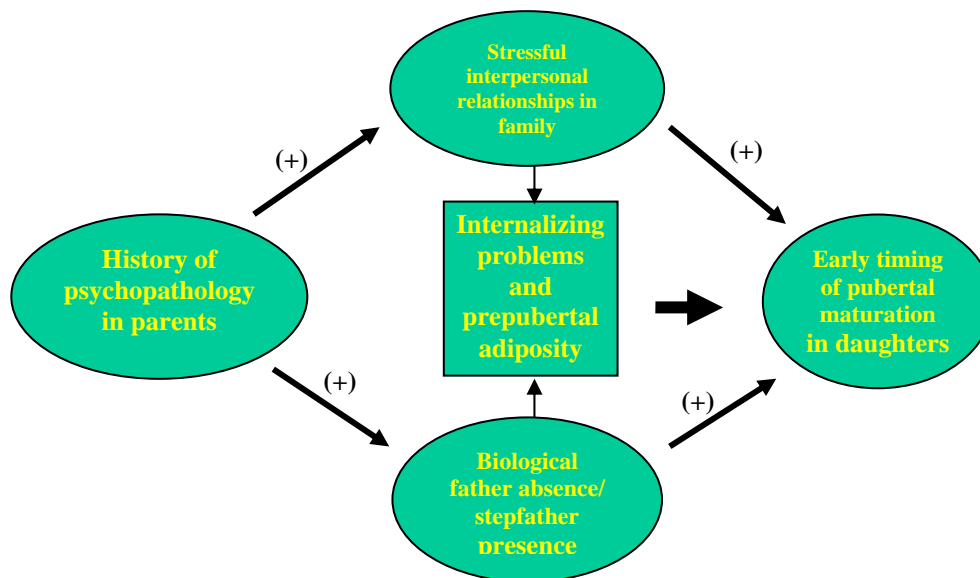


Psychosocial Studies of Girls and their Families in the Cincinnati Breast Cancer and the Environment Research Center

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Background: The psychosocial measures employed in the Cincinnati Breast Cancer and the Environment Research Center (BCERC) study represent dimensions of the social environment and behavior that may play a role in pubertal timing. **Work Performed:** We have assembled a battery of assessments to explore a hypothesis proposed by Ellis and others whereby parental psychopathology is associated with familial dysfunction (conflict, lack of cohesion and expressiveness) as well as biological father absence or emotional distance. We proposed that parental psychopathology (Center for Epidemiologic Studies Depression Scale), stressful interpersonal relationships in the family (Family Environment Scales), and father absence would be associated in turn with internalizing symptoms in girls (Behavioral Assessment System for Children-2), prepubertal adiposity, and early timing of pubertal maturation. This is illustrated in the figure presented below adapted from the work of Ellis and Garber (2000).

Figure. Psychosocial Antecedents of Variation in Girl's Pubertal Timing.



Results: In an interim cross-sectional analysis of 265 seven-year-old subjects from the Cincinnati BCERC, we used multiple regression analysis to explore associations between measures of parental psychopathology, family function, key sociodemographic factors and child psychopathology. We also examined the relationships between body mass index (BMI), Tanner staging of secondary sexual characteristics and measures of child psychopathology. Maternal depression and family conflict were strongly associated with girls' internalizing and externalizing psychological symptoms. Maternal depression, family conflict and lack of expressiveness were associated with lower scores for adaptive behaviors. Higher BMI and Tanner staging scores were also significantly associated with girls' internalizing and externalizing problems and lower scores on measures of adaptive social skills. The sociodemographic factor most strongly and independently associated with internalizing and externalizing problems was household size. Both household size and home ownership were associated with higher scores for adaptive behaviors. **Conclusion:** Measures of parental psychopathology, family function, body mass, and Tanner staging were significant predictors of internalizing and externalizing problems and adaptive behavior in girls from the Cincinnati BCERC. Future studies will take advantage of the longitudinal nature of the Cincinnati study to determine if the model presented above is supported by these data.

