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Effects of exposure to polychlorinated biphenyls during different periods of development on ethanol consumption by male and female rats

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ABSTRACT

In two experiments, male and female Sprague-Dawley rats were exposed to Polychlorinated Biphenyls (PCBs) to assess the effect PCBs, an estrogenic endocrine disrupting chemical (EEDC), would have on the voluntary consumption of alcohol. There are several EEDCs in our food that are known to increase estrogen in adolescent females. Our objective was to assess the effect that increasing estrogen, by adding the EEDC PCBs would have on volitional intake of alcohol. In Experiment 1, pregnant dams were exposed from gestational days 5–19 to a 1:1 mixture of Aroclor 1254/1260. In Experiment 2, lactating females were exposed to the same dose of 1254:1260 from postnatal days 1–21. In both experiments, a fade-in procedure was used to gradually introduce the rats to the taste of alcohol. At the end of the fade-in series all animals were given limited access (1 h/day) to a water/alcohol solution. We found that females exposed to PCBs, at two developmental periods, consumed significantly more alcohol than unexposed females and exposed and unexposed males. Results of the experiments are discussed in terms of how PCB exposure can disrupt endocrine processes (e.g., estrogenic endocrine disrupting chemicals, EEDC) that increase estrogen in females, thereby leading to increased alcohol consumption. Thus, the present findings suggest that EEDCs, such as PCBs, could contribute to the increase abuse of alcohol in adolescent females.

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Alcohol use among young females (9–14 year olds) has increased and young girls are drinking alcohol at the same rate as young boys (SAMSHA, 2015), suggesting that the gender-gap in consumption of alcohol for boys and girls has dramatically narrowed. Lynch's data (2006) showed that boys and girls were initiating use of alcohol at approximately the same age. Data from the United States (e.g., Keyes et al., 2011; White et al., 2015) and Europe (e.g., Bratberg et al., 2016) clearly shows the narrowing gender gap in alcohol use in adolescents. Increased use of alcohol in young girls and adolescents can set the stage for development of an alcohol use disorder. Dawson and Archer (1992) stated that early use of alcohol or drugs is a predictor of alcohol and drug use later in life, and Keyes et al. (2011) found women in recent cohort studies are drinking more heavily than women in earlier cohort studies. There is ample data suggesting that Keyes' et al. (2015) findings are related to the fact that young girls and adolescent girls are drinking as much as boys (White et al., 2015). This is not limited to the United States, for example, young women in the United Kingdom (Plant, 2008) are engaging in binge drinking more than their male counterparts. Increased alcohol use in young females can have very detrimental effects on women's health because women take less time than men to progress from starting the use of a substance such as alcohol to dependence on that substance (Lynch, 2006; Becker and Hu, 2008), and women suffer more of the physiological harmful effects from excessive drinking (Lenz et al., 2012).

Explaining the increased use of alcohol in young females is difficult because one must take into consideration the changing roles of females in our society, differences in the influence peers have on adolescent males and females, and differences in the role that affective disorders play in the initiation of alcohol use (Kuhn, 2015; Schulte et al., 2009; Spear, 2000, 2013; Windle et al., 2008). According to Becker et al. (2012), current attitudes towards drinking in young women have changed to being more acceptable and this could serve as an incentive for drinking among females. For example, in the United Kingdom and Italy where female alcohol use

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is considered acceptable there is more binge drinking in 15–16 year old girls relative to boys (Healey et al., 2014; Bartoli et al., 2014). In the United States, binge drinking among was reduced in 12–20 year old males, but a comparable reduction was not found in females in this age group (Grucza et al., 2009). Becker et al. (2012) stated "when social conditions allow for easy access to a drug of abuse, women are more likely to escalate use to addiction than men." (p. 17).

Estrogen may be a contributing factor for women's increased susceptibility to the effects of alcohol. That is, according to Carroll et al. (2004) and Carroll and Anker (2010) estrogen facilitates drug seeking behavior, while progesterone reduces drug seeking behavior. Their review clearly outlined sex differences, with females exceeding males, in initiation, escalation and relapse of drug use. Estrogen has also been shown to be related to increased use of stimulant drugs (Lynch, 2006) and alcohol use in women (Muti et al., 1998; Lenz et al., 2012; Hudson and Stamp, 2011). Hudson and Stamp (2011) suggested that estrogen mediated release of dopamine (DA) is a facilitating factor for drug use in females, and the release of DA is a result of one and/or two mechanisms. The first is related to estrogen inhibition of GABAergic neurons that synapse with DA terminals, thus suppressing inhibition of DA neurons and increasing the quantities of DA to be released. The second mechanism may be related to estrogen downregulating D2 autoreceptors. Downregulating D2 autoreceptors reduces DA reuptake and allows higher concentrations of extracellular DA to remain in synapses. It is well established that ingesting ETOH causes an increase in dopamine (Di Chiara and Imperato, 1985; Budygin et al., 2001), and after crossing the blood-brain barrier, alcohol affects many neurotransmitters including those for GABA, glutamate, serotonin, dopamine, and endogenous opiates. The data suggest that alcohol, in the presence of estrogen, agonizes dopamine release and inhibits the reuptake of dopamine. However, does this relationship help us understand the convergence that we see in initiation and use of alcohol in adolescent females? According to a model proposed by Lenz et al. (2012) early exposure to sex hormones may help explain some the relationship between the noted earlier consumption of alcohol in females. Their model suggested that early exposure to sex hormones, possibly through exposure to estrogenic environmental endocrine disrupting chemicals (EEDCs) may be a contributing factor. EEDCs are persistent organic pollutants (POPs), some of which have a long half-life like polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBB) and organochlorine pesticides (e g. DDT). EEDCs interfere with the endocrine system by altering hormone actions through the estrogen receptor and affecting synthesis of estrogen or receptor binding (Toppari et al., 2006). EEDCs are structurally similar to estrogen and this allows them to bind and activate estrogen receptors. Furthermore, EEDCs have been shown to be related to precocious puberty, earlier menarche, and earlier pubic hair stage in pubertal girls (Paris et al., 2013; Roy et al., 2009) suggesting that children are exposed to higher levels of sex hormones and at an earlier age. The EEDCs, by increasing estrogen in young girls, may cause an organizational neuroadaptation and these neuroadaptations may sensitize the brain to the reinforcing effects of alcohol (Lenz et al., 2012). In utero, perinatal, and childhood exposure to EEDCs could very well set the stage for young girls today, relative to girls in the 1960s and 1970s, to experience a more intense pleasurable effect from alcohol because of increased release of DA when they consume alcohol.

