Maternal stress regulates a soma-to-germline insulin-like signalling pathway to control progeny development

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In the late 19th century August Weismann proposed that information about the environment could not pass from somatic cells to germ cells, a hypothesis now known as the Weismann barrier.However, several recent studies have indicated that parental environment can alter progeny physiology.For example, parental exposure of the nematode *Caenorhabditis elegans* to mild osmotic stress enhances progeny survival during high osmotic stress.It remains unclear if these cases represent the direct effects of stress on the germline and early embryo that in turn result in an adaptation that persists in the progeny or if somatic signals can communicate with the germline to enhance progeny survival.Here we report that *C. elegans* arrests development in response to high osmotic stress; that this arrest is caused by a decrease in insulin-like signaling; that maternal exposure to mild osmotic stress protects against progeny arrest; and that this protection is a consequence of a decrease in maternal insulin-like signaling to the germline that in turn results in an increase in the expression of the glycerol-3-phosphate dehydrogenase *gpdh-2* in the progeny embryo which protects progeny from arrest by increasing glycerol production.We conclude that in the mother somatic signaling activates an insulin-like signal transduction pathway in the germline to transduce the signal from maternal osmotic stress to progeny stress-resistance.We speculate that maternal soma-to-germline insulin-like signaling similarly transduces effects of parental environment on progeny physiology in other species and that modulation of maternal insulin-like signaling might be responsible for effects of the maternal environment on diseases that involve insulin signaling, including type-2 diabetes.