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Iron Deficiency n Pregnancy and its Impact on Mother and Fetus: A Systematic Review in Experimental Animal Models

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Abstract
Iron deficiency is a common nutritional problem during pregnancy and is associated with maternal anemia and adverse pregnancy outcomes. Although clinical evidence in humans is widely available, mechanistic understanding of the impact of iron deficiency on the materno-fetal unit is still limited and requires support from experimental animal studies. This systematic review aims to comprehensively review the evidence from animal studies on the impact of iron deficiency during pregnancy on maternal health, placental function, and fetal development. Literature searches were conducted on PubMed, Scopus, and Web of Science databases for articles published between 2016–2025, following PRISMA guidelines. Studies that used models of pregnant animals with iron deficiency induction and reported maternal or fetal outcomes were included. Bias risk assessment was carried out using the SYRCLE tool. Seven studies met the inclusion criteria. Consistently, maternal iron deficiency causes anemia in the mother, placental dysfunction, increased oxidative stress, and inhibition of fetal growth and development. Evidence from animal models confirms the important role of iron in maintaining pregnancy health and provides a translationally relevant mechanistic basis for conditions in humans.

INTRODUCTION

Pregnancy is a physiological period characterized by a significant increase in metabolic and nutritional needs to support the adaptation of the mother's body as well as the growth and development of the fetus. Imbalances in intake and nutritional needs during this time can have a direct impact on maternal health and pregnancy outcomes, including an increased risk of maternal complications and impaired fetal growth.(Aguree & Gernand, 2019) One of the most common nutritional problems in pregnancy globally is iron deficiency, which is a condition when iron intake or reserves are insufficient to meet the needs of erythrocyte formation and oxygen transport, causing anemia. (Pratiwi et al., n.d.) Furthermore, a review of the clinical literature stated that anemia in pregnant women characterized by low hemoglobin levels as an indicator of iron deficiency was closely associated with an increased risk of adverse pregnancy outcomes.(McCann & Ames, 2007) Pregnant women with hemoglobin levels of less than 10 g/dL are reported to have a risk of preterm birth about 2.6 times higher than mothers with normal hemoglobin levels. In addition, hemoglobin levels below 9.0 g/dL during pregnancy are associated with an increased risk of giving birth to babies with low birth weight up to 3.6 times.(Wibowo et al., 2021) These findings suggest that the severity of iron deficiency anemia plays an important role in determining the risk of pregnancy complications.

The role of iron in normal pregnancy is very important because it functions in the transport of oxygen through hemoglobin, DNA synthesis, as well as a cofactor of various essential enzymes that support cell metabolism. (Fisher & Nemeth, 2017) In the World Health

Organization report during pregnancy there is an increase in plasma volume of up to about 40%, while the increase in erythrocyte mass takes place disproportionately, so that the total iron requirement increases significantly to maintain hemoglobin balance and prevent physiological anemia (WHO, 2024). Sufficient iron is needed to ensure optimal oxygen supply to the maternal-fetal unit and support the process of cell differentiation and development of fetal organs, especially the central nervous system. (Georgieff, 2020) Therefore, adequate iron status is an important prerequisite for maintaining a normal pregnancy and preventing the occurrence of maternal anemia and its accompanying pregnancy complications.

Various Studies say that iron deficiency during pregnancy is known to cause various adverse impacts on both mother and fetus, especially through the occurrence of maternal anemia and disruption of oxygen supply to the materno fetal unit. For example, Studieses that reported that iron deficiency anemia in pregnant women was associated with an increased risk of fatigue, decreased physical capacity, as well as obstetric complications such as bleeding and increased susceptibility to infection. (Kemppinen et al., 2021) In addition, from a fetal perspective, it is shown that maternal iron deficiency is related to stunted fetal growth, low birth weight, and an increased risk of premature birth. (Figueiredo et al., 2018) In addition, the World Health Organization report confirms that impaired iron status during pregnancy can affect placental function and the efficiency of oxygen and nutrient transfer to the fetus. (WHO, 2024) These findings confirm that iron deficiency is a pathological condition that has a systemic impact, not only on the mother's hematologic status but also on the development and health of the fetus.

