Impact of Environmental Chemicals on Children's Reproductive Systems



This focus sheet is to inform policy makers, government agencies, and disease prevention programs about the potential contribution of environmental chemicals to children's reproductive health.

Abnormal Reproductive System Development is Common in Children

Abnormal development of a child's reproductive system can have harmful consequences, affecting both childhood and adult life. This focus sheet will discuss three common abnormalities: early puberty in girls, hypospadias (opening of the urethra is not at the tip of the penis), and cryptorchidism (undescended testis).

Development of the reproductive system in girls and boys occurs in several steps, from the first trimester in the womb until the teenage years. Many of these steps are triggered by changes in the levels of certain hormones. If there is a problem with the normal strength or timing of these hormonal triggers, the reproductive organs may not form correctly or function properly.¹ Many genetic and environmental factors have been linked to early female puberty, hypospadias, and cryptorchidism. Chemicals and other environmental factors may alter underlying genetic risks or may directly interfere with normal development.¹

Early Puberty in Girls

On average, girls today are entering puberty at a younger age than they did in the 1940s.²

Health Consequences: Girls who go through puberty at an early age have increased risk of several health problems. Physically, premature puberty has been linked to increased risk of breast cancer, infertility, menstrual problems, and reduced adult height. Psychologically, since mental development lags behind physical development, early puberty can lead to depression as well as behavioral problems (cigarette, drug, and alcohol use) and social difficulties.³

Risk Factors: Genes appear to have a significant influence on the timing of puberty in girls. Environmental chemicals, diet, and lifestyle also play important roles.⁴ Low birth weight and childhood obesity have been linked to early puberty, while malnutrition with low protein intake can delay it.⁴ Animal studies suggest that certain chemicals may alter normal hormonal changes related to the development of the female reproductive system.⁵ For example, low doses of bisphenol A caused early puberty in mice while high doses caused late puberty in rats.⁶

Hypospadias

Hypospadias is a condition where the opening of the urethra is on the underside of the penis instead of at the tip. It is one of the most common birth defects, affecting about 1 out of 200 boys in Washington.⁷ Although normally repaired through surgery early in life, the repair is not always successful. In 2003, more than \$162 million in hospital charges were associated with about 13 thousand cases of hypospadias in the U.S.⁸

Health Consequences: Boys with hypospadias can have problems with urination and, compared to those without the defect, tend to be more timid and embarrassed, with more behavior problems and poorer social skills.⁹ Adults can have greater than normal problems with erections and ejaculation, and tend to be more fearful and inhibited than normal in seeking sexual contact.⁸

Risk Factors: Genes and environment are both thought to play a role in hypospadias. Birth to an older mother (>35 years), fertility treatments¹⁰, and low birth weight increase the risk for hypospadias.⁷ Vinclozolin and procymidone (fungicides), DBP and DEHP (phthalates), DDE (a breakdown product of the pesticide DDT), and DES are chemicals that can cause hypospadias in laboratory animals.¹¹

Cryptorchidism

At birth, 2 to 4 out of 100 boys have cryptorchidism, where a testicle has not fully descended into the scrotum.¹²

Health Consequences: While most descend without treatment within a few months, boys born with this condition have significantly greater risk of testicular cancer and infertility later in life.¹²

Risk Factors: Increased incidence of cryptorchidism in close relatives suggests a large genetic contribution.¹³ Non-genetic risk factors include low birth weight, premature birth, fetal growth restriction, and estrogenic medications (such as DES) given to the mother.¹⁴ In laboratory animals, chemicals such as DES, other estrogen medications, flutamide (a prostate cancer drug), and vinclozolin and procymidone (fungicides) caused cryptorchidism.¹⁵

Summary

Children and fetuses are exposed to many chemicals that have the potential to interfere with normal development of the reproductive system. Finding strong links between people's reproductive health problems and exposures to particular environmental chemicals has proven difficult. However, reducing exposure to the hundreds of endocrine disruptors that can interfere with proper function of the endocrine system is a reasonable preventive step to improve health that has been recommended by the American Medical Association.¹⁶

What are endocrine disruptors?

