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Maternal Diabetes and Risk of Childhood Cancer in Offspring: An Updated Systematic Review and Meta-Analysis of Observational Studies

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Abstract: ***Background:*** ***The association between maternal diabetes mellitus and childhood cancer risk in offspring remains controversial. Previous meta-analyses have been limited by the exclusion of relevant studies and insufficient stratification by diabetes type. We conducted an updated systematic review and meta-analysis to comprehensively evaluate this association.*** ***Methods:*** ***We systematically searched PubMed, Embase, and Web of Science databases from inception through January 2026. Studies investigating the association between maternal diabetes (preexisting diabetes mellitus [PDM] or gestational diabetes mellitus [GDM]) and childhood cancer risk were included. Pooled odds ratios (ORs) with 95% confidence intervals (CIs) were calculated using random-effects or fixed-effects models based on heterogeneity assessment. Publication bias was evaluated using funnel plots and Egger's test.*** ***Results:*** ***Twenty-six studies involving over 2 million participants were included. Maternal diabetes was significantly associated with increased risk of overall childhood cancer (OR = 1.29, 95% CI: 1.12–1.49, p = 0.0005; I^2 = 70.2%), childhood leukemia (OR = 1.33, 95% CI: 1.17–1.51, p < 0.0001; I^2 = 0.0%), and acute lymphoblastic leukemia (OR = 1.44, 95% CI: 1.27–1.64, p < 0.0001; I^2 = 0.0%). Subgroup analyses revealed that PDM was associated with higher risk estimates compared to GDM across all outcomes. Funnel plots indicated minimal publication bias for leukemia and ALL analyses.*** ***Conclusions:*** ***Maternal diabetes is associated with a significantly elevated risk of childhood cancer, particularly leukemia and acute lymphoblastic leukemia, in offspring. Preexisting diabetes confers greater risk than gestational diabetes. These findings underscore the importance of optimal glycemic control during pregnancy and highlight potential long-term consequences of the intrauterine diabetic environment on offspring health.***

Keywords: Maternal diabetes, Gestational diabetes mellitus, Preexisting diabetes, Childhood cancer, Leukemia, Acute lymphoblastic leukemia, Meta-analysis**.**

1. Introduction

Childhood cancer, though rare, remains a leading cause of disease-related mortality in children, necessitating the identification of modifiable maternal risk factors [1, 2]. Concurrently, the prevalence of diabetes mellitus (DM) among women of reproductive age is increasing globally [3]. The "fetal origins of cancer" hypothesis suggests that the intrauterine environment plays a critical role in early carcinogenesis [4]. Biologically, maternal hyperglycemia may expose the fetus to hyperinsulinemia and elevated levels of insulin-like growth factors (IGFs), potentially altering fetal programming and promoting cellular proliferation [5-7]. While the association between diabetes and malignancies in adults is well-established, the impact of maternal diabetes — encompassing type 1, type 2, and gestational diabetes mellitus (GDM)—on the risk of childhood malignancies in offspring remains a subject of ongoing debate7.

Previous attempts to synthesize the evidence have yielded inconsistent results. A systematic review reported an overall increased risk of childhood cancer, particularly acute lymphoblastic leukemia, among offspring of mothers with diabetes [8]. However, that analysis was limited by the inadvertent exclusion of several eligible studies published prior to its search cutoff, potentially compromising the precision of its estimates [9-11]. Furthermore, recent large-scale population-based studies published in 2022, notably involving cohorts from Denmark and Taiwan, have challenged earlier findings [12]. These new data suggest that while overall cancer risk may not be elevated for all diabetic pregnancies, specific associations exist between distinct diabetes subtypes and rare tumors, such as maternal type 1 diabetes with glioma and type 2 diabetes with hepatoblastoma [13, 14]. These granular distinctions were not fully captured in previous syntheses.

Given the omission of relevant literature in the previous meta-analysis and the emergence of recent, large-scale evidence highlighting subtype-specific risks, an updated and comprehensive evaluation is warranted. The present study aims to revisit this association by performing a systematic review and meta-analysis that incorporates both the previously overlooked studies and the most recent data. Unlike prior reviews, this study places specific emphasis on stratifying risk by maternal diabetes type (pre-existing type 1/type 2 versus GDM) and examining a broader spectrum of site-specific childhood cancers. By addressing these methodological gaps, we seek to provide more robust evidence to inform clinical counseling and clarify potential etiological mechanisms.