Although the impact of iron deficiency during pregnancy has been widely reported in clinical and epidemiological Studieses in humans, scientific evidence explaining the biological mechanisms in depth is still limited. This is mainly due to the limitations of human Studieses in controlling confounding factors such as nutritional status, environment, and comorbid conditions. Experimental animal models are indispensable to understand the consequences of iron deficiency at the cellular, tissue, and organ levels during pregnancy. (Roberts et al., 2021) However, to date the available animal Studieses show great variation in the type of animal model, the degree and duration of iron deficiency, and the *outcome parameters* evaluated, including aspects of maternal hematology, placental function, and fetal development. (Chen et al., 2024) The heterogeneity of the design and the results of the study makes it difficult to draw consistent and integrated conclusions. Therefore, a systematic review that comprehensively consolidates evidence from experimental animal Studieses is needed to identify patterns of effects and knowledge gaps that still exist.

Based on the above background, this systematic review aims to comprehensively review the scientific evidence from experimental animal Studieses examining the impact of iron deficiency during pregnancy on maternal health and fetal development. This review specifically included Studieses with models of pregnant animals with iron deficiency, with the main outcomes including maternal hematological parameters such as hemoglobin levels, changes in placental structure and function, fetal growth and weight, as well as indicators of oxidative stress and organ developmental disorders. By synthesizing the results of animal Studieses published in reputable journals, this review is expected to provide a deeper mechanistic understanding of the effects of iron deficiency on the materno-fetal unit. Furthermore, the findings of this systematic review are expected to provide practical benefits as a scientific basis for the development of nutritional intervention strategies, early detection programs, and evidence-based health policies to prevent iron deficiency anemia in pregnant women. In addition, this review also contributes to identifying research gaps that can guide the design of future experimental studies, particularly in exploring the reversibility of iron deficiency effects and sex-specific fetal responses. In addition, the results of this review are important for identifying research gaps that still exist and serve as a scientific basis for the development of further research and nutrition intervention strategies in pregnancy.

RESEARCH METHODS

This systematic review was prepared and implemented with reference to the Preferred Reporting Items for Systematic Reviews and Meta Analyses (PRISMA) guidelines to ensure transparency, reproducibility, and accuracy in the process of identifying, selecting, evaluating, and synthesizing relevant Studieses. This approach is used to ensure that all stages of the review are conducted systematically and free from unnecessary methodological bias. The literature search was conducted comprehensively using several major electronic databases, namely Google scholer, PubMed, Scopus, and Web of Science, which were chosen for their extensive coverage of the biomedical and experimental literature. The search was limited to articles published in the period of January 2016 to December 2025, to ensure that the scientific evidence reviewed is up-to-date and relevant to the latest research developments. The search keywords were compiled by combining terms related to iron deficiency, pregnancy, animal models, and maternal and fetal output. Key terms include a combination of keywords such as "iron deficiency", "pregnancy", "gestation", "maternal anemia", "placenta", "fetal growth", "birth weight", "oxidative stress", and "animal model". These keywords are tailored to each database to accommodate differences in indexing systems and search algorithms. In addition, a manual search of the bibliography of relevant articles and reviews was also conducted to identify additional Studieses that might not have been netted through electronic searches.

1. To maintain the focus and quality of the review, clear inclusion and exclusion criteria are applied from the start.
2. Inclusion criteria include:
3. Original research articles published in reputable and peer-reviewed journals;
4. Studieses that use models of pregnant animals e.g. mice, mice, or other mammals as research subjects;
5. Research that induces or evaluates iron deficiency during pregnancy, either through dietary modifications or other experimental approaches;
6. Studieses that report on at least one major outcome, such as maternal hematological parameters, placental development and function, fetal growth and weight, or markers of oxidative and inflammatory stress.

