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Could Environmental Exposures Facilitate the Incidence of Addictive Behaviors?

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Could environmental exposures be a factor in increased susceptibility to addictive behaviors? Environmental exposures have been associated with a wide range of negative health outcomes and tissue, organ, and system effects (e.g., see reviews by Guxens & Sunyer, 2012; Kampa & Castanas, 2008; Zivin & Neidell, 2013) and may also impair neurobiological functioning (Ethier et al., 2012; Guilarte, Opler, & Pletnikov, 2012; Pagani, 2013). Recently, Volk, Lurmann, Penfold, Hertz-Picciotto, and McConnell (2012) reported associations between air pollution exposure (via proximity to a busy freeway) and one facet of neurological functionality, autism risk. This led us to consider other potentially negative impacts on neurobiological functioning. In this commentary, we suggest that environmental exposures can contribute to dysregulation of mesolimbic dopamine (DA) turnover, resulting in an increased propensity for addictive behaviors to manifest themselves. Furthermore, we suggest that increased pollution exposure could be associated with increased prevalence of addictive behaviors. We explore the potential for that possibility, and identify possible supportive outcomes observed from previously identified pollution exposures and behavioral effects research.

Mesolimbic pathways, beginning in the ventral tegmental area and leading to the nucleus accumbens (NA) have been implicated in the development of addictive behavior (e.g., Wise, 2009). Addictive behavior may operate by increasing DA turnover in limbic structures such as NA and amygdala. Any behavior, substance, or environmental context that might inhibit or dysregulate mesolimbic DA turnover may therefore, in theory, increase the potential for addiction. More specifically, among other potential mechanisms, environmental exposures may impact neurobiological systems affecting the release of glucocorticoids, which have been implicated in biological reward, thereby affecting DA release and transport (e.g.,

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Piazza & Le Moal, 1996, 1997). Facilitating sustained increase in secretion of these hormones through the hypothalamic-pituitary-adrenal axis, or otherwise inducing a hypersensitivity to their rewarding effects, could predispose one to later drug abuse through increased sensitivity to drug effects as well as counteradaptation of mesolimbic DA release. For example, prenatal stress can increase sensitivity to drug effects among offspring later in life (Lupien, McEwen, Gunnar, & Heim, 2009; Piazza & Le Moal, 1996). Drug addiction (and to some extent other addictions) has been associated with inadequate mesolimbic DA turnover (“the dopamine hypothesis;” Blum et al., 2011; Brewer & Potenza, 2008; Goodman, 2008; Koob & Le Moal, 2001; Volkow & Fowler, 2000; Volkow & Wise, 2005). This may occur prior to onset of an addictive behavior (effectively increasing vulnerability) or subsequent to repeated drug misuse (e.g., due in part to an endogenous DA compensatory response). Children with apparent decreases in neuropsychological function (e.g., lessened executive processing, increased inattention, or lessened impulse control) are relatively likely to misuse drugs and suffer from substance abuse disorders (Fishbein, 2000; Pokhrel et al., 2013). These difficulties in brain function may reflect impairments in the prefrontal cortex (PFC), an overreactive amygdala, or impairment of networks linking the PFC with the amygdala and other brain regions (e.g., diminished hippocampus function). It should be noted that all of these impacts may reflect impairment in mesolimbic (and mesocortical) DA transmission (as well as norepinephrine and serotonin transmission). Genetic vulnerability (Fishbein, 2000), and/or exposure to toxins that disrupt brain function, may also facilitate later drug abuse.

