Early life and in utero factors found to influence testicular function in adulthood

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Studies over the past 20 years have suggested (though not unequivocally confirmed) that semen quality is in decline, reflected most evidently in falling sperm counts and reduced sperm motility.(1) The explanations have been controversially attributed to environmental factors (such as exposure to toxins) and to male smoking. Now, a new study based on a 20-year follow-up of one of the world's largest study cohorts, suggests that exposure to several factors in utero and in early life may also lead to reduced semen parameters in adulthood - and potentially to a decline in male fertility.

The study, which found that adverse fetal growth, exposure to maternal smoking, and a lower childhood growth trajectory were all associated with a subsequent decline in testicular function, is reported today at the annual meeting of ESHRE by Roger Hart, Professor of Reproductive Medicine at the University of Western Australia and medical director of Fertility Specialists of Western Australia in Perth.

The study was based on follow-up of the Western Australian Pregnancy (Raine) Cohort, which began in 1989-91 with the enrolment of 2900 mothers during pregnancy; their babies had regular assessment from birth, which included fetal growth measurements. Part of the 20-year follow-up of this cohort (in 423 of the men at the age of 20-22 years) involved a testicular assessment, which included measurement of testicular volume, analysis of semen quality and hormone production - as well as body composition for fat distribution.

Results showed that around one in six of the men tested had sperm parameters below the "normal" threshold recently defined by the World Health Organization (WHO). That is:

- 14.8% were below the threshold of 1.5 ml seminal volume
- 18.9% below the threshold of 39 million total sperm
- 17.5% below the threshold of 15 million sperms/ml sperm count
- 14.4% below the threshold of 32% motility

In addition, a quarter of the subjects (26.4%) had sperm whose appearance (morphology) did not meet the WHO's acceptable criteria.

When these results were correlated with the earlier fetal growth assessments, being consistently small in utero was associated with a significantly greater chance of having a sperm assessment within the lowest quartile of all the men assessed; men with good intrauterine growth were less likely to be in this lowest quartile of sperm production in adulthood. Being exposed to their mothers' smoking (18.6% of men) was also associated with lower sperm production.

Increased testicular volume was correlated with childhood growth, height and total lean body mass.

Commenting on the results, Professor Hart proposed that poor fetal growth, exposure to maternal smoking, poor childhood growth patterns, increased fat deposition in adolescence, and smoking and drug use in adulthood may ultimately lead to reduced semen parameters. Hence, public health measures to address these influences may help to reduce these risks in the future. Professor Hart said: "The main message from our study is that to reach adulthood with the best possible testicular function a man should not be exposed to his mother's smoking, should have good fetal growth and, in childhood and through adolescence, should be 'appropriately grown' - that is, neither underweight nor

overweight, and as an adult should not smoke or take drugs."

On the question of environmental "endocrine disruptors" as an explanation for a decline in semen quality, Professor Hart added: "The extent of the risk posed by environmental endocrine disrupters is still unclear, but some researchers do attribute the perceived decline in sperm counts to these chemicals within the environment. We do not have any evidence to suggest such a link in our study, but we do intend to measure the fetal exposure to endocrine disrupting chemicals from maternal blood that was stored in 1990, prior to the study recruits' birth, and to relate these chemical exposures to the men's semen counts in 2012-3."

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