Maternal contributions to pregnancy and maternal life after pregnancy have long been the focus of research studies (Coussons-Read, 2013). This focus has begun to shift as research has established a paternal role in pregnancy success (Khoshkerdar et al., 2021). Not only are paternal contributions able to impact fetal development but have also shown an increasingly impactful role in maternal health both pre- and postpartum. In recent research, paternal contributions have been linked to increased risk for potentially life-threatening diseases for the mother and the fetus. This research has found that these conditions are usually associated with interactions between the mother and the father’s semen (Khoshkerdar et al., 2021). These serious conditions can impact the health of the fetus and even maternal health after birth. Moreover, other factors like paternal age and lifestyle can impact a healthy pregnancy.

Diet can play a sizeable role in the development of a healthy pregnancy. Certain diets, such as those with minimal protein or increased lipid consumption, can decrease the weight of the placenta and can impact the formation of a healthy embryo (Danielewicz et al., 2017). In a recent study, mice embryos injected with RNA taken from male mice on high fat diets concluded that even with no difference in weight of mature offspring there was a difference in glucose tolerance and most mice injected with high fat RNA developed glucose intolerance (Chen et al., 2016). In another study, paternal mice fed low protein diets increased seminiferous epithelium size, but no other alterations occurred to anatomy. Upon analyzing sperm, the DNA was seen to be hypomethylated. During conception, the low protein fed mice showed inadequate cell signaling and impaired implantation (Watkins et al., 2018). This lack of protein could be why the offspring had higher birthweights, higher placental weight, and increased glucose intolerance (Watkins et al., 2018). The effect paternal diet has on offspring is profound, with low protein causing increased birthweight and high amounts of fat present causing low birthweight. Both of these diets cause glucose intolerance in offspring. Future research should explore different concentrations of macromolecules and the future effects on offspring.

Paternal contributions to pregnancy outcomes are vast and can range from poor lifestyle and diet to contributing to increased risk for preeclampsia. Less than adequate paternal diets can cause glucose intolerance and negatively impact fetal birthweight. These changes can be lifelong in the offspring. In addition to diets, when paternal secretions are from an overweight father, this can cause less blastocyst development which negatively affects the chance of pregnancy. On the maternal side, increased paternal age is a risk factor for developing preeclampsia due to hypomethylation on the father’s DNA. Greater paternal age is also correlated with the onset of GDM. A maternal immune response can also be induced by the father’s seminal antigens; the severity of the reaction increases with shorter exposure to these antigens before conception, correlating with preeclampsia and low birthweight. In summation, father’s health and lifestyle can impact gestation and offspring meaningfully. Future research should expand to include a more diverse paternal population to see better reproductive success and less maternal impact such as underweight fathers, multitudes of diets, or different ailments.


