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Introduction

Preeclampsia is a life threatening pregnancy disorder, which is classically characterised by hypertension and proteinuria and complicates 2-8% of all pregnancies [1]. e pathophysiology of preeclampsia is poorly understood, however, it is o en described as a 2-stage process whereby Stage I is characterised by abnormal placental invasion and formation resulting in impaired placental perfusion. It is thought that reactive oxygen species and pro-in ammatory cytokines released from the ischaemic placenta result in oxidative stress and placental endothelial cell dysfunction [2,3]. is creates a pathophysiological state resulting in Stage II of preeclampsia with the clinical detection of hypertension, proteinuria and eventual organ damage [2].

In recent times, it has become increasingly evident that preeclampsia is no longer an isolated disease of pregnancy, but rather has a signi cant impact on the risk of subsequent maternal and paediatric cardiovascular disease [4,5]. Preeclampsia has been shown to be an independent risk factor for maternal cardiovascular disease 10 to 15 years a er the a ected pregnancy, with an increase in the risk of cardiovascular disease of similar magnitude to that of dyslipidaemia [4,6]. Furthermore, children of preeclamptic pregnancies have been found to have elevated blood pressure and increased cardiovascular risk later in life [7]. Crng[to b0.6 (p elTw sord5 (Fuhighlight)0.6 (plow)0.6 (pl prlslar)0.6 (diseas15 yar)0.s 0 oid)0.6 (pr* (one (have)0.5 (be

Recent studies have demonstrated that in preeclamptic placentas there is increased expression of this catabolyzing enzyme, suggesting elevated degradation of active vitamin D in these placentas as compared to healthy placentas [3]. Furthermore, researchers have found reduced expressions of VDR and DBP in preeclamptic placentas as compared to normal placentas, providing direct evidence of disrupted vitamin D metabolism in the preeclamptic placenta [3].

Although the exact molecular mechanisms by which vitamin D de ciency a ects the risk of developing preeclampsia is yet to be determined, there are a number of potential avenues by which they are hypothesised to occur. Reduced placental perfusion during Stage I may result in the placenta producing substances, including pro-in ammatory cytokines that initiate the ensuing multi-system sequelae characterising Stage II of preeclampsia [17,19]. Pro in ammatory

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