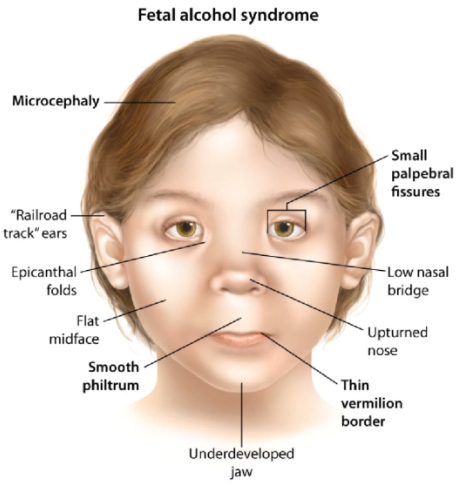
Fetal Alcohol Syndrome (FAS)

◆ What is Fetal Alcohol Syndrome?

The most notable features of Fetal Alcohol Syndrome involve the face and eyes, and include microcephaly, short palpebral fissures, an under-developed philtrum and a thin upper lip. Evidence of intrauterine or postnatal growth retardation, mental retardation or other neurologic abnormalities, and at least two of the typical facial features are necessary to make the diagnosis. Newborns with the syndrome may be irritable, with hypotonia, severe tremors and withdrawal symptoms. Mild mental retardation, the most common and serious deficit, and a variety of other anomalies may accompany Fetal Alcohol Syndrome. Sensory defects include optic nerve hypoplasia, poor visual acuity, hearing loss, and receptive and expressive language delays. Atrial and ventricular septal defects, as well as renal hypoplasia, bladder diverticula and other genitourinary tract abnormalities, may occur. Complete abstinence during pregnancy is recommended, since alcohol consumption in each trimester has been associated with abnormalities, and the lowest innocuous dose of alcohol is not known (Lewis and Woods, 1994).



◆ Paternal Contribution to Fetal Alcohol Syndrome

While it is possible that drinking fathers contribute to a wide variety of anomalies in offspring only because of 'social facilitation' (i.e. mothers drink more when in the company of men who drink heavily), there is also a growing body of preclinical evidence, reviewed in the next section, indicating that many of the other effects associated with maternal alcohol exposure are also the result of paternal alcohol exposure.

Epidemiological Studies

Epidemiologically, paternal alcohol consumption has been associated with abnormalities in offspring, such as decreases in birth weight and increases in ventricular septal defects in children — effects which are typically associated with maternal alcohol exposure. In addition, there is also suggestive evidence that hyperactivity and diminished cognitive abilities in some children are related biologically to an alcoholic father rather than to an adoptive alcoholic father. Despite these suggestive relationships, very little epidemiological research has been directed at this issue.

Paternal alcohol effects in animals: body and organ weights

Recent studies indicate that paternal alcohol exposure can result in an increase in the percentage of fetuses with the human equivalent of low birth weight. For example, a study by Wayne State University's Department of Obstetrics and Gynecology and Psychology has found an increase in the number of 'runts,' defined as individual birth weights two or more standard deviations below the mean for ad libitum control fetuses, as well as an increase in the percentage, not necessarily in

'runts,' of offspring with physical malformations, in animals whose fathers were treated with alcohol. These effects were associated with both acute and long-term alcohol treatments of adult males. These outcomes occurred when the data were analyzed on the basis of total population (total of runts and anomalies) and on the basis of litters (proportion of fetuses affected in each litter).

Organ weights and physiological effects

Other studies have found offspring of animals whose fathers were treated for several weeks with alcohol prior to breeding, have increased adrenal weights at birth, decreased spleen weights at weaning, decreased testosterone levels at sexual maturity, various behavioral effects and an increased susceptibility to Pseudomonas bacterial infection. In the latter case, the increased susceptibility was almost identical in severity to that of animals whose mothers consumed alcohol during pregnancy. The implication is that both short- and long-term treatments can affect offspring, but perhaps in different ways due to different biological mechanisms.

Behavioral effects

***Hyperactivity*.** In the 'open-field', hyperactivity is one of the more robust effects of both maternal and paternal alcohol exposure in rats. This effect in paternally alcohol-exposed animals is strain-dependent, occurring in Sprague-Dawley rather than Long-Evans rats or mice. This increased activity may be the animal counterpart to the hyperactivity seen in children with FAS and ARBDs mentioned earlier.

Learning/memory deficits. In a variety of learning tasks, learning/memory deficits have often been reported in animals prenatally exposed to alcohol. Rats sired by alcohol-treated fathers also have greater difficulty in certain learning tasks (e.g. passive avoidance). One explanation for the difficulty of learning a passive avoidance task is that the same result that causes rats to be more active in the open-field also causes them to be more active in the passive avoidance situation. This escape response is maladaptive in passive avoidance and results in an increased number of trials to criterion. Deficits in spatial learning have also been noted in rats sired by alcohol-treated fathers.

***Hyper-responsiveness to stressors*.** Children with FAS cope poorly in stressful situations. Results of testing in animals prenatally exposed to alcohol have corroborated these clinical observations and suggest that prenatal exposure to alcohol results in increased stress responsiveness mediated by enhanced corticosterone responses to stressors. Some of the abnormal behaviors of animals sired by alcohol-treated fathers can also be interpreted as exaggerated responses to stress. One such behavioral situation is the forced swim test, which we have studied in some detail. In this test, rats prenatally exposed to alcohol and those sired by alcohol-treated fathers41 exhibited decreased immobility (an indication of greater 128 Ernest L. Abel stress) in the forced swim test compared with controls.

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