The Endocrine Disrupting Chemicals (EDCs) have been defined as "an exogenous [non-natural] chemical, or mixture of chemicals, that interferes with any aspect of hormone action" (Zoeller et al., 2012). There are several possible routes of human exposure to EDCs such as through the foods they eat, the water they drink, through water from plastic bottles and plastic baby bottles, and through inhalation. Because EDCs readily cross the placental barrier (Gore et al., 2015) fetal exposure is common. Some EDCs are persistent and build-up over time in blood, fat (EDCs are fat soluble), and other tissues. According to Gore et al. (2015), a person's body burden of EDCs reflect both recent contact with EDCs; and past exposure to persistent EDCs such as PCBs. Gore et al. (2015) also stated that EDCs that are estrogenic (e.g. EEDCs) can affect an organism at very low concentrations and additional exposure to an EEDC that mimics the effects of estrogen may increase levels of estrogen to a point that it has an adverse effect on the organism and alters biological outcomes.

The experiments reported in this paper examined the effect that exposure to the EEDC PCBs during two different developmental periods had on the volitional intake of ETOH in rats. Male and female rats were exposed perinatally or through lactation alone. Polychlorinated biphenyls (PCBs) are a class of industrial chemicals that were mass-produced for industrial purposes from the late 1920s until their commercial production was banned in most countries, in the 1970s (ATSDR, 2000). Production of PCBs was banned because of concern that PCBs had adverse effects in humans, especially in children. PCBs are persistent in the environment and in living organisms, and they bioaccumulate. PCBs accumulate in adipose tissue and the lipid component of serum and therefore, persist in the human body for long periods of time. PCBs have been shown to alter sex hormone structure, with many congeners and hydroxylated metabolites having estrogenic effects. Hydroxylated metabolites of PCBs (OH-PCBs) are readily transferred from maternal blood to the fetal blood via the placenta (Meerts et al., 2004; Soechitram et al., 2004). According to (Gallenberg et al., 1990; Grimm et al., 2015; Ring et al., 1988) transplacental transfer seems to be more efficient for the OH-PCBs than for the PCBs themselves (Gallenberg et al., 1990; Ring et al., 1988). Concentrations of OH-PCBs can be present in higher concentrations (10%–30% of the PCB level) than many of the individual PCB congeners (Grimm et al., 2015; Marek et al., 2013; Hisada et al., 2013).

PCBs are metabolized in vivo to hydroxyl and sulfur compounds (Letcher et al., 2000). The hydroxylated metabolites (HO-PCBs) of PCBs have been shown to be either estrogenic or antiestrogenic (e.g. Arulmozhiraja et al., 2005; Connor et al., 1997; Moore et al., 1997; Vakharia and Gierthy, 2000), and the estrogenic HO-PCBs have been shown to bind to estrogen receptors ERα and ERβ (Ulbrich and Stahlmann, 2004). Research has shown that phenolic PCB metabolites bind to the ER, and that the PCB-ER phenol complexes will translocate into the nucleus and bind to ER response elements. In vivo results indicate that the ER-binding of phenolic PCB metabolites elicits an uterotrophic response in laboratory animals (Jansen et al., 1993; Korach et al., 1988; Connor et al., 1997; Fielden et al., 1997; Recio-Vega et al., 2013). Research has also shown that some HO-PCBs, found in blood may be significantly more estrogenic than the parent congener (see Arcaro et al., 1999; Fielden et al., 1997; Garner et al., 1998; Meerts et al., 2004). Several other studies found hydroxylated metabolites of PCBs to be estrogenic. For example, Meerts et al. (2004) found metabolites of PCBs in human blood to have strong estrogenic effects. Pregnant rats exposed from gestational day 10–16 to either Aroclor 1254 or to one of the major metabolites of PCBs found in human blood. They found maternal exposure to the hydroxylated metabolite resulted in a significant increase in of the estrous cycle length and increased estradiol/progesterone ratios. At 11 months of age they found a 230% increase in estradiol concentrations in female offspring. Additional support for the estrogenic effect of PCBs was found by Kester et al. (2000) who found several hydroxylated PCB metabolites increased estradiol bioavailabilty by inhibiting human sulfotransferace, thereby indirectly inducing estrogenic activity.

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