Psychosocial Studies of Girls and their Families in the Breast Cancer and the Environment Research Center

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Bay Area BCERC

Background: Early onset of puberty among females is a significant risk factor for numerous short-term negative outcomes in adolescence, such as initiation of substance use, early sexual intercourse, and mental health problems, and long-term negative health outcomes in adulthood, such as breast cancer. Studies have confirmed that various aspects of the environment in which a girl grows up put her at risk for early puberty. There are also various pathways through which environmental factors may impact puberty. The pathways that have received the most attention are those related to diet, nutrition, and physical activity. However, there is a growing body of evidence that suggests that other pathways, which may seem unlikely to have anything to do with diet and nutrition, may be important -- namely those pathways initiated by characteristics of psychological and social environment. We can conceptualize the impact of the social environment in several ways. First, the social environment may have an indirect effect on puberty through its influence on behaviors (e.g., dietary and physical activity) that impact pubertal timing. For example, neighborhood deprivation may increase stress levels for youth, which may lead to hormonal and behavioral changes that trigger earlier puberty. Second, there may be gene-environment interactions. For instance, if a girl has a genetic makeup that predisposes her to early puberty, this may be activated only under certain circumstances, such as stressful familial conditions. Third, the social environment may act directly on pubertal timing. Animal models suggest that maternal licking behavior among rodents (analogous to maternal warmth in humans) directly predicts pubertal timing in offspring.

BCERC: In the epidemiologic studies of the Breast Cancer and the Environment Research Center (BCERC), we have assembled a battery of measures to broadly assess socio-emotional environment. These measures include child and maternal psychopathology, aspects of the family environment, family composition, and socioeconomic status. All of the psychological and family environment measures are validated, standard measures available in English and Spanish. A summary of measures collected across the three sites will be presented. With the exception of Cincinnati, we are not gathering self-report data from the girls but rather from their primary caregivers, usually the mother. In addition, we are not obtaining information about fathers' functioning, the potential importance of which Bruce Ellis will address in his presentation.

Bay Area BCERC: In the Bay Area, we have successfully recruited 444 ethnically and socioeconomically diverse 6-8-year-old girls and their caregivers. Of these girls, approximately 80% of caregivers completed baseline measures of family and psychological functioning (i.e., Family Environment Scale, CES-D, BASC-2). All participants completed baseline measures of family composition (i.e., father residence). Descriptive statistics will be presented for the total baseline sample and by ethnicity. Interim cross-sectional analyses will also be presented.

Implications: By seeking to identify modifiable psychological and familial correlates of pubertal timing, the current research has potentially significant clinical implications for the health of young girls, which may extend into adulthood. As these data are examined over time, findings have the potential to provide more insight into causal pathways and thereby illuminate psychological and familial targets for intervention as a guide to physicians and other clinicians.

Impact of Fathers on Daughters' Age at Menarche: A Genetically- and Environmentally-Controlled Sibling Study

Jacqueline M. Tither, University of Canterbury, New Zealand
Bruce J. Ellis, University of Arizona

Girls growing up in homes without their biological fathers tend to go through puberty earlier than their peers. Whereas evolutionary theories of socialization propose that this relation is causal (i.e., that girls' sexual development is responsive to the father's role in the family), behavior genetic models contend that it is spurious. To distinguish between these competing explanations, we employed a genetically- and environmentally-controlled sibling design that examined the effects of differential exposure to family disruption/father absence within families. As specified by evolutionary causal theories, younger sisters had earlier menarche than their older sisters in biologically disrupted (N = 68) but not biologically intact (N = 93) families. This effect was superseded, however, by a large moderating effect of paternal dysfunction. Younger sisters from disrupted families who were exposed to serious paternal dysfunction in early childhood attained menarche about a year earlier than either their older sisters or other younger sisters from disrupted families who were not exposed to serious paternal dysfunction. These data indicate that early exposure to disordered paternal behavior, followed by family disruption and residential separation from the father, can substantially advance age at menarche.

