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PreventAble

Lifetime prevention must start before conception

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Introduction

Cardiovascular (CV) disease remains the world's leading killer, and much of preventive cardiology still unfolds as an urgent race to patch up damage already done. Yet the truth is plain: by the time traditional risk factors manifest in adulthood, or even as hypertension in pregnancy, the roots of disease reach back decades—often to the underestimated periods before and during pregnancy itself. While major reports and commissions have slowly shifted language from 'late-stage intervention' to 'life-course approach', this evolution in vocabulary has not translated into genuine, actionable shifts in timing. Most paradigms still treat primordial prevention as an afterthought, and when addressed at all, it is too often confined to modifiable adult lifestyle. We are missing the earliest, and potentially most transformational, windows of opportunity, particularly the periods just before conception and throughout gestation.[1,2]

Lifetime prevention: beyond adult risk factors

The reality is clear and becoming unavoidable. Pre-conception health status in mothers—body mass, blood pressure, glycemic control—is a powerful determinant of pregnancy complications and sets the future trajectory for both maternal and offspring CV risk.[3,4] But a similar story emerges for fathers: paternal obesity, diabetes, and smoking have been shown to affect offspring health through transmissible epigenetic alterations, with implications for lipid metabolism and vascular function among children.[5,6] These influences are not mere background noise. They represent a hardwired legacy, shaping early life biology long before clinical disease can be measured.

Yet, if these pre-conceptional factors set the stage, it is the *in utero* period that brings matters quickly to the fore. During pregnancy, even small disturbances—subclinical vascular dysfunction, metabolic perturbations, low-grade inflammation—can set in motion patterns of foetal CV programming with lifelong consequences. Maternal hypertension and pre-eclampsia do not merely complicate pregnancies but foreshadow increased risks of heart failure, ischaemic heart disease, and stroke for women years down the line. The offspring, too, are marked: babies born growth–restricted or from pregnancies riddled with placental vascular lesions are themselves on a trajectory towards higher rates of childhood hypertension, early onset metabolic syndrome, and atherosclerosis.[7–9] The circle is unbroken, and even the most sophisticated therapy can only play catch-up with biology already in motion.

Crucially, new evidence is revealing just how measurable—and how actionable—these early risk states are. The placenta emerges as a living record of both maternal and foetal vascular health. Elements classically associated with atherosclerosis, such as acute atherosis of the spiral arteries, have been repeatedly found in placental tissue from complicated pregnancies and tightly correlate with maternal CV risk up to decades later.[7–9] The ongoing PEARLS study is designed to examine whether such placental lesions act as an early signal for systemic atherosclerosis, potentially connecting pregnancy pathology directly to a woman's vascular future.[9] Epigenetic patterns and circulating markers (e.g. MicroRNAs), too, are beginning to tell a more granular story. The altered methylation seen in umbilical cord blood—reflecting not just maternal but also paternal exposures—can predict early vascular dysfunction and metabolic risk in childhood, blurring distinctions between nurture and nature and upending the illusion that risk is ever born purely of adult behaviour.[5,6,10]

Despite all this, mainstream clinical care continues to lag. Many obstetric visits still regard CV risk as largely future tense, something to address after the baby is born, while primary care focuses almost exclusively on risk scores built for middle-aged adults. This disconnect is not simply a function of 'gaps in evidence' or 'slow translation'. It is a failure of imagination and framing. By waiting until risk can be measured in conventional units—or worse, until disease presents itself—we ensure that prevention always follows exposure, never pre-empts it. Just as the dogma of 'residual risk' exaggerates the modifiability of entrenched pathology, our rhetorical embrace of the life-course model

