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Perinatal exposure to bisphenol A (BPA) has been shown to cause aberrant mammary gland morphogenesis and mammary neoplastic transformation, though the underlying mechanism is poorly understood. Mammary glands of 21-day-old Balb/c mice were exposed to BPA by gavage (25 μg/kg/day) during puberty for 3 weeks to determine whether mammary stem cell (MaSC) function was altered leading to increased susceptibility to tumorigenesis through a stem cell–mediated mechanism. The cover art is a stylistic representation of the quantification of immunohistochemical staining patterns of FosB and c-Fos expression of regenerated ductal outgrowths in control or BPA-treated MaSCs. Pubertal exposure to the low-dose BPA increased lateral branches and hyperplasia in adult mammary glands and altered the function of MaSC from different age groups, causing early neoplastic lesions in their regenerated glands, suggesting that MaSCs are susceptible to BPA-induced transformation. The present study is the first to show that pubertal BPA exposure altered murine MaSC gene expression and function such that they induced early neoplastic transformation. See the article by Wang and colleagues (beginning on page 445) for more information.