2. Methods

2.1 Search Strategy and Study Selection

We performed a comprehensive literature search, including the PubMed, Embase and Web of science databases from their inception to January 30, 2026, to identify studies that investigated the association between maternal diabetes and risk of childhood cancer in the offspring. The literature search strategy included the following key terms: "maternal

diabetes", "gestational diabetes mellitus", "pre-gestational diabetes", "pre-pregnancy diabetes", "pre-existing diabetes" (PDM), "childhood cancer", "childhood malignancies", "pediatric neoplasms" and "pediatric cancers". Besides, we manually searched for references cited in the original study and the review.

Eligible studies should meet the following inclusive criteria: (1) studies were cohort or case–control studies; (2) the exposure of interest was maternal diabetes including PDM and GDM, and the outcome of interest was the diagnosis of childhood malignancy in the offspring; (3) the sample size with maternal diabetes was available; (4) reported the effect estimates (ESs) (odds ratio (OR), relative risk (RR), standard incidence ratio (SIR), or hazard ratio (HR)) and their corresponding 95% confidence intervals (CIs) or gave sufficient data to compute them.

2.2 Data Extraction and Quality Assessment

The following data were extracted independently from the included literature by two reviewers: first author, year of publication, country, study type (cohort or case–control study), study period, type of maternal diabetes (diabetes (any), PDM and GDM), type of childhood cancer, number of cases with maternal diabetes, number of cancer cases, and ascertainment of maternal diabetes and cancer, adjusted ESs and corresponding 95% CIs, as well as confounders for adjustment.

Newcastle–Ottawa Scale (NOS) guidelines include 3 quality parameters: four items for selection, two items for comparability and three items for outcomes, to assess the quality of studies included in our meta-analysis [15]. Studies scoring 7–10 were identified as high-quality, those with scores of 3–6 were considered moderate quality, and the others were of low quality.

2.3 Statistical Analysis

The summary ORs and corresponding 95% CIs were calculated to examine the effect of maternal diabetes on the incidence of childhood cancer in the offspring. Since the risk of childhood cancer in the general population was very low, all ESs were interpreted as OR for simplicity. A fixed-effect model was used for outcomes with low heterogeneity ($I^2 < 25\%$) among included studies; otherwise, a random-effect model was used ($I^2 > 25\%$). Cochran Q and I2 statistics were used to evaluate statistical heterogeneity between the included studies. I2 values of < 25%, 25–75% and > 75% were defined as low, medium and high heterogeneity, respectively [16]. To investigate the sources of heterogeneity, we conducted subgroup analyses and meta-regression analyses according to maternal diabetes types (diabetes (any), PDM and GDM), study design (cohort and case–control studies), study location (Europe and other regions), number of cancer cases (≥ 3000 and < 3000), cancer diagnosis (medical records and cancer registry) and whether adjusted for birth weight and birth order (Yes and No). Sensitivity analyses were performed to evaluate the robustness of the results of our meta-analysis. Funnel plots were used to assess publication bias. Furthermore, Egger's linear regression tests and Begg's adjusted rank correlation test were conducted and a P value of < 0.05 indicated potential publication bias. All statistical analyses were performed using Stata, version 14.0 (Stata Corp, College Station, Texas).

3. Results

3.1 Literature Search and Study Selection

The detailed process of study identification and selection is illustrated in the PRISMA flow diagram (Figure 1). Our initial comprehensive search of PubMed, Embase, and Web of Science databases yielded a total of 1,083 citations. After removing 101 duplicates, 982 unique records remained for the initial screening phase. Based on a review of titles and abstracts, 865 records were excluded as they clearly did not meet the inclusion criteria. Consequently, 117 articles were retrieved for full-text evaluation. After a detailed assessment of eligibility, 26 studies were ultimately included in the present meta-analysis [9-12, 17-38].

