

# Rachel's Environment & Health News

## #566 - Girls Are Reaching Puberty Early

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Many girls in the U.S. are entering puberty much earlier than normal, according to a recent study reported in the journal PEDIATRICS.[1] And there is some evidence that exposure to environmental chemicals may be contributing to the phenomenon.

Current medical texts say that only 1% of girls show signs of puberty, such as breast development and pubic hair, before the age of 8.[2] But the PEDIATRICS study found that a substantial proportion of American girls have one or both of these characteristics at age 7 and that 1% of all girls now have one or both of them at age 3.

Data for the study were collected by 225 physicians in suburban practices who recorded the physical growth of 17,077 of their young female patients, of whom 90.4% were white and 9.6% were African-American.[1] The authors of the study say their sample of girls was not selected randomly and therefore may not accurately represent the entire U.S. population of female children. However, they know of no systematic bias in their sample and they believe the girls they studied are typical.

The early onset of puberty was observed in both white and African-American girls, but with significant differences between them. African-Americans showed the first signs of sexual maturity about a year earlier than whites. Previous studies had observed these racial differences, but no one has provided an explanation for them.[3] (There is some evidence that these racial differences have developed only recently. A 1944 study reportedly found no such differences.[4])

The new PEDIATRICS study found that, at age 7, 27.2% of African-American girls, and 6.7% of white girls had either breast or pubic hair development; by age 8, 48.3% of African-American girls and 14.7% of white girls had one or both of these characteristics. The study also found that 1% of whites and 3% of African-Americans had such characteristics at age 3.

The study found that the average age for onset of puberty was just under 9 for African-Americans and was 10 to 10 1/2 for whites. Current medical texts say puberty begins between the ages of 11 and 12, on average.

The authors say it is conceivable that their sample might have been biased by young girls entering puberty whose parents became concerned and sent them for medical examination. If so, they said, an equivalent parental concern should produce, in their sample, an excess of 12 year olds who show no development, but no such excess appeared in the data.

The study found that age of first menstruation has not changed. Average age of first menstruation in whites is 12.8 years and in African-Americans is 8 months earlier. This is a pattern that has held steady for 30 or 40 years, the authors say.

The principal author of the study, Dr. Marcia E. Herman-Giddens told the NEW YORK TIMES, "The reason I did this study is that in my clinical practice, I was seeing a lot of young girls coming in with pubic hair and breast development, and it seemed like there were too many, too young. But I don't think any of us expected to see such a large proportion of girls developing this early," she said.[5] Dr. Herman-Giddens is an adjunct professor of maternal and child health at the University of North Carolina (Chapel Hill) School of Public Health.

The PEDIATRICS study suggests that environmental chemicals that mimic estrogens might be involved. The authors point to a small study of 10 girls who entered puberty early as a result of exposure to hair-care products that had estrogenic properties.[6] They suggest that other well-known estrogenic chemicals, such as PCBs (polychlorinated biphenyls) should be studied to see if they are implicated in early-onset puberty.

As it happens, a very recent preliminary report indicates that PCBs and DDE (a breakdown product of the pesticide DDT) may indeed be associated with early sexual development in girls. Both DDE[7] and PCBs[8] are known to mimic, or interfere with, sex hormones.

According to the British journal NEW SCIENTIST, Dr. Walter Rogan described preliminary data at a conference on environmental estrogens in July in Arlington, Va.[9] Rogan is acting clinical director at the U.S. National Institute of Environmental Health Sciences (NIEHS) in Research Triangle, North Carolina.

According to NEW SCIENTIST, between 1979 and 1982 Rogan and his colleagues measured PCBs and DDE in blood and breast milk of hundreds of pregnant women in North Carolina. They also measured the chemicals in fetal blood collected from umbilical cords after birth. They then monitored the physical growth and maturity of 600 of the children of these women. According to NEW SCIENTIST, girls with the highest pre-natal exposures to the chemicals entered puberty 11 months earlier than girls with lower exposures. For boys, exposures to the chemicals before birth made no apparent difference in sexual development.

Rogan minimizes the importance of his data, but others say his findings are significant because few studies have ever looked at chemical effects on the offspring of exposed women, and the women Rogan studied were exposed to PCBs and DDE from normal diet and environmental sources, not from industrial accidents or other abnormally high exposures.

Is there other evidence that estrogen-mimicking chemicals could speed up the sexual maturation of mammals? At least three laboratory studies seem relevant here:

\*\* Female rats were fed a diet that contained a phytoestrogen (a naturally-occurring plant that mimics estrogen). The ovulation of their offspring was prematurely terminated --a sign that their sexual development had been speeded up by their mother's diet.[10]

\*\* Exposing immature female mice to high levels of methoxychlor stimulated them to early sexual maturity.[11] Methoxychlor is currently used in this country as a substitute for DDT which was banned in the 1970s, partly because of its estrogenic properties. The estrogenic properties of methoxychlor have become well-established in recent years, but its use continues.

\*\* Rats treated once with certain PCBs on the second or third day of life exhibited a permanent alteration in sexual development. Specifically, young female rats treated once with Monsanto's Arochlor 1221 (a PCB) achieved sexual maturity in 28 days whereas untreated controls reached sexual maturity in 42 days.[12]

The authors of the PEDIATRICS study wrote, "This study strongly suggests that earlier puberty is a real phenomenon, and this has important clinical, educational, and social implications."

As the authors of the pediatrics study hint, the clinical implications may be serious. The arrival of puberty is driven by naturally-occurring estrogenic hormones coursing through the blood stream. There is now considerable evidence that breast cancer is promoted by the presence of these same naturally-occurring estrogens. Women who go through puberty early have a longer-than-normal exposure to these estrogens and therefore may be in greater danger of getting breast cancer.[13,14]

Breast cancer now kills 46,000 American women each year and the number is steadily rising; the reasons for the rise are poorly understood but there is widespread agreement that estrogen plays a role in the disease.[15] In recent years, researchers have hypothesized that environmental chemicals that mimic estrogens may also promote breast cancer.[16]

The social implications of early-onset puberty are obvious: young

children with mature bodies must cope with feelings, urges and differences from their peers that most children are not well-equipped to handle. For many children, early pubescence may be a significant burden to bear.

--Peter Montague (National Writers Union, UAW Local 1981/AFL-CIO)

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[11] Laura M. Walters and others, "Purified Methoxychlor

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[12] Ronald J. Gellert, "Uterotrophic Activity of Polychlorinated Biphenyls (PCB) and Induction of Precocious Reproductive Aging in Neonatally Treated Rats," *ENVIRONMENTAL RESEARCH* Vol. 16 (1978), pgs. 123-130.

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[15] Eliot Marshall, "Search for a Killer: Focus Shifts from Fat to Hormones," *SCIENCE* Vol. 259 (January 29, 1993), pgs. 618-621.

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