Exclusion criteria include:

1. In vitro Studieses or non-animal Studieses;
2. Research on humans;
3. Review articles, editorials, case reports, and conference abstracts;
4. Studieses that do not report outcomes relevant to maternal or fetal health;
5. Publications not available in English.

The study selection process is carried out through two stages, namely title and abstract screening, followed by a full text assessment. Two independent reviewers conducted an initial screening to assess the suitability of the study based on the established inclusion and exclusion criteria. Articles that are considered relevant are then thoroughly reviewed at the full-text stage to ensure their feasibility and methodological quality. Differences of opinion between reviewers are resolved through discussion until consensus is reached. Meanwhile, data extraction is carried out using a standard form that has been prepared in advance to ensure consistency. The information collected included study characteristics such as authors, year of publication, animal types and models, iron deficiency induction methods, duration of interventions, and major reported outcomes. The data obtained were then synthesized descriptively and narratively, with groupings based on the type of maternal and fetal outputs, in order to provide a comprehensive picture of the impact of iron deficiency during pregnancy on experimental animal models.

RESULTS AND DISCUSSION

The process of identifying and selecting Studieses in this systematic review was carried out following the PRISMA guidelines, as shown in Figure 1. An initial search of the four major electronic databases, namely Google Scholar, PubMed, Scopus, and Web of Science, yielded a total of 1345 articles. In the initial identification stage, a number of articles were eliminated before the screening process, consisting of 400 duplicate articles, 200 articles that were declared unsuitable based on the automatic selection tool, and 400 articles that were removed for other reasons, leaving 345 articles for the screening stage. Furthermore, the screening of titles and abstracts of the 345 articles resulted in 275 articles being excluded because they did not meet the inclusion criteria. A total of 73 articles were then attempted to be obtained in the form of full text, but 56 articles were not successfully obtained, so that only 17 articles could be assessed for feasibility through full text review. At this stage, 10 articles were again excluded, with details of 5 articles excluded because they were not published in the last 10 years, 2 articles were not published in English, 1 article used inappropriate methods and 2 articles were not suitable for the purpose of the study. Thus, 7 Studieses met all inclusion criteria and were included in this systematic review as the basis for data analysis and synthesis.

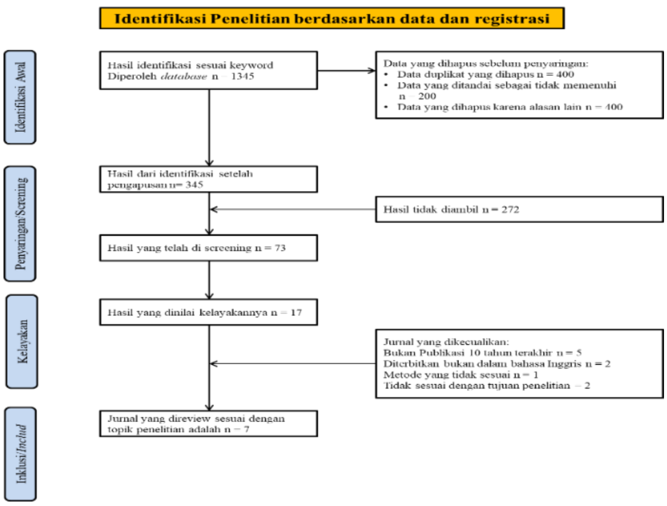

Figure 1. Diagram PRISMA

Source: Author's analysis based on database search results, 2026

Based on figure 1, the study selection flow is systematically described based on the PRISMA guidelines, starting from the identification stage to the final inclusion. This diagram shows a rigorous screening process to ensure that only Studieses that are relevant, qualitative, and compliant with the inclusion criteria are included in the review. The selected Studieses were then analyzed in depth to examine the characteristics of the animal model, the study design, and the various main outcomes reported, including maternal parameters, fetal development, and findings related to placental function. The results of the synthesis of these Studieses are presented in the next section.

