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Adverse Pregnancy Outcomes and Cardiovascular Risk

Adverse pregnancy outcomes (APOs) are increasingly recognised as significant female-specific risk factors for cardiovascular disease (CVD). Conditions such as pregnancy-induced hypertensive disorder, preterm birth, foetal growth restriction, placental abruption, and stillbirth have been linked to an elevated risk of both atherosclerotic and non-atherosclerotic CVD. While the underlying mechanisms remain unclear, shared genetic and pre-pregnancy lifestyle factors are a leading hypothesis. Studies suggest pathophysiological similarities, including endothelial dysfunction and inflammatory activity, between APOs and CVD.

If genetic and environmental predispositions contribute to both APOs and CVD, then sisters of affected women—who share familial and environmental factors—may also face an increased CVD risk, even without experiencing APOs themselves. However, this connection has not been thoroughly investigated.

A new study explores the role of shared familial factors by assessing CVD risk in sisters of women with APOs compared to unrelated APO-free women, as well as conducting within-family analyses. The study included primiparous women without prior CVD who had singleton births in Sweden between 1992 and 2019. Participants were categorised into three groups: women with at least one APO(165,628), their APO-free sisters (60,769), and unrelated APO-free women (992,108). All participants were followed until 2021 to assess major adverse cardiac events (MACE) and its components: ischaemic heart disease, heart failure, and cerebrovascular events.

Over a median follow-up of 14 years, women with APOs had higher CVD rates than APO-free women. APO-free sisters also showed an increased risk of major adverse cardiac events, heart failure, and cerebrovascular events compared to unrelated APO-free women, though no significant increase was observed for ischaemic heart disease. Within-family analysis showed lower CVD rates in APO-free sisters compared to their APO-exposed counterparts, except for cerebrovascular events, where no significant difference was found.

Before their first pregnancy, APO-exposed women and their APO-free sisters had similar CVD prevalence. However, after delivery, APO-free sisters had a higher CVD risk than unrelated APO-free women, though lower than their APO-exposed sisters. CVD risk increased rapidly in APO-exposed women post-pregnancy, whereas APO-free sisters exhibited a more gradual rise.

The study revealed variations in CVD risk across different APOs and CVD subtypes. Notably, cerebrovascular event risk was elevated in APO-free sisters, while no increased risk was observed for ischaemic heart disease (IHD). This may reflect different genetic and environmental contributions to stroke versus coronary disease. Furthermore, the study found that APO-free half-sisters had a higher heart failure and MACE risk than full sisters, suggesting that environmental and socioeconomic factors play a significant role.

These findings suggest that APOs may act as a catalyst for CVD in genetically or environmentally predisposed individuals, potentially due to inflammation and endothelial dysfunction persisting after complicated pregnancies. The results highlight the importance of integrated obstetric and cardiovascular care and suggest that both women with APOs and their sisters could benefit from targeted prevention strategies to mitigate long-term CVD risk.

APOs may act as triggers for cardiovascular events in predisposed individuals, leading to a rapid rise in CVD risk, especially in APO-exposed women and, to a lesser extent, their APO-free sisters. These findings underscore the importance of early identification and prevention strategies to reduce pregnancy complications and long-term CVD risk. Implementing tailored interventions for both women with APOs and those with a family history of APOs is crucial in mitigating cardiovascular risk in this vulnerable group.

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