The endocrine system helps the body develop properly and stay healthy by sending and receiving numerous chemical messages (hormones) that coordinate and control many important functions. Illnesses (such as diabetes) or abnormal development of parts of the body, including the reproductive system, can occur when the system is out of balance. Chemicals that can interfere with the normal operation of the endocrine system are often called endocrine disruptors.

Some endocrine disruptors occur naturally in plants, animals, and the environment, while others are manmade. Everyone has ongoing exposure to a mixture of many different endocrine disruptors from their diet, from products in their homes, from medicines, and from the environment. It is difficult to know the overall effect from exposure to this mixture, since the amounts and types of chemicals are always changing, some of them have opposing effects, and our bodies are constantly adapting.

Evidence from studies in animals suggests that a wide variety of endocrine disruptors can affect the formation of the reproductive system if sufficient exposure occurs at critical periods of development and the endocrine system is unable to adapt.

In people, diethylstilbestrol (DES) was a synthetic estrogen-like medication given to pregnant women from the 1940s to the 1970s. It has been linked to many abnormalities in the development of the reproductive systems of the sons and daughters.

For More Information

- Birth Defects Hypospadias, CDC: www.cdc.gov/ncbddd/birthdefects/Hypospadias.html
- Exposure to Toxic Environmental Agents, The American College of Obstetricians and Gynecologists: http://www.acog.org/About_ACOG/ACOG_Departments/Health_Care_for_Underserved_Women/~/media/Committee%20Opinions/Committ ee%20on%20Health%20Care%20for%20Underserved%20Women/ExposuretoToxic.pdf
- Fertility and Infertility and the Environment, CDC: http://ephtracking.cdc.gov/showRbFertilityInfertilityEnv.action
- State of the Art Assessment of Endocrine Disrupters, 2nd Interim Report, European Union, 2011: http://ec.europa.eu/environment/endocrine/documents/summary_state_science.pdf

References

¹European Commission. (2012) http://ec.europa.eu/environment/endocrine/documents/summary_state_science.pdf

²Euling, SY et al. (2008) 121 (suppl): S172-S191.

⁴Golub, MS et al. (2008) Pediatrics 121 (suppl): S218-S230.

⁴Parent, A-S et al. (2003) Endocrine Reviews 24: 668-693. ⁵Buck Louis CM at al. (2008) Padiatrics 121 (suppl) 5102 520

⁵Buck Louis, GM et al. (2008) Pediatrics 121 (suppl): S192-S207.

⁶NTP-CERHR monograph on the potential human reproductive and developmental effects of bisphenol A. September, 2008. National Toxicology Program, US Department of Health and Human Services. NIH Publication No. 08-5994.

⁷Porter, MP et al. (2005) Pediatrics 115(4): e495-e499.

⁸Robbins, JM et al. (2007) MMWR 56(2): 25-29.

⁹Mieusset, R and Soulie, M. (2005) Journal of Andrology 26(2): 163-168.

¹⁰US CDC www.cdc.gov/ncbddd/birthdefects/Hypospadias.html

¹¹Baskin, LS et al. (2001) Environ Health Perspect 109: 1175-1183.

¹²CHEM Trust. www.chemtrust.org.uk/documents/ProfRSHARPE-MaleReproductiveHealth-CHEMTrust09.pdf

¹³Barthold, JS. (2008) Curr Opin Urol 18(4): 395-400.

¹⁴Thonneau, PF et al. (2003) J Andrology 24(2): 155-162.

¹⁵Virtanen, HE et al. (2011) Mol Cell Endocrinol 355(2): 208-220.

¹⁶The Endocrine Society. www.endo-society.org/advocacy/insider/AMAAdoptsSocietyEDCPolicy.cfm

¹⁷Diamanti-Kandarakis, E et al. (2009) Endocrine Rev 30(4): 293-342.

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