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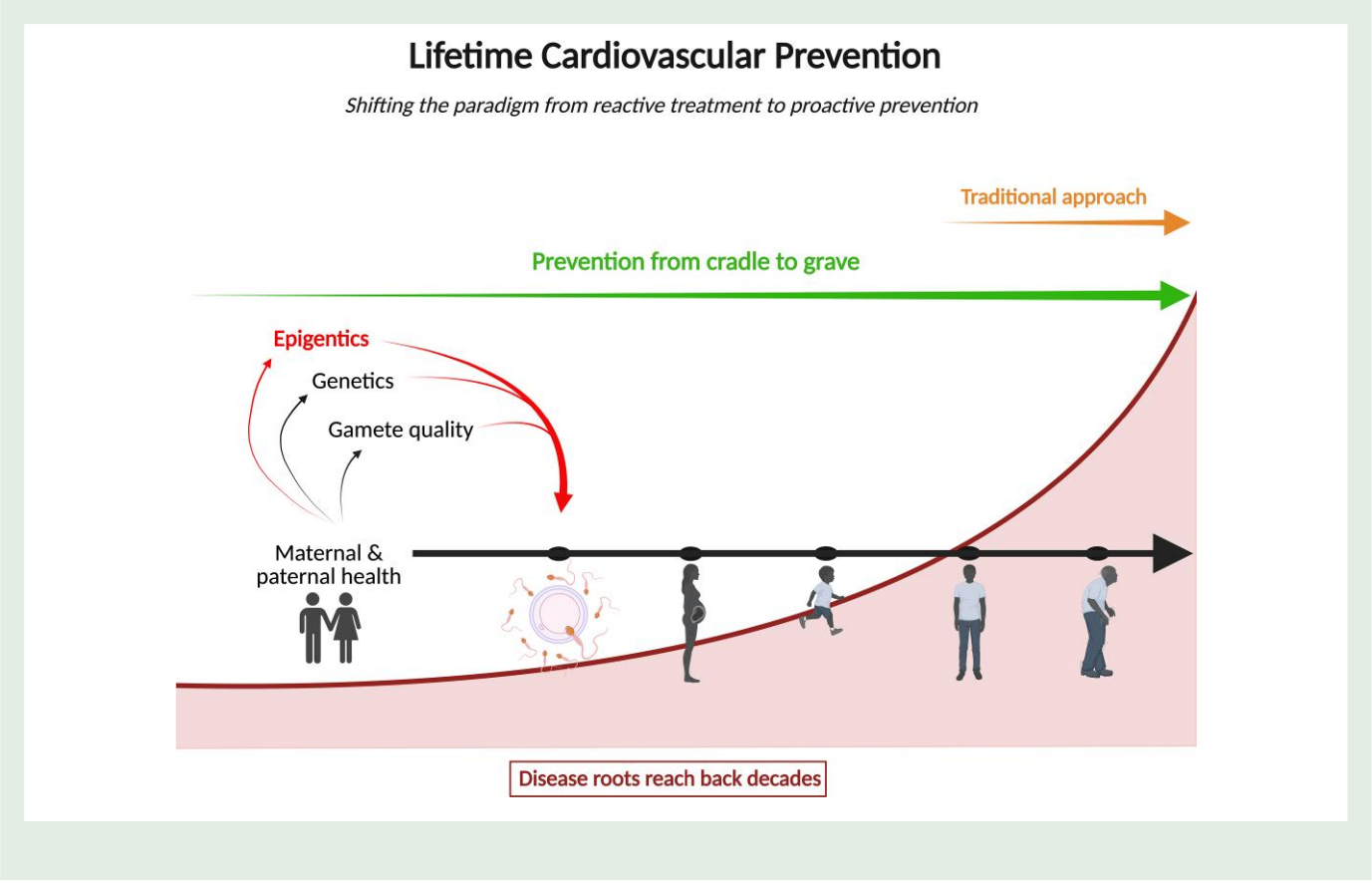

masks a reluctance to move truly upstream. Intervening during adulthood is too late; interventions limited to pregnancy often miss the window. Only by prioritizing the continuum from the period before conception through gestation do we stand a chance of shifting outcomes—for mother, child, and generations hence.

The implications for practice and policy are uncomfortable but urgent. CV risk assessment and support must become embedded in the decisions couples make before pregnancy. Both women and men should be empowered with concrete, actionable targets—CV risk factors—prior to conception, not as an afterthought nor as a footnote to routine gynecological care.[3–5] During pregnancy itself, the identification of high-risk vascular states and emerging placental biomarkers should trigger not fleeting concern, but sustained postpartum and childhood surveillance for both mother and child.[7–9] Crucially, this cannot exist solely within obstetrics; it demands direct and systematic cross-specialty collaboration, uniting primary care, cardiology, reproductive medicine, and paediatrics.

The concept of 'prevention from cradle to grave', as promoted by the European Association of Preventive Cardiology (EAPC), provides a clear framework for this ambition. Early intervention—starting before conception, continuing through gestation, and extending into the postpartum period and beyond with systematic CV risk monitoring and preventive care—is not only aligned with the EAPC's vision but should be recognized as a cornerstone of truly effective lifetime CV prevention.[1]

Conclusion

A paradigm rooted in truly lifetime prevention must resist the persistent comfort of assuming risk begins later than it actually does. The story is recursive: health and disease spiral across generations, quietly written into tissues and blood long before symptoms appear. If our goal is real prevention—not just rhetorical—our timeline must shift. Only by bringing pre-conception, *in utero* life, and the life course thereafter to the very centre of the prevention narrative, and by leveraging the tools of early risk identification now within our reach, can we hope to block CV disease at its invisible origin rather than forever treating its visible aftermath. In so doing, we fulfil the vision of 'prevention from cradle to grave' that the EAPC and leading scientific bodies continue to advance.

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Data availability

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