J Phys Med Rehabil. 2022;4(1):25-27.

Journal of Physical Medicine and Rehabilitation Commentary

Commentary on Association between Birth Weight and Risk of Cardiovascular Disease: Evidence from UK Biobank

Jingjia Liang[1,2], Aihua Gu[1,2,*]

[1]State Key Laboratory of Reproductive Medicine, School of Public Health, Nanjing Medical University, Nanjing, China

[2]Key Laboratory of Modern Toxicology of Ministry of Education, Center for Global Health, Nanjing Medical University, Nanjing, China

[*]Correspondence should be addressed to Aihua Gu, aihuagu@njmu.edu

Received date: July 01, 2022, **Accepted date:** July 13, 2022

Citation: Liang J, Gu1 A. Commentary on Association between Birth Weight and Risk of Cardiovascular Disease: Evidence from UK Biobank. J Phys Med Rehabil. 2022;4(1):25-27.

Abstract

Cardiovascular disease (CVD) is one of the major causes of death worldwide. The risk of CVD in adulthood is influenced not only by genetic and adult lifestyle factors, but also by early environmental factors. Low birth weight is a marker of intrauterine stress and is associated with susceptibility to CVD. This commentary discussed the role of various causes of birth weight changes, such as preterm birth, intrauterine growth restriction, and multiple births, in increasing susceptibility to cardiovascular disease in adults. Early identification of individuals at risk of cardiovascular disease is critical for developing effective preventions.

Keywords: Cardiovascular disease, Birth weight, Preterm birth, Intrauterine growth restriction, Multiple births

Cardiovascular disease (CVD) is one of the major causes of death worldwide and poses a huge public health burden [1,2]. Early identification of individuals at risk of cardiovascular disease is crucial to develop effective preventions. The DOHAD hypothesis proposed by Professor Barker et al. states that an abnormal environment early in life can alter an individual's organ structure, biochemistry, genetics and/or physiology, thereby increasing the risk of diseases later in life, namely that cardiovascular disease can be programmed in early life [3,4].

In our study, the association between birth weight and adult CVD was observed by Cox regression using recall of birth weight data from a large UKB cohort [5]. There was a J-shaped correlation between birth weight and CVD, in which low birth weight was strongly correlated with the occurrence of CVD, and macrosomia (high birth weight) also had a certain increase in the risk of CVD, notably coronary heart disease. we redefined a more precise birth weight range with a lower risk of CVD, based on the WHO standard birth weight range. This provides a clear target for the prenatal and perinatal care of children in the future.

Consistent with our study, a previous meta-analysis showed that a U-shaped non-linear relationship between birth weight and overall CVD and its subtypes [6]. Birth weight lower than ~2500 g and higher than ~4000 g had a positive effect on CVD risk. Furthermore, low birth weight was found to be directly associated with CVD subtype, e.g. coronary heart disease (CHD) and stroke. However, the dose-response relationship described in the meta-analysis was based on classification data of birth weight, which might affect the correlation compared to the direct analysis of continuous variables. The high heterogeneity between studies could not be avoided even after subgroup analysis. In our study, the large sample data of UK Biobank can effectively avoid such bias caused by insufficient sample size in the analysis.

Birth weight is an intuitive reflection of the overall condition of the baby during pregnancy, which can approximately judge the living environment and nutritional status of the baby *in utero* [7-10]. Our study focused on the association between birth weight and the risk of CVD, but reckoned without the effect of other prenatal factors on CVD, such as premature birth and intrauterine malnutrition, because of the lack of prenatal factors in UK Biobank. It is necessary to further subdivide the low birth weight caused by different causes, so that it may be

easier to find the important factors causing low birth weight and its important influence on cardiovascular disease. This will provide the basis for clearer guarantees of health care during pregnancy and the perinatal period.

The current study also found that preterm birth is associated with an increased risk of lipid disorders in early to middle adulthood [11]. In addition, preterm birth was found to be a predictor of CVD risk in young women before they developed identified CVD risk factors [6]. In premature infants, gestational age was negatively correlated with school-age systolic blood pressure [12,13]. In other words, preterm infants were smaller but had higher systolic blood pressure at school age. However, blood pressure is affected by many factors, including age [14], weight [15], socioeconomic status [16, 17] etc. Further studies are needed to clarify the exact mechanism of preterm birth and the risk of elevated blood pressure in adulthood. Moreover, a recent study reported that preterm birth was associated with greater total fat mass, suggesting that preterm birth may affect cholesterol metabolism [18]. Thus, preterm birth may be one of the major gestational factors causing low birth weight and may contribute to an increased risk of cardiovascular disease.