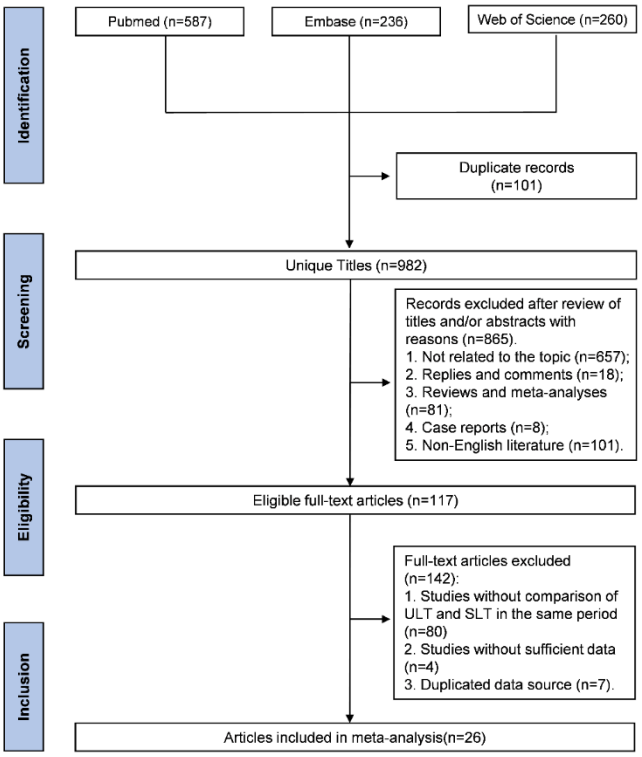

Figure 1: PRISMA flow diagram for identification of relevant articles for the meta-analysis

3.2 Study Characteristics and Quality Assessment

The baseline characteristics of the 26 identified studies are summarized. In terms of study design, the majority were case–control studies (n=20) [9-11, 17, 18, 20-26, 29-32, 36, 38], with the remaining six utilizing a cohort design [17-29, 31-37]. regarding the outcome of interest, 18 studies focused exclusively on a single cancer site [9-11, 17, 18, 20-26, 28, 29, 31, 32, 34-36], whereas eight assessed the overall risk of childhood malignancy [12, 19, 21, 27, 30, 33, 37, 38]; among these, four also provided granular data on multiple specific cancer subtypes [12, 30, 33, 38]. Regarding the exposure classification, ten studies reported aggregate data on maternal diabetes without stratifying by specific subtypes (e.g., type 1, type 2, or GDM).

Methodologically, the ascertainment of maternal diabetes was primarily based on objective medical birth registries or hospital records (*n*=19) [10, 12, 17, 19, 21-27, 29-31, 33-35, 37, 38]. However, seven studies relied on self-reported data obtained via questionnaires or telephone interviews [9, 11, 18, 20, 28, 32, 36]. In contrast, childhood cancer diagnoses were universally confirmed through cancer registries or medical records across all included studies.

Most studies (*n*=24) provided effect estimates adjusted for potential confounders, whereas two studies presented raw data from which unadjusted ORs were likely calculated [31, 36]. The most frequently adjusted covariates included maternal age (*n*=16), birth order (*n*=6), birth weight (*n*=5), and maternal smoking status (*n*=4). Quality assessment using the Newcastle–Ottawa Scale (NOS) is detailed. Seven studies were classified as high quality, while the remaining 19 were designated as moderate quality.

3.3 Maternal Diabetes and Overall Childhood Cancer Risk

A total of eight studies were included in the meta-analysis examining the association between maternal diabetes and overall childhood cancer risk in offspring [12, 19, 21, 27, 30, 33, 37, 38]. As shown in Figure 2, the pooled analysis demonstrated a statistically significant association between maternal diabetes and increased risk of childhood cancers (OR = 1.29, 95% CI: 1.12–1.49, p = 0.0005). Substantial heterogeneity was observed across studies (I^2 = 70.2%, τ^2 = 0.032, p = 0.0004). When stratified by diabetes type, studies examining preexisting diabetes mellitus (PDM) showed a stronger association with childhood cancer risk. Notably, Westbom 2002 reported the highest effect estimate (OR = 2.25, 95% CI: 1.22–4.15) [21], while Aberg 2001 (GDM) showed no significant association (OR = 0.95, 95% CI: 0.61–1.49) [19]. The prediction interval ranged from 0.83 to 2.00, indicating that while the overall effect is positive, individual study settings may yield varying results.

