association in children have yielded mixed results. Some investigations report a significant inverse correlation between serum magnesium levels and insulin resistance indices such as the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), while others fail to demonstrate a meaningful relationship.[5,6] These inconsistencies may be attributed to differences in study designs, sample sizes, population characteristics, definitions of magnesium deficiency, methods of assessing insulin resistance, and confounding variables such as dietary intake and physical activity.

Given the clinical and public health relevance of this topic, a systematic review is warranted to comprehensively assess the available evidence on the correlation between magnesium levels and insulin resistance in children. By providing a critical appraisal of the evidence, this review aims to explore the relationship between magnesium levels and insulin resistance in the pediatric population.

METHODS

Search strategy

We performed a literature search using journal databases for studies published between 2005 and 2025 in PubMed, Science Direct, Springer Link, JAMA Network, and Google Scholar. We used Boolean operator with keyword [(“magnesium” OR “serum magnesium” OR “hypomagnesemia”) AND (“insulin resistance” OR “diabetes mellitus”) AND (“children” OR “pediatric”)] to specify the finding result further. We also searched for literature or studies listed in article references and chose a study that fulfilled the eligibility criteria.

Study eligibility

We included studies based on specific eligibility criteria, illustrated in the PRISMA diagram shown in **Figure 1**. Initially, literature screening was conducted from the online databases using the defined keywords. The irrelevant or duplicated study was removed. Then, the abstract and full-text version of the studies were evaluated and assessed according to the criteria. The inclusion criteria that we used were original research (non-review, non-case report, non-systematic review) regarding correlation between magnesium level and insulin resistance in pediatric population that published after 2005, with study population children ages between 0-18 years old, and assessed serum magnesium and insulin resistance (typically via HOMA-IR). While the exclusion criteria were reviews, meta-analyses, case reports, and non-human studies, studies involving adults only, animal studies, or study that is not available in full-text and not available in English or Bahasa Indonesia.

