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Data Article

Prenatal alcohol exposure alters gene expression in the rat brain: Experimental design and bioinformatic analysis of microarray data

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ABSTRACT

We previously identified gene expression changes in the prefrontal cortex and hippocampus of rats prenatally exposed to alcohol under both steady-state and challenge conditions (*Lussier et al., 2015, Alcohol.: Clin. Exp. Res., 39, 251–261*). In this study, adult female rats from three prenatal treatment groups (ad libitum-fed control, pair-fed, and ethanol-fed) were injected with physiological saline solution or complete Freund's adjuvant (CFA) to induce arthritis (adjuvant-induced arthritis, AA). The prefrontal cortex and hippocampus were collected 16 days (peak of arthritis) or 39 days (during recovery) following injection, and whole genome gene expression was assayed using Illumina's RatRef-12 expression microarray. Here, we provide additional metadata, detailed

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explanations of data pre-processing steps and quality control, as well as a basic framework for the bioinformatic analyses performed. The datasets from this study are publicly available on the GEO repository (accession number GSE63561).

Specifications	
Organism/cell line/ tissue	*Rattus norvegicus* – Sprague Dawley (Charles River-derived) – prefrontal cortex and hippocampus
Sex	Female
Sequencer or array type	Illumina Rat-Ref12 beadarray
Data format	Raw and quantile normalized
Experimental factors	Prenatal treatment: control, alcohol, pairfed Postnatal treatment: saline-injected, adjuvant-injected Sample collection: day 16 post-injection (peak of arthritis), day 39 post-injection (during recovery)
Experimental features	Adult female rats from three treatment groups (prenatal alcohol exposed, pair-fed, ad libitum-fed control) were injected with saline or complete Freund's adjuvant. The prefrontal cortex and hippocampus of these animals were collected either 16 or 39 days following injection to test the effects of prenatal alcohol exposure and adjuvant-induced arthritis on gene expression.
Consent	NA
Sample source location	Vancouver, BC, Canada
Keywords	Gene expression, prenatal alcohol, adjuvant, arthritis, rat

Direct link to deposited data
http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE63561

1. Experimental design, materials and methods

1.1. Experimental design

Fetal alcohol spectrum disorder (FASD) currently affects 2–5% of children in North America, making in utero alcohol exposure one of the leading causes of neurodevelopmental disorder [9]. Prenatal alcohol exposure (PAE) adversely alters the development of the immune system, increasing the organism's susceptibility to immune and inflammatory challenges throughout life. Utilizing an adjuvant-induced arthritis (AA) paradigm, we have previously shown that PAE increases the course and severity of arthritis in female rats [14]. However, the molecular mechanisms underlying this vulnerability are not fully understood. As the prefrontal cortex (PFC) and hippocampus (HPC) are involved in neuroimmune function, alterations to their gene expression profile could result in abnormal steady-state functions and response to immune challenges. To test this hypothesis, we investigated the long-term effects of PAE on gene expression in the adult rat brain, resulting in the identification PAE-specific changes under both steady-state and challenge conditions [8]. These data were deposited into the Gene Expression Omnibus (GEO) Series GSE63561, which contains 192 gene expression microarray samples, and includes both technical replicates and analyzed samples. These samples were generated from adult female Sprague–Dawley rats from three prenatal treatment groups: ad libitum-fed control (C), pairfed (PF), and prenatal alcohol exposed (PAE). Animals were injected with either saline or complete Freund's adjuvant (CFA) to induce arthritis, and terminated at the peak of inflammation or during the recovery phase of arthritis (16 days or 39 days post-injection, respectively). The prefrontal cortex (PFC) and hippocampus (HPC) were dissected from whole frozen