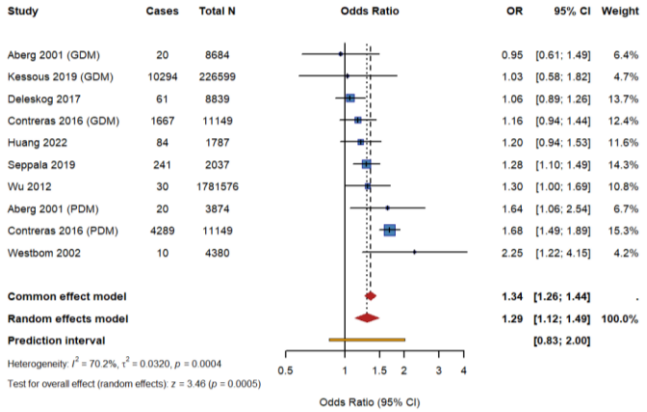

Figure 2: Pooled effect of maternal diabetes on the risk of childhood cancers in the offspring. Data are presented as OR for each study (boxes), 95% CIs (horizontal lines) and summary as RR with 95% CI (diamond). OR odds ratio, CI confidence interval, GDM gestational diabetes mellitus, PDM preexisting diabetes mellitus

3.4 Maternal Diabetes and Childhood Leukemia Risk

Seven studies comprising 34,169 participants evaluated the relationship between maternal diabetes and childhood leukemia risk [11, 17, 18, 22, 30, 33, 38] (Figure 3). The pooled analysis revealed a significant positive association (OR = 1.33, 95% CI: 1.17–1.51, p < 0.0001). Importantly, no heterogeneity was detected among the included studies (I^2 = 0.0%, τ^2 = 0, p = 0.60), suggesting consistent findings across different study populations and settings. The effect estimates ranged from 1.14 (Contreras 2016, GDM) [30] to 2.99 (Petridou 1997) [18], with the majority of studies showing point estimates above 1.0. The largest contribution to the pooled estimate came from Contreras 2016 (PDM) [30] with a weight of 41.2%, followed by Deleskog 2017 (22.2%) [33] and Seppala 2019 (17.8%) [38]. The prediction interval (1.15–1.54) further supported a consistent positive association across potential future studies.

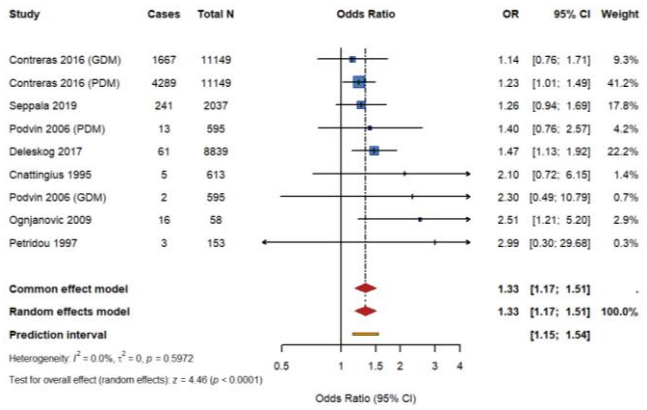

Figure 3: Pooled effect of maternal diabetes on the risk of childhood leukemia risk in the offspring

3.5 Maternal Diabetes and Childhood ALL Risk

Seven studies with 22,669 participants specifically examined the association between maternal diabetes and ALL in offspring [23, 25, 30, 33-35, 38] (Figure 4). The meta-analysis demonstrated a significant 44% increased risk of ALL among children born to mothers with diabetes (OR = 1.44, 95% CI: 1.27–1.64, p < 0.0001). Similar to the leukemia analysis, no heterogeneity was observed (I^2 = 0.0%, τ^2 = 0, p = 0.57). Among the included studies, Soegaard 2018 (PDM) reported the highest risk (OR = 2.90, 95% CI: 1.30–6.49) [34], followed by Borsari 2019 (PDM) (OR = 2.60, 95% CI: 0.62–10.88) [35]. Studies focusing on GDM generally showed lower effect estimates compared to those examining PDM. The prediction interval (1.24–1.68) indicated robust evidence for the positive association.