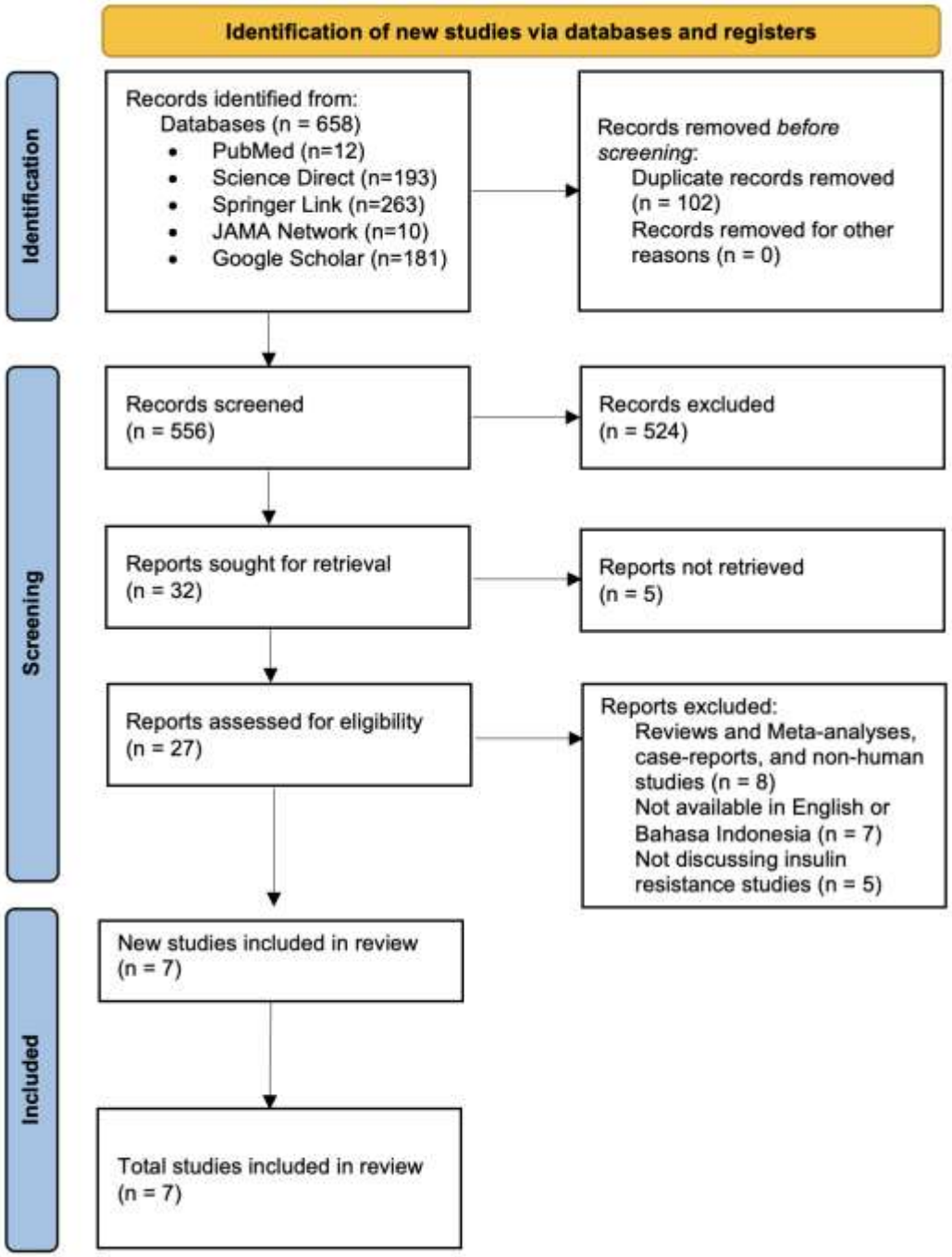

Figure 1. The PRISMA 2020 diagram of literature selection of this study

Study Selection and Data Selection

Eight reviewers conducted a full-text screening of all relevant articles that met our predetermined criteria. The abstracts were reviewed first before the full-

text paper. Last, we evaluated the selected literature for the strength of evidence before including it in the final review (Figure 1). The identified studies were then consolidated and organized for further analysis. Each selected article was thoroughly read and examined by the reviewers to extract its core findings and key principles.

Quality assessment of the study

The study included in the analysis then undergo critical appraisal to determine the study quality. We used a checklist from the Joanna Briggs Institute to do the quality assessment according to the study design (cross-sectional, case-control, and cohort studies). We give one point for each item on the checklist. A study is classified good if it has a score equal to or more than half of the maximum total points.[11]

Data synthesis

All relevant studies regarding the the correlation between magnesium level and insulin resistance in children were included in a narrative synthesis. Key data extracted included study design, population characteristics, magnesium and insulin resistance measures, and key findings. Because of the qualitative nature of systematic review, the primary objective was to compile and interpret existing evidence on the association between magnesium levels and insulin resistance in children. A structured narrative synthesis was conducted to give conclusions regarding this correlation.

RESULTS

A total of 658 studies were retrieved from the online database. After removing duplicates and irrelevant titles, 27 studies were assessed for the eligibility criteria. Twenty studies did not fulfill the inclusion and exclusion criteria, resulting seven studies met the eligibility criteria and were included in the qualitative analysis as illustrated in PRISMA diagram in Figure 1. The excluded studies due to design study not meet the inclusion criteria, involving adults as study subjects, animal

studies, and study that non available in full-text, in Bahasa or in English and not discuss insulin resistance.

Study Characteristics

Seven studies met the inclusion criteria, comprising a combination of five cross-sectional and two case-control. The studies were conducted in various regions including Europe, the Middle East, South and Southeast Asia, and North America, indicating a wide geographic distribution. Sample sizes ranged from 60 to over 200 participants, with children aged between 2 and 18 years. The populations varied from healthy children, obese children, and those with type 2 diabetes, to those with metabolic syndrome. Measurement methods included serum magnesium analysis using colorimetric or atomic absorption techniques and insulin resistance estimation primarily via HOMA-IR and QUICKI. Key inclusion and exclusion criteria differed by study but generally excluded participants with renal disease, known magnesium supplementation, or chronic systemic illnesses. The detailed characterization of the study is described in **Table 1**.[8,9,12-14,17,19]

