The mammary gland is a unique tissue. Even though development begins before birth, it is the only tissue in the body that undergoes the majority of its development after birth. Because of this phenomenon, the development of the mammary gland can be influenced both in utero and during adolescent/adult life. Specific stages of mammary gland development are known to be highly susceptible to external influences. Studies in animal models have identified three "critical windows" of development: 1) prenatal stage of epithelial bud development, 2) rapid, peri-pubertal epithelial outgrowth, and 3) the latter stages of pregnancy when the gland is differentiating in order to provide nourishment to the impending offspring. Environmental chemicals have been reported to induce abnormal mammary development in animals if exposures occur during these critical windows. Early life environmental exposures also induce altered breast growth patterns or breast cancer risk in humans. Exposures that have been shown to cause abnormal development of the mammary gland include pesticides, persistent organic pollutants, xenoestrogens, dietary compounds, pharmaceuticals, radiation, and industrial chemicals such as flame retardants and surfactants. Although animal studies have clearly demonstrated impaired or insufficient lactational ability following late gestation chemical exposures, those types of effects are difficult to demonstrate in women due to multiple choices of nourishment for infants. However, both animal and epidemiological studies have reported altered pubertal timing of breast tissue development following early life environmental insults. Dioxin is perhaps the most well studied chemical in this regard, with data from animal models and humans. Because breast cancer risk has been associated with early life exposures and altered peri-pubertal development, it is important to understand how early life exposures affect mammary gland development and cancer. Although dioxin is the most well known example, other endocrine disrupting compounds have caused hyperplasia or increased mammary tumor incidence following early life exposures. Bisphenol A, atrazine, diethylstilbestrol, and genistein are some examples. It is hypothesized that early life exposures influence the rate of maturity of the gland, potentially affecting breast cancer risk later in life. In order to identify chemicals that may affect breast development and possibly breast cancer, chemical screening and toxicity tests need to evaluate effects on mammary gland development and proliferation, and additional research is needed to relate changes in gland development with carcinogenesis later in life. This abstract does not reflect NIEHS policy.