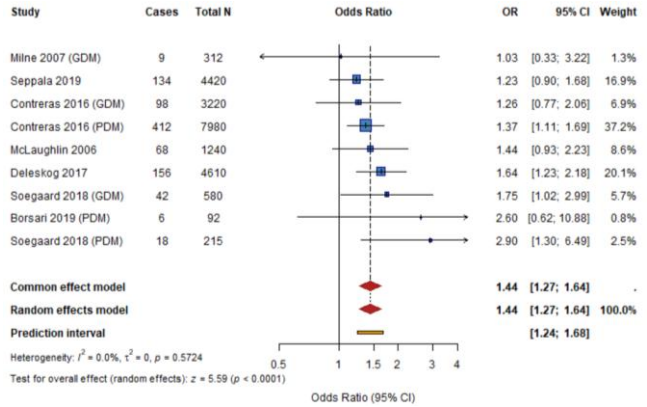

Figure 4: Pooled effect of maternal diabetes on the risk of ALL risk in the offspring. ALL acute lymphoblastic leukemia

3.6 Publication Bias Assessment

Funnel plots were constructed to assess potential publication bias for each outcome (Figure 5). For childhood cancer (Figure 5a), the funnel plot showed slight asymmetry with some smaller studies appearing on the right side of the pooled estimate, suggesting possible publication bias or small-study effects. However, the funnel plots for leukemia (Figure 5b) and ALL (Figure 5c) appeared relatively symmetric, indicating minimal evidence of publication bias in these analyses. These findings should be interpreted with caution given the limited number of studies in each analysis.

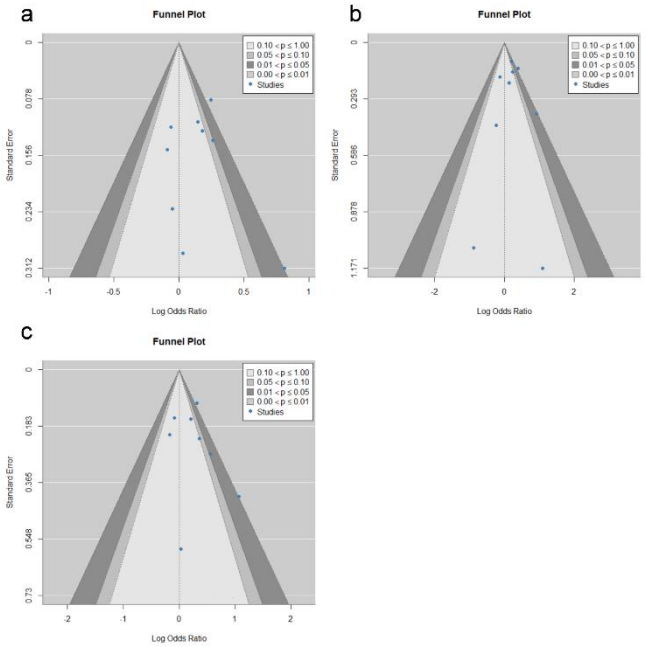

Figure 5: Funnel plots of studies exploring the association between maternal diabetes and the risk of childhood cancer (a), leukemia (b) and ALL (c) in the offspring. ALL acute lymphoblastic leukemia

4. Discussion

The present systematic review and meta-analysis incorporating 26 studies provides comprehensive and updated evidence regarding the association between maternal diabetes and childhood cancer risk in offspring. Our findings demonstrate that maternal diabetes is significantly associated with an increased risk of overall childhood cancer (OR = 1.29), childhood leukemia (OR = 1.33), and acute lymphoblastic leukemia (OR = 1.44). These results not only corroborate but also extend previous findings by addressing methodological limitations of earlier syntheses and incorporating recently published large-scale population-based studies.

The biological plausibility underlying the observed associations is supported by several mechanistic pathways [25, 39]. The intrauterine environment of diabetic pregnancies is characterized by maternal hyperglycemia, which induces fetal hyperinsulinemia and elevated circulating levels of insulin-like growth factors (IGFs), particularly IGF-1 and IGF-2 [40]. These growth factors are potent mitogens that promote cellular proliferation and inhibit apoptosis, thereby potentially initiating or accelerating carcinogenic processes during critical windows of fetal development [41]. Additionally, oxidative stress and chronic low-grade inflammation associated with maternal diabetes may cause DNA damage and epigenetic modifications in fetal cells, further contributing to cancer susceptibility. The "fetal origins of cancer" hypothesis posits that such adverse intrauterine exposures can permanently alter organ structure, cellular function, and gene expression patterns, establishing a predisposition to malignancy that may manifest during childhood or later in life [42].