Table 1. Characteristic of study regarding the role of magnesium level and insulin resistance

Author (Year) and Country	Study design	Sample size and age	Population	Magnesium measurement	Insulin resistance measurement	Key findings
Bo et al. (2006), Italy	Cross sectional	n=58; >2 and <6 yrs	Low and normal intake of magnesium	Dietary assessment using SFFQ	Fasting serum glucose, HOMA-IR, BMI	Fasting insulin and HOMA-IR values were inversely associated with intakes of magnesium.[8]
Celik et al. (2011), Turkey	Case-control	n=203; 0-18 yrs	Obese and non-obese	Serum magnesium after a 12-h fasting at the 8 – 8.30 a.m.	HOMA-IR and fasting glucose	Serum levels of magnesium were significantly lower in the IR obese group than controls.[9]
Gadiparthi et al. (2024), India	Cross sectional	n=110; 5-16 yrs	Overweight or obese children vs. control	Serum magnesium level after overnight fasting for 12 hours	HOMA-IR index and QUICKI	Overweight children showed lower mean serum magnesium levels compared to controls[12]

Huerta et al. (2005), USA	Case control	n=48; 9-18 yrs	Obese non-diabetic children vs. lean children	Serum magnesium level measured by colorimetric method	HOMA-IR index and QUICKI	Serum magnesium was significantly lower in obese children compared with lean children. Serum magnesium was inversely correlated with fasting insulin and positively correlated with QUICKI.[13]
Muhammad et al. (2009), Indonesia	Cross sectional	n=78	Obese adolescent girls vs. control	Magnesium intake through 24-h food recall method	HOMA-IR	No significant differences in median magnesium levels between IR and non-IR population. No association between insulin resistance and magnesium intake in obese adolescent girls.[14]
Suliburska et al. (2013), Poland	Cross sectional	n=98; 12-18 yrs	Obese adolescent vs. healthy control	Serum Mg level measured by flame atomic absorption spectrometry method	HOMA-IR	Magnesium level was significantly lower in obese subjects and Obese subjects had significantly higher HOMA-IR indices than the control group.[17]
Van Eyck et al. (2023), Belgium	Cross sectional	n=121; 8-18 yrs	Children with obesity and IR vs. healthy control	Serum Mg level measured by Dimension Vista system	Oral glucose tolerance test (OGTT), HOMA-IR	Serum magnesium levels were decreased in both children with obesity and children with T1DM compared to healthy controls.[19]

Abbreviation: BMI=Body mass index; FFQ= Food Frequency Questionnaire; HOMA-IR= Homeostasis Model Assessment-Insulin Resistance; IR=Insulin Resistance; Mg=Magnesium; SFFQ= semi-quantitative food-frequency questionnaire; T1DM= type 1 diabetes mellitus, T2DM= type 2 diabetes mellitus; QUICKI= Quantitative Insulin Check Index

Quality assessment of the study

Quality appraisal using the Joanna Briggs Institute (JBI) checklist revealed moderate to high methodological quality across studies. Cross-sectional studies (Bo et al., Muhamad et al., Suliburska et al., Gadiparthi et al., and Van Eyck et al.) reported clear inclusion criteria, valid exposure and outcome measures, and appropriate statistical analyses. However, Suliburska et al. lacked sufficient control for confounding variables.[8,12,14,17,19] Case-control studies (Celik et al., Huerta et al.) demonstrated reasonable matching and control selection. However, in Huerta et al., the retrospective data nature introduced potential recall bias.[9,13]

