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Male germ cell DNA damage in diabetes: What is the counter strategy?

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Hyperglycemia up-regulates oxidative stress, which forms a basis for the onset of both macro- and micro-vascular diseases. In addition, hyperglycemia in males also induces hypogonadism, alterations in hypothalamo-hypophyseal-testicular hormonal axis, erectile dysfunction and infertility in both humans and animals. One of the serious effects of hyperglycemia in males is the induction of DNA damage in germ cells and spermatozoa. Hyperglycemia causes DNA single and double-strand breaks. TUNEL and DNA fragmentation assays have reproducibly shown that hyperglycemia induces DNA double-strand breaks in immature germ cells, Sertoli cells and mature spermatozoa. Moreover, ELISA and immunohistochemistry/immunofluorescence studies have shown significant increases in stage-dependent expression of 8-oxo-dG, a marker for base oxidation in the testes and also in spermatozoa. Although DNA repair mechanisms repair the damaged DNA, which seems to be associated with several molecular mechanistic pathways, including poly (ADP-ribose) polymerase, MAPKs and p53-p21 signaling, not all damaged DNA is recovered in germ cells. The cells with huge amount unrepaired DNA damage undergo apoptosis. Interestingly, supplementation of some antioxidants, for example, Resveratrol (trans-trihydroxystilbene) appears to be promising to alleviate the DNA damage in germ cells, at least in diabetic animals, but such attempts in humans have not been undertaken. Taken together, published scientific data from our laboratory and from that of other researchers indicate that diabetes-induced DNA damage in germ cells have widespread implications as regards to fertility and quality of offspring. Thus, there is a need to develop a counteracting strategy to alleviate the induced DNA damage.

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New therapeutic approaches for management of pre-diabetes

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The prevalence of pre-diabetes and Type 2; diabetes has increased considerably over the past few decades. The 2014 National Diabetes Statistics report from the CDC reports that 37% of US adults aged 20 years or older have pre-diabetes. Applying this percentage to the entire US population would yield an estimated 86 million American adults with pre-diabetes, with an estimated 50% progressing to frank diabetes within five years. This emphasizes the need to develop and implement effective preventive interventions to curtail the upward trend in diabetes incidence. Recent screening guidelines by the US Preventive Services Task Force advocated screening for pre-diabetes, given the potential for timely interventions to prevent or delay progression to diabetes. It is also been noted that the prevalence of vitamin D deficiency has increased over the past few decades, likely due to such lifestyle changes as sunscreens, diminished outdoor activities and decreased milk intake. In various areas of the US, an estimated 30% of individuals may be vitamin D deficient or insufficient. There has been considerable recent attention surrounding a potential link between vitamin D deficiency and metabolic disease. Recent studies suggest that correcting vitamin D deficiency in obese individuals with pre-diabetes reduces the likelihood of developing type 2; diabetes. Given the increasing prevalence vitamin D deficiency and pre-diabetes, there is an urgent need to implement new therapeutic interventions aimed to prevent progression from pre-diabetes to diabetes.

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