Our stratified analyses revealed important distinctions between PDM and GDM in their associations with offspring cancer risk. Studies examining PDM consistently demonstrated stronger effect estimates compared to those focusing on GDM across all cancer outcomes [5, 11, 12]. This differential risk profile may reflect the longer duration and greater severity of metabolic dysregulation in pregnancies complicated by pre-existing type 1 or type 2 diabetes [41]. Women with PDM experience hyperglycemic exposure throughout the entire gestational period, including the critical first trimester when organogenesis occurs, whereas GDM typically develops during the second or third trimester [2]. Furthermore, PDM is often accompanied by additional comorbidities such as obesity, hypertension, and nephropathy, which may independently contribute to adverse fetal programming through inflammatory and vascular mechanisms.

The notably consistent findings for childhood leukemia and ALL, evidenced by the absence of statistical heterogeneity (I^2 = 0%), warrant particular attention. Leukemia is the most common childhood malignancy, and accumulating evidence suggests that its origins may be traced to prenatal events. The hematopoietic system undergoes rapid development during fetal life, making it particularly vulnerable to environmental insults. Maternal diabetes-induced hyperinsulinemia may directly stimulate the proliferation of fetal hematopoietic progenitor cells, while concurrent metabolic perturbations could promote the acquisition of pre-leukemic mutations. The consistent effect estimates across diverse geographic regions and study designs strengthen the causal inference and suggest that the association is robust and generalizable.

However, several limitations should be considered when interpreting our results. The substantial heterogeneity observed in the overall childhood cancer analysis (I^2 = 70.2%) suggests that unmeasured study-level factors may influence the association. Potential sources of heterogeneity include variations in the definition and ascertainment of maternal diabetes, differences in follow-up duration, and inconsistencies in confounder adjustment across studies. Although most studies controlled for maternal age, adjustment for other important confounders such as maternal obesity, socioeconomic status, and environmental exposures was inconsistent. Additionally, seven studies relied on self-reported diabetes status, which may introduce misclassification bias. The relatively small number of studies examining specific cancer subtypes other than leukemia precluded definitive conclusions regarding site-specific associations. Finally, the observational nature of included studies inherently limits causal inference, and residual confounding cannot be entirely excluded.

The clinical and public health implications of our findings are noteworthy. Given the rising global prevalence of diabetes among women of reproductive age, even modest increases in

relative risk could translate to substantial absolute numbers of affected children. Our results underscore the importance of optimal glycemic control before and during pregnancy, not only for preventing well-established perinatal complications but also for potentially reducing long-term cancer risk in offspring. Healthcare providers should counsel women with diabetes about comprehensive risk management, and future clinical guidelines may need to incorporate childhood cancer risk into the broader framework of preconception care for diabetic women.

Future research directions should prioritize several key areas. Large prospective cohort studies with detailed diabetes phenotyping, including hemoglobin A1c levels, duration of disease, and treatment modalities, are needed to further elucidate dose-response relationships and identify high-risk subgroups [43]. Mechanistic studies examining epigenetic modifications, cord blood biomarkers, and neonatal immune function in offspring of diabetic mothers could provide insights into underlying pathophysiology [44]. Additionally, investigations into whether specific childhood cancer subtypes beyond leukemia show differential associations with maternal diabetes types would help refine risk stratification [45]. Finally, studies evaluating whether improved maternal glycemic control during pregnancy attenuates offspring cancer risk would have direct clinical relevance.

5. Conclusion

In conclusion, this updated meta-analysis provides robust evidence that maternal diabetes is associated with a significantly increased risk of childhood cancer, particularly leukemia and acute lymphoblastic leukemia, in offspring. The stronger associations observed with preexisting diabetes compared to gestational diabetes suggest that the duration and timing of hyperglycemic exposure during pregnancy may be critical determinants of risk. These findings highlight the potential long-term consequences of the intrauterine diabetic environment and emphasize the importance of optimizing maternal metabolic health to protect offspring from adverse outcomes extending beyond the perinatal period.

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