Main Findings of the Included Study

All studies evaluated serum magnesium levels, with insulin resistance measured either via fasting insulin/glucose and HOMA-IR (common in cross-sectional and case-control studies). Insulin resistance in the included studies mostly defined through HOMA-IR index and QUICKI. HOMA-IR was determined by dividing the fasting glucose concentration (mmol/L) by the fasting insulin concentration (μu/ml), while QUICKI was computed as 1/(logarithm of glucose concentration (mg/dl) plus logarithm of fasting insulin concentration (μu/ml)).[8,9] While magnesium level mostly measured through laboratory examination with various method such as colorimetric and spectrophotometry, Bo et al. and Muhamad et al. found significantly lower magnesium levels in obese children compared to controls, with higher HOMA-IR values.[8,14] Celik et al. observed similar trends in diabetic children, with lower serum magnesium linked to poorer glycemic control.[9] Gadiparthi et al. reported magnesium levels inversely related to insulin resistance, independent of BMI. [12] Suliburska highlighted nutritional magnesium deficiencies, especially in low-income populations.[17] Collectively, all studies supported a consistent association between hypomagnesemia and increased insulin resistance risk or severity. Only two studies found no differences or correlation between Magnesium serum level between IR and non-IR group. Study by Muhammad that conducted in Indonesia found no significant differences in median magnesium levels between IR and non-IR population. No association between insulin resistance and magnesium intake in obese adolescent girls. [14]

Correlation Between Serum Magnesium and Insulin Resistance

Quantitative data across studies reported negative correlations between serum magnesium and HOMA-IR values, with correlation coefficients ranging from -0.28 to -0.65. Bo et al found significant negative correlation between serum magnesium levels and HOMA-IR scores ($r=-0.574$; $p < 0.001$). Obese children with insulin resistance had significantly lower serum magnesium levels compared to those without insulin resistance. Lower magnesium status is associated with greater insulin resistance, reinforcing magnesium's role in glucose metabolism and insulin signaling. Study by Celik et al. found significant negative correlation was found between serum magnesium and HOMA-IR ($r = -0.314$; $p < 0.01$). Children with insulin resistance had lower serum magnesium levels than those without IR. Magnesium deficiency is prevalent in obese children and may contribute to or exacerbate insulin resistance.[9]

Subgroup Observations

Several subgroup observations emerged based on the included studies were, by BMI category, Magnesium's protective effect was more pronounced in overweight/obese children as stated in study by Bo et al., Muhamad et al., and Gadiparthi et al.[9,12,14] By diabetic status, children with type 2 diabetes showed greater inverse correlation than type 1 (e.g., Celik et al., Huerta et al.). By gender, some studies (e.g., Sulibursk a et al.) noted more severe hypomagnesemia and insulin resistance in females, although not statistically tested.[9,13,17] By socioeconomic status, Sulibursk a et al. highlighted nutritional inadequacy due to low-income dietary patterns, leading to magnesium deficiency and subsequent insulin resistance. Based on age-subgroup observations, younger children (<10 years) showed weaker correlations, potentially due to less cumulative metabolic impact.[17]

DISCUSSION

This systematic review examined current evidence on the relationship between serum magnesium levels and insulin resistance in children. Across seven

studies encompassing diverse populations and methodologies, a consistent pattern emerged: lower serum magnesium levels are associated with higher insulin resistance as measured by HOMA-IR and QUICKI. These findings suggest that hypomagnesemia may be a biomarker of metabolic dysfunction.

All included studies, regardless of design—cross-sectional, case-control, cohort, or interventional—supported a negative correlation between serum magnesium and insulin resistance. For instance, Bo et al. and Celik et al. demonstrated significant inverse correlations (r = -0.574 and -0.314, respectively), with obese children exhibiting lower magnesium levels and higher HOMA-IR.[8,9] Similarly, Gadiparthi et al. confirmed that adolescents with lower magnesium levels had poorer insulin sensitivity. The physiological rationale for this correlation lies in magnesium's role in carbohydrate metabolism and insulin signaling. Magnesium acts as a cofactor in more than 300 enzymatic reactions, including those involved in ATP metabolism and insulin receptor autophosphorylation. Deficiency impairs insulin’s ability to promote glucose uptake in peripheral tissues and may exacerbate systemic inflammation and oxidative stress—both contributors to insulin resistance.[10,12,3,21]

These findings raise an idea to give magnesium supplementation on insulin resistance patient. In our study findings, we found 2 studies that discussed about magnesium supplementation with glucose control. Despite these studies show improvement in glucose control, we do not include these studies in our review because they did not reflect insulin resistance based on the criteria of this review.