The Role of the Neighborhood Environment in Children's Health

Maida Galvez, MD, MPH, Mount Sinai School of Medicine
Irene Yen, PhD, MPH, University of California at San Francisco

The objectives for this presentation include providing:

1. An overview of research on the neighborhood or "built" environment
2. A description of the methodology utilized to assess the built environment
3. A report on preliminary data findings from Year 1 data on the role of the built environment in children's health

This presentation will provide an overview of the role of the neighborhood or "built environment" in children's growth and development. The built environment encompasses the entire range of structural elements that make up the fabric of the neighborhood: housing, roads and walkways, density, transportation networks, shops, markets, parks, public amenities and public spaces. This discussion will describe how the built environment is conceptualized as a potential stressor and key research that documents associations between the built environment and children's health behaviors and health status. Preliminary data analyses from the BCERC Year 1 baseline data will be presented. Some of the methodological issues that this body of research confronts will be described, including a comparison of methodological approaches to assessing the impact of the built environment on children's diet, physical activity level and growth and pubertal development.

Why Practice the Precautionary Principle in Everyday Life

Julia Brody, PhD, Silent Spring Institute

New scientific evidence about chemicals that affect prenatal development, mimic or block hormones, damage DNA, or are linked to higher risk in health studies can make everyday decisions about “what’s safe” seem overwhelming. Consumers navigate between the quick news media “tease” about possible risks and corporate counter-moves with full-page ads assuring the safety of chemicals you never thought about before. This talk will help sort priorities by reviewing scientific evidence and hypotheses about the links between environmental pollutants and breast cancer. It will discuss ways to evaluate the quality of research, translate findings into precise statements of what is and is not known, and apply “strength of evidence” and precautionary standards in everyday decisions.

Research on environmental pollutants and breast cancer remains limited. Most chemicals have never been tested for breast cancer effects, and only a small fraction of chemicals that *do* cause mammary gland tumors in animals have ever been evaluated in a human breast cancer study. Nevertheless, we are beginning to see a coherent body of evidence. The strongest epidemiologic evidence so far shows higher risk in women with high PCB levels coupled with a particular genetic variation. Evidence is also emerging in some studies of PAHs, DDT, dioxin, and chlorinated solvents when researchers consider exposures at a younger age and/or diagnosis before age 50. The need to assess exposures at different stages of life, for many common chemicals, and in relation to specific types of breast cancer makes breast cancer epidemiology a challenge. Animal and cells studies can help fill the knowledge gaps. Chemicals shown to cause mammary gland tumors in animal studies include 73 that have been in consumer products or food and 35 in air pollution, so exposure is widespread. Too often, expert statements that “there’s no evidence that ‘*something*’ causes breast cancer” ignore laboratory studies of plausible biological mechanisms that could underlie human disease. The Breast Cancer and the Environment Research Centers are already making critical contributions to this field.

Given what we do know and the limits of our knowledge, many people chose a “better safe than sorry” approach when alternatives to suspect chemicals are at hand. The “innocent until proven guilty” rubric that underlies existing US policies for chemicals in most consumer products has led us to costly mistakes with lead in paint, toys, and gasoline; and with asbestos, formaldehyde, PCBs, and other hazards. What are the parallels to PBDE flame retardants, perfluorinated compounds, and plastics? What are steps we can take as individuals and as a society to reduce exposures? A variety of online resources along with model legislation, including the California biomonitoring bill and the Massachusetts safer alternatives legislation, can guide better decisions. In this centennial year since Rachel Carson’s birth, many of us have reflected on the leadership of women like her who, despite their own breast cancer diagnosis, dedicate their efforts to precautionary alternatives for the next generation.