The population study in Preston and Sheffield focused on examining birth records such as physical measurements taken at birth [19]. Among men born in Sheffield, those who were younger at term showed an increased incidence of risk factors for CHD, suggesting that low birth weight from other causes in addition to preterm birth is associated with cardiovascular disease in adulthood. Fetal intrauterine growth restriction (IUGR) is defined as occurring when fetal weight is below the 10th percentile of gestational age [20]. IUGR is associated with nutrition during pregnancy [21], multiple birth [22,23], maternal cardiovascular disease [24], placental insufficiency [25], drug abuse [26], smoking during pregnancy [27,28]. Clinical study showed a link between maternal malnutrition, low birth weight and the incidence of CVD [20]. It has been suggested that postpartum catch-up growth plays an important role in the increasing incidence of CVD due to adaptation to the adverse environment *in utero* [29]. Children with a history of IUGR generally have a lower growth rate and a smaller body size around puberty compared to healthy infants born at term with adequate birth weight, although catch-up growth occurs in the first years of life [30]. The association between the occurrence of advanced metabolic diseases and cardiovascular diseases and IUGR may be related to the low inflammatory manifestations [30]. It has proved that IUGR may induce significant metabolic and inflammatory abnormalities, which increases the risk of obesity and cardiovascular disease later in life [31]. Together, low birth weight caused by IUGR may be one of the important causes of cardiovascular disease in adulthood.

According to Barker's hypothesis, adverse outcomes for twins are expected because their birth weight is usually lower than that of single birth. However, twin studies have been inconclusive in terms of increased incidence of adult disease. de Boo and Harding [32] compared adult twins to singleton siblings or other singleton births. They found no difference in CHD blood pressure or death rates between multiple and single births. The relationship between low birth weight and the development of advanced disease in twins and singleton cases may differ [33], as it has been suggested that the intrauterine environment of twins and singleton cases is also different [32].

Longer follow-up periods and better information collection systems during pregnancy are necessary so as to determine the impact of low birth weight caused by different prenatal factors, such as preterm birth, IUGR and multiple births, on cardiovascular risk factors and cardiovascular disease risk.

References

1. Abrams LR, Myrskyla M, Mehta NK. The growing rural-urban divide in US life expectancy: contribution of cardiovascular disease and other major causes of death. Int J Epidemiol. 2022;50(6):1970-8.

2. Zhao D, Liu J, Wang M, Zhang X, Zhou M. Epidemiology of cardiovascular disease in China: current features and implications. Nat Rev Cardiol. 2019;16(4):203-12.

3. Barker DJ. The fetal and infant origins of adult disease. BMJ. 1990;301(6761):1111.

4. Lapehn S, Paquette AG. The Placental Epigenome as a Molecular Link Between Prenatal Exposures and Fetal Health Outcomes Through the DOHaD Hypothesis. Curr Environ Health Rep. 2022.

5. Liang J, Xu C, Liu Q, Fan X, Xu J, Zhang L, et al. Association between birth weight and risk of cardiovascular disease:Evidence from UK Biobank. Nutr Metab Cardiovasc Dis. 2021;31(9):2637-43.

6. Mohseni R, Mohammed SH, Safabakhsh M, Mohseni F, Monfared ZS, Seyyedi J, et al. Birth Weight and Risk of Cardiovascular Disease Incidence in Adulthood:a Dose-Response Meta-analysis. Curr Atheroscler Rep. 2020:22(3):12.

7. Günther V, Alkatout I, Vollmer C, Maass N, Strauss A, Voigt M. Impact of nicotine and maternal BMI on fetal birth weight. BMC Pregnancy Childbirth. 2021;21(1):127.

8. Kozłowska A, Jagielska AM, Okręglicka KM, Oczkowski M, Przekop D, Szostak-Węgierek D, et al. Maternal Nutritional and Water Homeostasis as a Presage of Fetal Birth Weight. Adv Exp Med Biol. 2019;1176:89-99.

9. Mélançon J, Bernard N, Forest JC, Tessier R, Tarabulsy GM, Bouvier D, et al. Impact of maternal prenatal psychological stress on birth weight. Health Psychol. 2020:39(12):1100-8.

10. Murata T, Kyozuka H, Fukuda T, Yasuda S, Yamaguchi A, Morokuma S, et al. Maternal sleep duration and neonatal birth weight:the Japan Environment and Children's Study. BMC Pregnancy Childbirth. 2021;21(1):295.

11. Crump C, Sundquist J, Sundquist K. Association of preterm birth with lipid disorders in early adulthood:A Swedish cohort study. PLoS Med. 2019;16(10):e1002947.

12. Belfort MB, Gillman MW, Mccormick MC. Prenatal and perinatal predictors of blood pressure at school age in former preterm, low birth weight infants. J Perinatol. 2012;32(4):265-9.

