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Modeling ADHD-type arousal with unilateral frontal cortex damage in rats and beneficial effects of play therapy

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Abstract

It has been recently shown that human adolescents with Attention Deficit/Hyperactivity Disorder (ADHD) have frontal lobe deficits, especially on the right sides of their brains (Castellanos et al., 1996). ADHD is commonly treated with psychostimulants which may have adverse consequences. Hence, less invasive therapies need to be developed. In the present work, we tested the ability of right frontal lesions to induce hyperactivity in rats. We also evaluated the effects of chronic play therapy during early adolescence to reduce both hyperactivity and the elevated playfulness later in development. Play therapy was able to reduce both hyperactivity and excessive playfulness. In additional work, we found that access to rough-and-tumble play in normal animals could enhance subsequent behavioral indices of behavioral inhibition (i.e., freezing in response to a startle stimulus) that appeared to be independent of increased fearfulness and fatigue. Overall, these results suggest that (1) neonatal frontal lobe lesions can be used as an animal model of the overactivity in ADHD and (2) rough-and-tumble play therapy may be a new useful treatment for ADHD.

Keywords: ADHD; Animal models; Behavioral inhibition; Frontal cortex; Play behavior; Rats; Spontaneous activity

1. Introduction

Affective neuroscience is a new discipline which attempts to conceptualize the evolutionary nature of brain systems that generate emotional behaviors and affective states within the nervous system of both animals and humans (Panksepp, 1998a). A basic premise is that all mammals possess a variety of homologous emotional operating systems in subcortical regions of the brain, even though there are bound to be more abundant differences in higher brain areas where the amount of evolutionary divergence among species is more abundant (e.g., neocortex). One of the main roles of animal models in affective neuroscience is to clarify the basic principles by which the subcortical systems operate, which may yield practical knowledge for conceptualizing and treating emotional disorders that may arise from imbalances of the underlying system. For instance, if there are specific neurochemistries such as neuropeptides that control specific emotional tendencies (Panksepp, 1993, 1998a), various new treatments for psychiatric disorders could be envisioned (Panksepp, 2000a, 2000b, 2001). Likewise, if we understand the dynamics and long-term consequences of certain emotional processes in the brain, it is possible to envision new non-biological therapies for psychiatric problems.

This paper highlights one affective neuroscience approach by re-conceptualizing the increasingly common childhood problem called Attention Deficit/Hyperactivity Disorder (ADHD) in terms of over-activity of play urges in the nervous system. It is now recognized that mammalian brains contain fundamental systems for the generation of rough-and-tumble play (Panksepp, 1998a; Vanderschuren, Niesink, & Van Ree, 1997). Because of various lines of convergent evidence, it has been proposed that some of the undesirable impulsive symptoms of ADHD may reflect overactive playful urges in some children (Panksepp, Burgdorf, Turner, & Walter, 1997). We know from animal work that play is a well-regulated

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brain process, and that young mammals appear to have a neurobiological "need" for play that "builds up" if it is not dissipated (Panksepp, Siviy, & Normansell, 1984; Panksepp, 1993).

Although it has been widely believed that play is an experience-expectant process which governs neuropsychological maturation in children (Gross, 1898), an affective neuroscience view further entertains the possibility that ADHD symptoms may diminish if children are given more consistent opportunities for rough-and-tumble activities throughout their early development when such urges are especially insistent in the nervous system (Panksepp, 1998a, 1998b, 1998c). The present work provides a preliminary evaluation of this idea using an animal modeling approach.

To set the stage, let us consider the nature of this hyperactivity "disorder." From a prevalence of no more than 1% of the population, when this problem was first conceptualized by the British physician George Still in 1902, the average incidence has risen to the current ~5%, at least in the United States. What are the reasons for this? The likelihood that the incidence of a true neurological problem has increased during the intervening century seems more remote than the possibility that diagnostic practices have become more liberal. It is certainly possible that the high incidence of ADHD is a reflection of our increasingly regimented society as much as a medical disorder of our children. The idea that a substantial part of ADHD may reflect normal human brain/personality variation is being increasingly entertained (Hartmann, 1996; Jensen et al., 1997). Even though most people would expect that ADHD children will have demonstrable differences in their nervous systems, a critical conceptual issue is at what point differences should be deemed disorders.

The most compelling overall hypothesis right now is that ADHD symptoms tend to emerge from some type of slow development or dysfunctions of frontal lobe areas that mediate executive functions related to attention, planning ahead, and social sensitivities (Barkley, 1997; Baving, Laucht, & Schmidt, 1999). Evidence for diminished frontal lobe activity comes from measures of EEG (Baving et al., 1999; Chabot & Serfontein, 1996), brain metabolic studies (Ernst, Cohen, Liebenauer, Jons, & Zametkin, 1997) as well as MRI structural analyses (Castellanos et al., 1996). It is well known that frontal lobes normally mediate attention, helping explain why children with slow frontal lobe maturation may have poor attentional abilities (Pardo, Fox, & Raichle, 1991). It is also well known that frontal lobe damage can result in abnormal social responsivity (Damasio, 1994) and symptoms of ADHD do generally resemble those of frontal-lobe injured individuals (Shue & Douglas, 1992). The fact that ADHD seems to emerge more from right frontal deficits (Castellanos et al., 1996) makes ADHD resemblances to right-hemisphere deficits especially noteworthy (Voeller, 1986). Thus, the overall picture right now is that ADHD may reflect, at least in part, slow or diminished frontal lobe maturation, although the biological reasons for this remain unknown.

In this context, it is noteworthy that bilateral damage to the frontal cortex can lead to marked hyperactivity as well as a marked elevation of playfulness (Panksepp, Normansell, Cox, & Siviy, 1994). This may support the propositions that some of the symptoms of ADHD could be caused by a natural disposition of some children (i.e., those with diminished frontal lobe activity) to be more playful than others. In a classroom setting this could easily translate into hyperactive, inattentive, and disruptive children—the classic symptoms of ADHD.

Parenthetically, since psychostimulants are powerful play reducing drugs in animals (Beatty, Dodge, Dodge, White, & Panksepp, 1982; Panksepp, Normansell, Cox, Crepeau, & Sacks, 1987), it is possible that their ability to reduce some of the symptoms of ADHD might be due to their ability to reduce play (Panksepp, 1998b). On the other hand, since we now know that the urge to play is regulated in the life-span of animals (e.g., Panksepp et al., 1984), we might anticipate that giving children greater access to play throughout early development may help to regulate such impulsive urges, and perhaps to facilitate brain maturation, as children grow up. To put it simply, in the long term, access to lots of play during early development may help ameliorate some of the symptoms that characterize the diagnosis of ADHD (Panksepp, 1998b, 1998c). Likewise, in the short term, play may produce a brain state comparable to that induced by low-doses of psychostimulants that is more conducive to behavioral inhibition and heightened attention.

In the following experiments, we evaluated such possibilities in a presumptive rodent model. In the first experiment we determined whether unilateral right frontal lobe damage, simulating the best documented brain anatomical change in ADHD children (Castellanos et al., 1996), would amplify activity and rough-and-tumble play in young rats. It did. Further, we determined whether chronic play exposure (play therapy) would be able to decrease the levels of playful impulsivity in such animals. It did. In a second experiment, we determined whether recent access to rough-and-tumble play could increase attentional behaviors (using a simple measure of behavioral inhibition). It did. We interpret these results to highlight the likelihood that a focus on the dynamics of rough-and-tumble urges in the developing nervous system may provide special insight into the genesis and non-pharmacological treatment of ADHD-type disorders in human children.