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5. Insects were collected from infested bins as larvae or pupae in strips of corrugated paper placed on the grain surface. The paper containing insects was returned to the laboratory and placed in jars, where adults emerged. Eggs were collected from these adults and used to start colonies on the standard laboratory larval diet of cracked wheat fortified with wheat germ, yeast, honey, and glycerol. All insect rearing and testing was done at 25°C and 60 to 70 percent relative humidity. Bioassays were conducted with Dipel, a wettable powder formulation of *B. thuringiensis* subsp. *kurstaki* containing 16,000 IU of potency per milligram of formulation. Powder was suspended in water at 5 mg/ml and serial 1:2 dilutions were prepared to provide nine doses ranging from 500 to 1.95 mg/kg when applied to larval diet at 0.1 ml/g. Each concentration of suspension was thoroughly mixed into a 30-g sample of diet. Samples of diet were treated with water to serve as controls. Samples of diet were placed in Mason jars with filter-paper caps and 50 eggs of the appropriate insect colony were added to each jar. Mortality levels were determined by counting the adults that emerged and correcting for control mortality. Three to six bioassays (replicates) were done on different generations of each colony. The LC_{50} and the slope of the dose-mortality relation were calculated for each bioassay with the probit analysis procedure of the Statistical Analysis System, SAS Institute, Cary, N.C.
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8. The colony was bioassayed periodically to monitor for changes in susceptibility. The bioassay procedure was similar to that used on the original colonies except that three replicate bioassays were done on each generation tested, and eventually the upper dose was raised to 2000 mg/kg. Data from the three replicates were pooled for calculating dose-mortality regressions.
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Androgens and Prenatal Alcohol Exposure

McGivern *et al.* (*1*) reported that altered nonreproductive, sexually dimorphic behavior is associated with exposure of rats to ethanol during development. They concluded that the results were evidence of a direct effect of ethanol on testicular or adrenal function. There have been many studies of monoamine control, direct and indirect, of neuroendocrine function (for example, inhibition of prolactin release by dopamine) which, in turn, may influence sexually dimorphic behaviors. In 1974 I reported that nonreproductive, sexually dimorphic behaviors could be altered by treating pregnant rats early during gestation with drugs that affect the synthesis or storage of such monoamine transmitters (*2*), so I am not questioning the observations of McGivern *et al.* (*1*). However, there are alternative interpretations of their results.

Caution should be used when data from other laboratories, based on studies with different experimental designs, are used to support one's own experimental design decisions or interpretation of results. For example, if other laboratories had studied the same behavioral variables and reported that cross-fostering has no effect on that measure of behavioral teratogenicity in fetal alcohol-exposed (FAE) animals, then McGivern *et al.* could use such studies to defend their decision not to remove the pups from their biological mothers at term. The early experience literature supports the possibility that residual ethanol effects (more likely withdrawal) in the dams may have affected the outcome of the experiment. Furthermore, neonatal withdrawal might also have acted alone or in concert with altered maternal behavior to contribute to or be responsible for the outcome. It would have been preferable to cross-foster half of each litter to pair-fed dams and the other half to ethanol-fed dams to clarify this issue. While perinatal birth weights are not given for experiment 1, McGivern *et al.* report a significant reduction in body weight of day-old ethanol-exposed males and females, weights being not significantly different from controls at 35 or 90 days of age in experiment 2. Even if greater malnutrition did not occur on succeeding days, when maternal or neonatal withdrawal might have been more severe, the fact that a significant weight differential existed at this early stage is problematic.

The suggestion that adrenal steroids may be responsible for at least the masculinization of adult FAE females, because others have reported increased brain and plasma levels of corticosterone in 1-day-old pups after prenatal alcohol exposure, begs the question related to the contribution of perinatal withdrawal and malnutrition and its associated stresses. We have recently reported such a potential source of epiphenomena in studies of the effects of opiates during development (*3*). We used opiate-naive subjects, rendered neonatally undernourished to an extent essentially identical to some and less severe than others exposed to opiates during development (*4*). These subjects showed significant differences in basal body temperature and in hyperthermic and behavioral responses to morphine administration later in life compared to fully nourished littermates. Similar results have been offered by others as evidence of a direct perinatal opiate effect in mature subjects whose body weights were even more severely reduced at the time of testing. While it may be necessary to include an isocaloric pair-fed group as a control, it is not a sufficient control if significant body weight differences of offspring emerged during the perinatal period, unless the data are interpreted within a framework that includes the concept that exposure to ethanol prenatally invariably leads to undernutrition or runting in offspring and that this effect is part of the fetal alcohol syndrome. Thus it may be premature to attribute to testosterone the effects observed by McGivern *et al.* until there is evidence that identical behavioral outcomes can be obtained in subjects with testicular hormone production suppressed in some other manner.

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Our work demonstrating long-term alterations in saccharin preference and maze performance of animals exposed to alcohol in utero was based on the hypothesis that an alcohol-induced inhibition of fetal testicular steroidogenesis would feminize adult male behaviors that are organizationally dependent on perinatal androgen concentrations (*1*). Both saccharin preference and maze behavior are such behaviors. Moreover, alcohol is well known to inhibit testicular steroidogenesis in adult rats and humans (*2*). Our results revealed a clear feminization of both behaviors in adult fetal alcohol exposed (FAE) males. Our pair-fed control dams received the same number of calories during pregnancy as the alcohol-fed dams, but no alcohol. Offspring from these control dams showed the normal sex differences reported by numerous investigators over the past 20 years [see

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