Table 1. Study Characteristics Include

ID Studieses	Animal Model (Species, Strain, Sex)	Iron Deficiency Induction	Diet / Method of Giving	Duration of Exposure	Number of Samples/Batches	Outcome Maternal	External Placenta & Fetal	Key Findings
Studieses 1	Pregnant female Sprague-Dawley rat	A low-iron diet begins before pregnancy and continues until the end of gestation.	Standard feed low in iron compared to control diet	Pre-pregnancy to late pregnancy	±10 parents/group	Decreased maternal hemoglobin levels and iron reserves	Fetal weight loss, changes in placental weight and structure	Maternal iron deficiency causes anemia in the mother, inhibits fetal growth, and affects placental development and function.
Studieses 2	Pregnant female Wistar rat	Low iron diet to model iron deficiency anemia idA	Low iron diet until late pregnancy; some additional groups received iron supplementation	Pre-pregnancy and pregnancy (until P0-P 42 postpartum)	IDA model: n≈12; Supplementation group: n≈12 per type	Maternal hematological parameters improved after supplementation; anemia was characterized by low Hb in the IDA model.	Physical growth and neurodevelopment in offspring were better after supplementation; number of live offspring per litter increased.	Iron deficiency causes hematological and physical and neurological developmental disorders in offspring, but iron supplementation improves many of these outcomes.

Studieses 3	Pregnant female C57BL/6 mice	Low iron diet (ID) versus iron-sufficient diet	ID diet starts before and during pregnancy	Until mid-gestation	n≈8 broodstock per diet	Changes in maternal iron gene expression & proteome profile	Changes in the expression of placental transcriptomes and proteomes, including iron and transporter genes	Maternal iron deficiency modifies the expression of genes and proteins in the placenta of mice, with changes in iron transporter genes and several metabolic pathways that differ according to the sex of the fetus.
Studies 4	Pregnant female C57BL/6 mice	Diet low in iron (<6 ppm) from before mating to gestation	Diet low in iron compared to control	Until *mid-gestation*	Not mentioned (general model C57BL/6)	Maternal hemoglobin & iron stores decreased significantly	Iron deficiency increases the incidence of congenital heart defects, including septal defects and fetal heart structural disorders	Maternal iron deficiency significantly interferes with the cardiovascular development of the embryo, causing structural defects of the heart in offspring, and this effect can be prevented by iron supplementation early in pregnancy.

Studies 5	Pregnant female Wistar rat	A low-iron (ID) diet given from pre-pregnancy to the end of gestation	Low iron diet vs control diet	Pre-pregnancy to the 20th day of pregnancy	±8-10 parent/group	decreased maternal Hb, low serum ferritin, increased anemia parameters	Decrease in fetal weight and placental size; increased oxidative stress of the placenta	Maternal iron deficiency causes significant anemia, decreased fetal growth, as well as increased indicators of placental oxidative stress.
Studies 6	Pregnant female C57BL/6 mice	A low-iron diet (4 ppm) is given 2 weeks before and during pregnancy	ID diet compared to replete diet or iron dextran injection	2 weeks pre-mating + pregnancy (E12.5, E15.5, E18.5)	±8–10 per group	Maternal hepcidin regulation and iron parameters change according to iron status	Changes in the expression of placental iron transporters (TFR1 and Ferroportin) as well as the distribution of placental and fetal iron	Maternal iron deficiency alters the expression of iron transporters in the placenta, prioritizing iron retention in the placenta but reducing supply to the fetus, reflecting adaptive mechanisms that impact fetal iron homeostasis.
Studies 7	Pregnant female Sprague-Dawley rat	A low-iron diet is given from pre-mating to the end of pregnancy	Iron deficiency diet compared to replete diet	±2 weeks pre-mating + entire pregnancy	±7–8 dams per group	Significant decrease in maternal Hb and fetal Hb	Weight loss of the fetus; resistive changes of the umbilical index; Fetal organ-specific hypoxia	Prenatal iron deficiency causes maternal and fetal anemia, asymmetrically restricted fetal growth, and organ-specific hypoxia in the fetus, although not always

									with severe maternal anemia.

