

# Fetal and postnatal environmental exposures and reproductive health effects in the male: recent findings

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As Sharpe and Skakkebaek (1) have convincingly argued, there are now substantial data supporting the hypotheses that: [1] some aspects of human male reproductive health are deteriorating (at least in some parts of the world), and [2] these declines are associated with early exposure to a range of hormonally active xenobiotics. Testing these hypotheses are challenging because many consequences of early exposures—including impaired fertility—are not observable until adulthood. But several new results are helping to make these links.

First, studies in three cities (Boston, MA, Copenhagen, Denmark, and Turku, Finland) demonstrate a significant secular trend in serum testosterone (2–4). The details vary somewhat, but together these studies suggest that testosterone has declined around 1% per year for the past 40 to 50 years. This decline is consistent with the reduction in sperm concentration reported by Carlsen (5) in 1992, and these two trends taken together increase the plausibility of a significant decrease in male reproductive function.

Second, our finding of a significant shift in the direction of a shortened (and thus less masculine) male anogenital distance following prenatal exposure to several phthalates that are known to be androgenic, and that have been shown to cause a spectrum of alterations in male genitaly (known as the phthalate syndrome) further supports the plausibility of testicular disruption in utero (6).

Third, by looking at levels of phthalates in babies' urine samples we have shown that all babies (male and female) are exposed to these ubiquitous chemicals, and that the more "baby care products" a mother applies, the higher the levels in her babies' urine (7). Although we have not examined long-term consequences of early postnatal exposure, recent data from Main, Hauser, and others (8) suggest that

it is not only prenatal exposures have the potential to alter male reproductive capacity.

These new findings, added to the already substantial literature on this subject, make the links between early exposure to xenobiotics and later challenges to fertility more plausible. Links between these exposure and impaired fertility would be further strengthened by well-conducted, large epidemiologic studies that prospectively assess the later reproductive performance of pre- and perinatal exposures to these ubiquitous chemicals.

## REFERENCES

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