Impacts of early intervention with fluoxetine following early neonatal immune activation on depression-like behaviors and body weight in mice

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1. Introduction

There is a great deal of evidence from human and animal studies indicating that adverse events in neonatal period can negatively affect the trajectory of normal brain development and function of physiological and behavioral systems across the life span (Korosi et al., 2011; Lee and Dammann, 2011; Pesonen and Räikkönen, 2011; Skripuletz et al., 2010; Walker et al., 2011; Zakharova, 2009). Several experimental models have proven a significant link between neonatal exposure to inflammatory agents like lipopolysaccharide (LPS) and increased likelihood of neuropsychiatric disorders in later life. In this context, multi-laboratory studies have shown that LPS-induced neonatal immune activation alters hypothalamic–pituitary–adrenal (HPA) axis activity (Nilsson et al., 2002; Shanks et al., 1995, 2000) resulting in modifications of physiological (Iwasa et al., 2010), immunological (Boissé et al., 2004), behavioral (Shanks et al., 1995), and neuroendocrine (Iwasa et al., 2009) systems in adulthood.

Previous studies have indicated that LPS exposure on postnatal days (PNDs) 3 and 5 facilitates anxiety-like behaviors in adult rats (Sominsky et al., 2011; Walker et al., 2004, 2009). However, little attention has so far been devoted to the evaluation of early postnatal inflammation impacts on depression-like behaviors in animal models. In this regard, we and others have shown that there might be an association between anxiety and depression-like behaviors (Beuke et al., 2003; Enayati et al., 2012) in which genes likely have a crucial role as well as the etiology of these two behaviors (Field et al., 2010; Kendler et al., 2007; Williamson et al., 2005). Although,