Precocious Puberty: What is known?

Marcia Herman-Gidden’s 1997 report in Pediatrics and subsequent study of 17,000 girls, across the US:

Early development too wide-spread to be aberration

- Age of menarche has remained stable at 12.5 yrs since the 1960s
- Onset of secondary sexual characteristics (pubic hair and breast buds) is what is happening earlier:
  - 5% of white girls by age 7, 15% by age 8
  - 15% of Afr. Am by age 7, 40% by age 8
  - Too few Asian, Hispanic girls to draw conclusions, but Hispanic look similar to white
Theory #1: Weight Gain, Obesity

- High fat foods, lack of exercise
- Long been observed that (statistically), overweight girls mature earlier
- Very thin girls have later menarche
- Increase in obesity (% of overweight children doubled, from 6% to 12%, 1970-90)
- Fat cells produce leptin, a protein required for pubertal development
- Overweight girls --> more insulin circulating in blood; insulin stimulates production of sex hormones from ovaries and adrenal gland
Theory #2: Environmental Chemicals

- Hormones fed to livestock: Inclusive. These hormones break down quickly in the body.
- Chemicals which mimic estrogen: DDE (breakdown of the pesticide DDT), PCBs. Pervasive in the environment, persist for years in the body.
- Rogan’s longitudinal study: measured DDE and PCB exposure of 600 women during pregnancy and lactation. Girls with high prenatal PCB exposure were heavier at age 14 and had earlier puberty (1st stage).
- Plastics contain phthalates, estrogen mimicker
Theory #3: Sexualized Images
- Herman-Giddens, Drew Pinksy

Theory #4: Familial Stress
- Jay Belsky: Girls with distant family relationships entered puberty earlier
- Bruce J. Ellis: Confirmed with Longitudinal study (coded warm-positive, negative-coercive -- fathers made the difference)

Theory #4: Absence of Biological Father
- Experiments with prairie dogs and mice: puberty is inhibited when biological father present