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Women's cardiovascular health following a hypertensive disorder of pregnancy

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Over a quarter of parous women experience a complication of pregnancy, such as a hypertensive disorder of pregnancy (HDP, preeclampsia and gestational hypertension), gestational diabetes (GDM), foetal growth restriction (FGR) or preterm delivery. There is now substantial evidence that these women are approximately twice as likely to go on to develop cardiovascular disease (CVD) compared to women with uncomplicated pregnancies.¹

Using data from Nurses' Health Study II, Stuart and colleagues report associations between a history of a first pregnancy complicated by HDP and physician-diagnosed hypertension, hypercholesterolemia and type 2 diabetes over the course of three decades following pregnancy[ref]. They found that women who had preeclampsia in their first pregnancy had a 2.2-fold increased risk of developing hypertension, a 75% increased risk of type 2 diabetes and a 31% increased risk of hypercholesterolemia compared to women with a normotensive first pregnancy. Equivalent estimates for women who had gestational hypertension in their first pregnancy were 2.8, 65% and 36%, respectively. Results were similar when analyses were not limited to first pregnancies.

Stuart and colleagues were able to account for a wide range of pre-pregnancy risk factors including pre-pregnancy body mass index that may confound the relationship between HDP and cardiovascular risk factors. However due to the lack of relevant data, they were unable to adjust for pre-pregnancy measures of blood pressure, cholesterol and/or glucose. Therefore, the nature of the association between HDP (and/or other pregnancy complications) and CVD remains to be clarified. Do HDP *per se* contribute to the increased CVD risk; or do HDP simply identify women who have 'failed' the cardiometabolic stress test of pregnancy due to a pre-existing increased propensity for CVD. This question has practical implications. If HDP

increase CVD risk independently of pre-pregnancy cardiovascular health (by causing end organ damage for example) then prevention of such complications has the potential to reduce the burden of CVD in women. If, on the other hand, pre-pregnancy cardiovascular health is key, then prevention efforts should be aimed at young women. Establishing the nature of this relationship requires pre-pregnancy measures of cardiovascular health in addition to information on pregnancy complications and cardiovascular health after pregnancy, but pregnancy and birth cohorts typically recruit women who are already pregnant. In theory, Mendelian randomization could also be used, but to date there are no known genetic instruments for HDP.

Stuart and colleagues also find that women with a history of HDP developed CVD risk factors at an earlier age than women without HDP. This finding is important as it suggests that women with a history of HDP could benefit from undergoing cardiovascular risk assessment at an earlier age than women without HDP. Recent European and American guidelines^{2, 3} include a woman's obstetric history in the evaluation of CVD risk with a recommendation that "appropriate referral postpartum by the obstetrician to a primary care physician or cardiologist should occur so that in the years after pregnancy, risk factors can be carefully monitored and controlled."³ However, there is a dearth of evidence to inform optimal monitoring and prevention protocols. The current study by Stuart and colleagues makes an important contribution to fleshing out these recommendations. Additional prospective studies with repeat measures of these cardiovascular risk factors are needed to build on the present study findings.

The burden of cardiovascular disease is Pregnancy may provide an opportunity to identify and ameliorate this risk. The long time gap between pregnancy and CVD

events makes this an challenging area for research but Stuart and colleagues' findings strengthen the need to invest in furthering this research agenda in order to reduce the burden of CVD in women.

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