source: secondary data compiled by the author, 2026

Table 2. Risk Assessment of Bias Studies of Animals Using SYRCLE

ID Studies	Sequence Generation (Randomization)	Allocation Concealment	Blinding Animal Researchers & Nurses	Blinding Outcome Assessors	Incomplete Outcome Data	Selective Reporting	Other Biases	Overall Risk of Bias	Brief Justification
Studies 1	Unclear	Unclear	Unclear	Unclear	Low	Low	Unclear	Medium	The methods of randomization and blinding are not described; All animals were analyzed and key outputs were reported according to the study objectives.
Studies 2	Unclear	Unclear	Unclear	Unclear	Low	Low	Low	Medium	Studies reported some intervention groups well, but did not describe the randomization and blinding procedures in detail.
Studies 3	Unclear	Unclear	Unclear	Unclear	Low	Low	Unclear	Medium	The main focus is on molecular analysis; Allocation and blinding procedures are not reported, but complete data are available.

ID Studies	Sequence Generation (Randomization)	Allocation Concealment	Blinding Animal Researchers & Nurses	Blinding Outcome Assessors	Incomplete Outcome Data	Selective Reporting	Other Biases	Overall Risk of Bias	Brief Justification
Studies 4	Unclear	Unclear	Unclear	Unclear	Low	Low	Unclear	Medium	There is no explicit description of randomization and blinding; however, all major outcomes (heart defects) were reported consistently.
Studies 5	Unclear	Unclear	Unclear	Unclear	Low	Low	Unclear	Medium	Hematological parameters and oxidative stress were reported to be complete, but experimental bias control methods were not described.
Studies 6	Unclear	Unclear	Unclear	Unclear	Low	Low	Low	Medium	The experimental design was relatively strong with several comparison groups, but the details of randomization and blinding were not mentioned.
Studies 7	Unclear	Unclear	Unclear	Unclear	Low	Low	Unclear	Medium	Studies report maternal and fetal outcomes comprehensively, but without explicit

ID Studies	Sequence Generation (Randomization)	Allocation Concealment	Blinding Animal Researchers & Nurses	Blinding Outcome Assessors	Incomplete Outcome Data	Selective Reporting	Other Biases	Overall Risk of Bias	Brief Justification
									explanations of blinding procedures and allocation.

source: author's analysis, 2026

Based on table 1 have been presented the characteristics of seven experimental animal studies that meet the inclusion criteria in this systematic review. All studies used rodent models, especially Sprague-Dawley rats, Wistar, and pregnant female C57BL/6 mice, with iron deficiency induction generally carried out through the administration of a low-iron diet from the pre-pregnancy period to the end of gestation. The duration of exposure and the number of samples varied between studies, but all of them assessed the key relevant outcomes, including maternal hematological parameters, placental changes, and fetal growth and development. Consistently, the Studies reported that maternal iron deficiency causes anemia in the mother and negatively affects fetal weight, placental size and function, and fetal organ development.

The risk assessment of bias against the included studies was conducted using the SYRCLE tool, as summarized in Table 2. The results of the assessment showed that most Studies had an overall risk of bias in the medium category. This is mainly due to the lack of clear reporting on randomization methods, allocation concealment, and blinding of researchers and outcome assessors, which are common in animal studies. Nevertheless, all studies show a low risk of bias in the domain of incomplete outcome data and selective reporting, as the number of animals analyzed is as planned and the reported outputs are aligned with the research objectives.

