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# Metabolomics Based Investigation of Prenatal Congenital Anomalies

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## Introduction

Fetal growth restriction (FGR) is a common pregnancy complication and a major cause of neonatal morbidity and mortality. The negative effects of FGR can last a lifetime and increase the risk of various diseases in adulthood. The aetiology and pathogenesis of FGR, however, are unknown. This study thoroughly reviewed metabolomics studies on FGR in pregnancy in order to identify potential metabolic biomarkers and pathways. Several amino acids, including alanine, valine and isoleucine, were found in high concentrations in both neonatal and maternal studies [1].

## Description

Meanwhile, several pathways, including arginine biosynthesis, arginine and proline metabolism, glyoxylate and dicarboxylate metabolism and alanine, aspartate and glutamate metabolism, have been proposed to be involved in the development of FGR. Furthermore, we included eight animal model studies in which three commonly reported metabolites (glutamine, phenylalanine and proline) were also present in human studies [2]. In general, this study summarised a number of metabolites and metabolic pathways that may aid in our understanding of the underlying metabolic mechanisms of FGR.

Fetal growth restriction (FGR) is an obstetric complication defined as a foetus failing to reach its predetermined intrauterine growth potential, also known as intrauterine growth restriction (IUGR). These two terms (FGR and IUGR) are commonly used interchangeably. When a newborn's birth weight falls below a predetermined threshold for gestational age, it is classified as small for gestational age (SGA). In comparison to FGR, SGA is an auxological but not an etiological definition. It is commonly assumed that FGR results in SGA [3].

FGR is typically classified as early onset (32 weeks) or late onset (32 weeks) based on the gestational age of diagnosis. FGR is the second leading cause of infant morbidity and mortality, after preterm birth, with an incidence of around 10%. The consequences of poor foetal growth may last throughout one's life, not just during the neonatal period. Several studies, for example, have found that FGR increases the risk of cognitive delay, obesity, cardiovascular disease and type 2 diabetes later in life. Because of its low cost and convenience, the former is widely used.

Maternal obesity, uterine leiomyomas and polyhydramnios, on the other hand, may limit its effectiveness in detecting FGR. On the one hand, the relatively high cost of ultrasound may limit its use in low-resource areas. Body habitus, operator experience and foetal position, on the other hand, may influence its accuracy. Meanwhile, the phenotypes of late-onset FGR are markedly different from those of early-onset FGR. Thus, except in the case of extremely small foetal size, a single foetal biometric measurement is insufficient to evaluate foetal growth. Perhaps complementary biological tests will aid in the identification of FGR [4,5].

## Conclusion

Furthermore, despite the fact that FGR is a multifactorial disease influenced by maternal, placental, foetal and genetic factors, its aetiology and pathogenesis are unknown. In order to accurately monitor foetal growth, more research on the aetiology, pathogenesis and metabolic drivers of FGR is urgently needed. Metabolomics is a new high-throughput technique for identifying and quantifying small molecules in biological systems in a comprehensive and systematic manner. The imbalance between reactive oxygen species (ROS) and protective antioxidants is referred to as oxidative stress. ROS overproduction can disrupt normal placental functions. In the current study, mothers who gave birth to FGR infants had a glutathione metabolism disorder. Glutathione, a natural antioxidant in the body, is a tripeptide made up of glycine, cysteine and glutamate. Glutathione metabolism disruption may result in increased oxidative products and oxidative stress, which may induce placental vascular lesions and lead to foetal compromise. The main vasodilatory agent of the placenta, nitric oxide (NO), is involved in implantation, fetoplacental vascular reactivity and placental perfusion. The activation of nitric oxide synthases produces this gas from arginine. Pathway analysis revealed arginine and proline metabolism as well as arginine biosynthesis, supporting NO synthesis dysfunction in FGR.

## Acknowledgement

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## Conflict of Interest

There are no conflicts of interest by author.

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