13. Vohr BR, Heyne R, Bann C, Das A, Higgins RD, Hintz SR, et al. High Blood Pressure at Early School Age Among Extreme Preterms. Pediatrics. 2018;142(2).

14. Oliveras A, De La Sierra A. Resistant hypertension:patient characteristics, risk factors, co-morbidities and outcomes. J Hum Hypertens. 2014;28(4):213-7.

15. Sagaro G G, Di Canio M, Amenta F. Correlation between body mass index and blood pressure in seafarers. Clin Exp Hypertens. 2021;43(2):189-95.

16. Mourtzinis G, Manhem K, Kahan T, Schiöler L, Isufi J, Ljungman C, et al. Socioeconomic status affects achievement of blood pressure target in hypertension:contemporary results from the Swedish primary care cardiovascular database. Scand J Prim Health Care. 2021;39(4):519-26.

17. Leng B, Jin Y, Li G, Chen L, Jin N. Socioeconomic status and hypertension:a meta-analysis. J Hypertens. 2015;33(2):221-9.

18. Mortaz M, Fewtrell MS, Cole TJ, Lucas A. Birth weight, subsequent growth, and cholesterol metabolism in children 8-12 years old born preterm. Arch Dis Child. 2001;84(3):212-7.

19. GODFREY K M, BARKER D J. Fetal nutrition and adult disease. Am J Clin Nutr. 2000;71(5 Suppl):1344S-52S.

20. Le Clair C, Abbi T, Sandhu H, Tappia PS. Impact of maternal undernutrition on diabetes and cardiovascular disease risk in adult offspring. Can J Physiol Pharmacol. 2009;87(3):161-79.

21. Henriksen T. Foetal nutrition, foetal growth restriction and health later in life. Acta Paediatr Suppl. 1999;88(429):4-8.

22. Townsend R, Khalil A. Fetal growth restriction in twins. Best Pract Res Clin Obstet Gynaecol. 2018;49:79-88.

23. Gao Y, He Z, Luo Y, Sun H, Huang L, Li M, et al. Selective and non-selective intrauterine growth restriction in twin pregnancies:high-risk factors and perinatal outcome. Arch Gynecol Obstet. 2012;285(4):973-8.

24. Raia-Barjat T, Prieux C, Gris JC, Chapelle C, Laporte S, Chauleur C. Angiogenic factors for prediction of preeclampsia and intrauterine growth restriction onset in high-risk women:AngioPred study. J Matern Fetal Neonatal Med. 2019;32(2):248-57.

25. Audette MC, Kingdom JC. Screening for fetal growth restriction and placental insufficiency. Semin Fetal Neonatal Med. 2018;23(2):119-25.

26. Cirronis M, Vezzoni GM, DePaulis NG, Capra ME, Biasucci G, Locatelli CA. Neonatal abstinence syndrome and intra-uterine growth restriction secondary to maternal antimigraine drug abuse. Basic Clin Pharmacol Toxicol. 2021;128(4):625-7.

27. Vogt Isaksen C. Maternal smoking, intrauterine growth restriction, and placental apoptosis. Pediatr Dev Pathol. 2004;7(5):433-42.

28. Walfisch A, Nikolovski S, Talevska B, Hallak M. Fetal growth restriction and maternal smoking in the Macedonian Roma population:a causality dilemma. Arch Gynecol Obstet. 2013;287(6):1131-6.

29. Kelishadi R, Haghdoost AA, Jamshidi F, Aliramezany M, Moosazadeh M. Low birthweight or rapid catch-up growth:which is more associated with cardiovascular disease and its risk factors in later life? A systematic review and cryptanalysis. Paediatr Int Child Health. 2015;35(2):110-23.

30. Fanaro S. Which is the ideal target for preterm growth?. Minerva Pediatr. 2010;62(3 Suppl 1):77-82.

31. Ortiz-Espejo M, Pérez-Navero JL, Olza-Meneses J, Muñoz-Villanueva MC, Aguilera-García CM, Gil-Campos M. Prepubertal children with a history of extra-uterine growth restriction exhibit low-grade inflammation. Br J Nutr. 2014;112(3):338-46.

32. De Boo HA, Harding JE. The developmental origins of adult disease (Barker) hypothesis. Aust N Z J Obstet Gynaecol. 2006;46(1):4-14.

33. Bloomfield FH, Oliver MH, Harding JE. Effects of twinning, birth size, and postnatal growth on glucose tolerance and hypothalamic-pituitary-adrenal function in postpubertal sheep. Am J Physiol Endocrinol Metab. 2007;292(1):E231-7.