Table 3 further summarizes the histopathological changes in maternal, placenta, and fetal tissues, as well as their relationship to increased oxidative stress and inflammatory responses reported in each of the studies.

Table 3. Histopathological Findings and Mechanistic Linkages Due to Iron Deficiency During Pregnancy in Animal Models

ID Studies	Organ	Histopathological findings (description, location, severity)	Markers of Oxidative Stress / Lipid Peroxidation	Markers of Inflammation	Mechanistic Linkages
Studies 1	Placenta	Thinning of the trophoblast layer, decreased vascularization of the labyrinth zone, changes in villious morphology (mild-Medium)	Increased placental MDA compared to controls	Increased expression of local TNF-α	Iron deficiency triggers placental oxidative stress that interferes with the transfer of nutrients and oxygen to the fetus

	Fetus	Fetal weight loss; Inhibited tissue maturation	↑ Fetal tissue MDA	-	Disruption of oxygen and iron supply due to placental dysfunction
Studies 2	Induct (systemic)	Non-specific histopathology of tissues; Clear maternal anemia	-	-	Maternal anemia affects the postnatal development of the offspring
	Hereditary brain	Delayed neuronal development; Impaired synapse maturation	↑ MDA brain	↑ IL-6	Prenatal iron deficiency has a long-term impact on neuro development
Studies 3	Placenta	There is no real necrosis; changes in iron transporter gene expression (TfR, Fpn)	Increased placental oxidative stress markers	-	Molecular adaptation of the placenta to maternal iron deficiency
Studies 4	Fetal heart	Structural abnormalities of the heart (septal defects, trabeculation disorders)	Indications of increased embryonic oxidative stress	-	Iron deficiency interferes with the differentiation of heart cells during organogenesis
Studies 5	Placenta	Trophoblast cell degeneration, interstitial edema, increased oxidative stress (Medium)	↑ MDA placenta	↑ Pro-inflammatory cytokines	Placental oxidative stress worsens fetal growth
	Fetus	The weight of the fetus decreases; intrauterine growth retardation	↑ MDA fetal tissue	-	Combination of maternal anemia and placental dysfunction
Studies 6	Plasenta	Changes in the expression of the iron transporter; Iron retention in the placenta	↑ Oxidative Markers	-	Placental compensation mechanism that actually decreases iron supply to the fetus
	Fetus	Decreased fetal iron status without severe anemia	-	-	Maternal–placental iron homeostasis priority over fetal iron
Studies 7	Fetal brain	Degeneration of neurons in the cortex and hippocampus; increased apoptosis	↑ 4-HNE brain tissue	↑ TNF-α	Hypoxia and oxidative stress due to prenatal iron deficiency
	Janin (systemic)	Stunted fetal growth; organ-specific hypoxia	↑ Oxidative markers	-	Maternal anemia and disorders of uteroplacental flow

source: data synthesis by the author, 2026

The results of this systematic review found that iron deficiency during pregnancy in animal models consistently had an adverse impact on maternal hematological status, placental function, and fetal growth and development. Clinical evidence in humans suggests that iron deficiency is a major cause of maternal anemia and is associated with poor pregnancy outcomes, such as low birth weight and premature birth. Induction of iron deficiency through a low-iron diet since pre-pregnancy leads to a decrease in hemoglobin levels and maternal iron reserves, which represents a condition of iron deficiency anemia as it occurs in human pregnant women with inadequate iron intake. (Roberts et al., 2021)

From a maternal perspective, the decrease in hemoglobin and ferritin levels observed in mouse and mouse models showed that pregnancy aggravates iron requirements and accelerates the occurrence of anemia if not balanced with adequate intake. (Khan et al., 2024) This condition is particularly relevant to humans, where increased plasma volume during pregnancy leads to physiological hemodilution and increases iron requirements by almost double compared to before pregnancy. Thus, the findings in animals reinforce the understanding that maternal iron deficiency is not just a mild condition, but a systemic disorder that can affect the mother's oxygen transport capacity and metabolic homeostasis.