Recently, researchers have begun to investigate the effect of indoor and outdoor toxicants on neurodegenerative disorders and examined the impact of toxicants on DA function. In a population of Parkinson’s disease patients and adult controls (over 70% of whom were older than 60 years of age), Ritz et al. (2009) found that pesticides exposure affected DA transporter function and increased the likelihood of the disease. Parkinson’s disease involves SN deterioration and impact of the nigrostriatal DA pathway from the SN to the striatum. While DA impact was found, the SN–striatum pathway, in general, has not been known or asserted to be associated with addiction (even though DA pathways have overlapping projection fields). Still, there is recent evidence that the SN–striatum pathway may be involved in reward function and addiction (Wise, 2009). In other words, environmental exposure studies that suggest increased vulnerability to Parkinson’s may also provide support for hypotheses of increased vulnerability to drug abuse. Other researchers investigating the effects of environmental toxicants on DA function have also reported impacts on the nigrostriatal pathway (e.g., also see Hussain et al., 2006, on effects of cytotoxic silver nanoparticles on DA).

Morales et al. (2009) found that children exposed to nitrogen dioxide (from indoor sources such as gas stoves and heating units) in the first 3 months of life showed a decrease in cognitive function and attention symptoms up to 4 years of age. Drug addiction has also been associated with attention problems (Fishbein, 2000), suggesting that there are potentially some common downstream psychophysiological effects of pollution exposure and drug addiction, two seemingly disparate entities.

Yokota et al. (2009) investigated the effects of diesel exhaust (DE) exposure on DA turnover in male mice. They found that prenatal exposure to DE was associated with decreased DA turnover in the striatum and NA, which might result in behavioral problems (related to nigrostriatal and mesolimbic DA turnover dysfunction). This finding is consistent with the possibility that pollution (DE) can lead to addiction (also see Miller, 2000). Curiously, automobile exhaust has been reported to have been misused to achieve a psychophysiological “high” (<http://www.inhalant.org/inhalant-abuse/inhalant-types/>; accessed June 3, 2013) suggesting a scenario whereby pollutant exposure might lead to both negative acute health outcomes and destructive addictive behavior.

Kampa and Catanas (2008) reviewed research on air pollution and suggested that lead exposure can cause injury to the DA and glutamate (general excitatory neurotransmitter) systems and interfere with memory function, perhaps increasing vulnerability to Alzheimer’s disease. Likewise, Guxens and Sunyer (2012) reviewed epidemiological studies on child development and found some support for the impact of air pollution (e.g., car exhaust) on increased inattention, prevalence of attention deficit and hyperactivity disorders, and lowered academic performance. Block et al. (2012) reviewed the impact of outdoor air pollution on central nervous system function and reported findings on decreased cognitive function, decreased neuropsychological development among children in the first 4 years of life, and neuropathy-related conditions (including Parkinson’s and Alzheimer’s).

Some of the neuropsychological effects reported across these illustrative reviews may be related to later drug abuse (Pokhrel et al., 2013). However, the impact of air pollution on addictive behaviors has not been directly addressed. We located only one researcher who presented a theoretical approach that discussed the implication of environmental toxicants on addiction. Miller (1999, 2000) presented a notion she termed “toxicant-induced loss of tolerance” and argued (and provided anecdotal evidence) that people exposed to pollutants may become more susceptible to the effects of lower quantities of drugs and face greater difficulties with withdrawal. While not describing neurobiological mechanisms, her ideas may be consistent with the potential impact of toxicants on mesolimbic DA shrinkage or depletion.

Future Health Professions Research and Practice Needs

If Miller is correct, the incidence or prevalence of drug addiction might be observably higher in areas of greater pollution, after controlling for appropriate confounders (such as socioeconomic status, access to drugs, age of exposure, and population density). Studies are needed to investigate this provocative possibility. Disruption of DA neurotransmission function, whether from indoor or outdoor environmental toxicants, or from drug misuse, may lead to functionally decreased endogenous mesolimbic DA biosynthesis, or increased sensitivity to events. This could lead to a motivation to experience novelty (increase DA turnover) which, in turn, leads to drug misuse and addiction. The possibility that environmental exposures might lead to enhanced addictive propensity has implications for understanding neurophysiological processes, public health protective strategies, and social regulatory policies. If this were demonstrated, low-pollution environments might become an increased priority across a wider set of the health professions, pursued as a contributing

component of addiction prevention or treatment programs. Objective focused data are needed to address this intriguing possibility.

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