

Obesity in pregnancy increases risk of lifelong cardiovascular disease in offspring

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Maternal obesity impairs heart health and function of the fetus according to a new study in mice. The study, published in *The Journal of Physiology* found that maternal obesity causes molecular changes in the

heart of the fetus and alters expression of genes related to nutrient metabolism, which greatly increases offspring's risk of cardiac problems in later life.

This is the first study to show that the heart is 'programmed' by the nutrients it receives in fetal life. Changes in the expression of genes alter how the heart normally metabolizes carbohydrates and fats. They shift the heart's nutrient preference further toward fat and away from sugar. As a result, the hearts of fetuses of obese female mice were larger, weighed more, had thicker walls and showed signs of inflammation. This impairs how efficiently the heart contracts and pumps blood around the body.

The researchers from University of Colorado, US, used a [mouse model](#) that replicates human maternal physiology and placental nutrient transport in obese women. Female mice (n=31) were fed a diet with a high fat content together with a sugary drink, which is equivalent to a human regularly consuming a burger, chips and a fizzy drink (1500kcal). The female [mice](#) ate this diet until they developed obesity, putting on about 25% of their original body weight. 50 [female mice](#) were fed a control diet.

Mouse pups (n=187) were studied *in utero*, as well as after birth at 3, 6, 9 and 24 months using imaging techniques, including echocardiography and positron emission tomography (PET) scans. Researchers analyzed genes, proteins and mitochondria of the offspring.

The changes in offspring cardiac metabolism strongly depended on sex. The expression of 841 genes were altered in the hearts of female fetuses and 764 genes were altered in [male fetuses](#), but less than 10% genes were commonly altered in both sexes. Interestingly, although both male and [female offspring](#) from mothers with obesity had impaired cardiac function, there were differences in the progression between sexes; males

were impaired from the start, whereas females' cardiac function got progressively worse with age.

The sex-difference in the lasting impairments of cardiovascular health and function could be due to estrogen. Higher levels in young females may protect cardiovascular health, the protection diminishes as estrogen levels deplete as the females age. The molecular cause for the sex difference is not yet understood.

Lead author, Dr. Owen Vaughan, University of Colorado, US said: "Our research indicates a mechanism linking maternal obesity with cardiometabolic illness in the next generation. This is important because obesity is increasing rapidly in the [human population](#) and affects almost one third of women of childbearing age. By improving our understanding of the mechanisms involved, this research paves the way for treatments that could be used in early life to prevent later-life cardiometabolic illnesses, which are costly for health services and affect many people's quality of life. For example, we could offer more tailored advice on nutrition to mothers or children based on their [body mass index](#) or sex, or develop new drugs that target metabolism in the heart of the fetus."

Mice have shorter pregnancies, more offspring and different diets to humans so further studies in human volunteers would be required to extrapolate the findings to women's health. Loss-of-function studies also need to be carried out to prove this mechanism linking [maternal obesity](#) and offspring heart function and pinpoint the exact molecules responsible.

More information: Maternal obesity causes fetal cardiac hypertrophy and alters adult offspring myocardial metabolism in mice, *The Journal of Physiology* (2022). [DOI: 10.1113/JP282462](https://doi.org/10.1113/JP282462)

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