At the placental level, several studies have shown that maternal iron deficiency induces structural and molecular changes in the placenta, including changes in the expression of iron transporters such as *transferrin receptor* 1 (TFR1) and *ferroportin*, as well as increased oxidative stress indicators. (Cao et al., 2021) This finding has high translational relevance, because in humans the placenta plays a role as the main regulator of iron transfer from mother to fetus. Disruption of placental iron transporter regulation due to maternal iron deficiency can lead to an iron distribution imbalance, in which the placenta retains iron for its own metabolic functions, but reduces its supply to the fetus. These adaptive mechanisms, although short-term protective, have the potential to be detrimental to fetal development.

The most consistent impact of maternal iron deficiency across the reviewed studies was impaired fetal growth and development. Fetal weight loss, intrauterine growth restriction, and changes in the oxygen and iron distribution of fetal tissue were reported in almost all studies.(Woodman et al., 2017) In addition, maternal iron deficiency can interfere with organogenesis, particularly the cardiovascular development of the embryo, leading to an increased incidence of congenital heart defects.(Kalisch-smith et al., 2021) These findings support epidemiological evidence in humans linking iron deficiency anemia during pregnancy with impaired organ development and an increased risk of cardiovascular disease in offspring. Furthermore, several studies have revealed that the effects of maternal iron deficiency are complex and are influenced by the time of exposure and the sex of the fetus. (Cao et al., 2021) Changes in the expression of different placental genes and proteins according to the sex of the fetus suggest that the adaptive response to iron deficiency is not uniform. (Sangkhae et al., 2021) This is relevant to human studies that increasingly recognize differences in male and female fetal responses to intrauterine nutritional stress, including micronutrient deficiencies.

The findings of adding an important dimension related to the reversibility of iron deficiency effects. Iron supplementation in animal models with iron deficiency anemia has been shown to improve maternal hematological parameters as well as improve physical growth and neurological development of the offspring.(Zhang et al., 2021) This reinforces clinical recommendations in humans regarding the importance of early detection and iron supplementation during pregnancy to prevent both short-term and long-term impacts on the mother and child. Nevertheless, the results of this review need to be interpreted taking into account the limitations of available animal studies. Variations in experimental design, the degree and duration of iron deficiency, and the outcome parameters measured. (Sangkhae et al., 2021) contribute to the heterogeneity of the findings. In addition, physiological differences between rodents and humans, particularly in the regulation of iron metabolism and the duration of pregnancy, limit the generalization directly to the human population. Nevertheless, the consistency of the direction of the findings between studies strengthens the biological validity of the relationship between maternal iron deficiency and pregnancy disorders.

Overall, the results of this systematic review confirm that iron deficiency during pregnancy is an important risk factor that has a broad impact on maternal health, placental function, and fetal development. Evidence from animal models provides a strong mechanistic basis for understanding causal relationships that have been observed in human clinical studies, as well as emphasizing the importance of preventing and managing iron deficiency from before and during pregnancy.

CONCLUSION

Based on the results and discussion, this systematic review shows that iron deficiency during pregnancy in animal models consistently leads to impaired maternal hematological status, placental dysfunction, and fetal growth and development inhibitions, including increased oxidative stress, altered iron transporter regulation, and disruption of organogenesis. The findings provide strong mechanistic evidence that maternal iron deficiency not only impacts maternal health, but also affects the intrauterine environment and fetal developmental programming. The consistency of results between studies reinforces the translational relevance to humans, in line with clinical evidence linking iron deficiency anemia in pregnancy with adverse perinatal outcomes. Therefore, prevention, early detection, and management of iron deficiency from before and during pregnancy are important strategies to support maternal health and optimize fetal development

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