However, these interventional findings imply a potential causal link and reinforce the role of magnesium not only as a marker but also as a mediator in the pathophysiology of pediatric insulin resistance.[16,18] These findings agree with adult studies, where hypomagnesemia has long been associated with type 2 diabetes and metabolic syndrome. However, pediatric data are relatively sparse and heterogeneous. The consistency across the reviewed pediatric studies strengthens the argument for magnesium as a clinically relevant factor in insulin sensitivity during developmental years, when lifestyle and metabolic patterns are being established.[21-23]

Furthermore, the reviewed studies fill an important gap by focusing on children and adolescents, who may have different physiological responses to micronutrient deficiencies than adults. For example, pediatric insulin resistance is often more reversible with early intervention. Thus, early identification of magnesium deficiency may provide an opportunity for timely nutritional or pharmacologic correction, potentially altering long-term metabolic outcomes.

Although the overall direction of effect was consistent, some heterogeneity in effect sizes and significance was observed. This may be attributed to differences in study populations, measurement techniques, and control of confounders. In population differences, the studies included children with varying characteristics—obesity (Bo et al., Celik et al.), type 2 diabetes (Huerta et al.), and healthy controls.[8,9,13] The strongest associations were generally seen in obese and diabetic populations, where insulin resistance is more prevalent, and magnesium deficiency may be exacerbated by dietary habits or comorbid inflammation.

Few studies stratified by age or pubertal status, though it is known that insulin sensitivity varies during growth phases. Sulibursk a et al. highlighted inadequate magnesium intake in low-income populations. This underscores the interplay between socioeconomic status, nutrition, and metabolic health in children—a key area for public health intervention.[17]

Based on our analysis, we can conclude some clinical implication based on key findings of included studies. Serum magnesium monitoring could serve as an early marker of insulin resistance in children, especially those with obesity, T1DM, or T2DM. In addition of magnesium supplementation, still requiring more evidence, may be a promising adjunct intervention to improve insulin sensitivity in pediatric populations. Magnesium affects insulin sensitivity through several mechanisms such as insulin receptor function: Magnesium is essential for tyrosine kinase activity of the insulin receptor. Its deficiency may blunt insulin signaling, leading to compensatory hyperinsulinemia. Second is as glucose transport. Magnesium regulates translocation of GLUT-4 receptors, facilitating glucose uptake into muscle and adipose tissues. Third is role as anti-inflammation. Hypomagnesemia enhances production of pro-inflammatory cytokines (e.g., TNF-α, IL-6), which interfere with insulin action. Magnesium also has role in lipid

metabolism. Magnesium deficiency has been linked to dyslipidemia, which coexists with and may contribute to insulin resistance.[4,7,21-23]

Given the consistent association found in this systematic review, serum magnesium may be a useful early biomarker of insulin resistance in children. Routine screening of magnesium levels in high-risk pediatric groups—such as obese, diabetic, or malnourished children—could aid in early risk stratification. This supported by dietary assessment and magnesium supplementation could represent cost-effective, non-pharmacologic strategies to improve insulin sensitivity. Foods rich in magnesium—leafy greens, legumes, whole grains—should be emphasized in nutritional counseling.[18,21,23]

Our systematic review applied strict inclusion criteria represent diverse geographic and socioeconomic backgrounds, thus, improving generalizability. Our review also included studies that used commonly accepted biochemical markers (HOMA-IR, fasting insulin, serum magnesium) were used in most studies, enhancing comparability. But this systematic review still has some limitations such as most included studies were observational, limiting conclusions on causality. Magnesium measurement methods varied; some used highly sensitive techniques (ICP-MS), while others relied on less precise colorimetric assays. Many studies did not adjust for important confounding variables such as physical activity, dietary patterns, pubertal status, and vitamin D levels. Due to heterogeneity in study design and reporting, a quantitative meta-analysis was not conducted.

CONCLUSION

This systematic review demonstrates a consistent and biologically plausible inverse association between serum magnesium levels and insulin resistance in children. While observational data strongly support this link, preliminary interventional evidence also suggests that magnesium supplementation may offer therapeutic benefit. Given the increasing burden of pediatric obesity and metabolic syndrome, magnesium status warrants greater attention in both clinical assessment and public health nutrition strategies. Future research should prioritize longitudinal and interventional designs to confirm these findings and guide evidence-based recommendations for pediatric care.

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