Annotated Bibliography


A primary research paper that found prenatal stress decreases the level of p53 expression in the HHP Axis. This paper talks about other apoptosis genes as well like Bcl-2 and Caspase. Overall came to the conclusion that prenatal stress decreases cell turnover rate which can cause challenges physiologically and psychologically in the long term for individuals. This paper also provides some methods information. For example, this paper used restraint stress as their prenatal stress stimuli. They also used qPCR for analysing gene expression levels from RNA isolated from the fetal brain.


This paper investigated the evolutionary conservation between rat, humans, and mouse. They found that the 5’ region of the p53 gene including the promoter region showed high amount of conserveness. They also found that the regulation mechanisms for the gene are highly complex, but there is strong evidence that there is a negative regulatory element that is involved in silencing of the gene. Can be used as a piece of evidence of why I want to use the rat model for this experiment.


This paper is about how the p53 tumor suppressor protein is involved in central cellular processes. This paper provides me with background on the importance of p53 and how it acts to prevent mutation accumulation. The paper focuses on the how p53 is able to activate biochemical pathways to induce cell cycle arrest and apoptosis. Identification of these pathways gives insight to developing cancer therapies and gene therapy.

This paper investigated prenatal stress and its effect on growth rate, learning and memory capability of the offspring by using nociceptive stimulus to induce stress. This is one of the only studies that used other stress inducing mechanisms other than restraint stress. They found that using footshock stress impaired the learning and memory capabilities in adult rats but did not affect the growth rate. Since the results seem to be similar to papers using only restraint stress, I will be incorporating the footshock stress conditions into my experiment.


Some background reading on how prenatal stress can relate to behavioural and neurobiological alterations. The main point from the paper was that prenatal restraint stress can result in increases in susceptibility to diseases or other mental disorders.


This primary research paper investigated prenatal stress and its effect on 11β-hydroxysteroid dehydrogenase-2, which is involved in converting cortisol/corticosterone into inactive metabolites. They found that PS increased the DNMT1 mRNA expression and the DNA methylation levels within the HSD11B2 gene promoter region was elevated thus causing gene expression alteration. This paper is a great example for what I can do for the experimental set up.

7. Pham TD, MacLennan NK, Chiu CT, Laksana GS, Hsu L, and Lane RH.


This paper investigates how uteroplacental insufficiency can cause a marker to be expressed that is associated with adult onset diseases such as hypertension. They found that the expression of the marker is due to a decrease in p53 CpG
methylation at the promoter region which then leads to the upregulation of p53 and increase of p53 transcripts. This paper investigated the mRNA levels of the p53. This is a good example for what my experimental set up will be like.

A review paper that focuses on the adaptive stress-related processes that takes place in the hippocampus, and discusses how stress can produce individuals that have higher genetic predisposition to certain cognitive and mood disorders. This review provides a lot of background on how stress can lead to greater susceptibility at a physiological level.

This paper provides a lot of information on how p53 gene is regulated epigenetically. My main focus was how histone modification and chromatin structure effects its regulation. This paper provided the model for which I based my hypothesis on. The paper proposes that there are regions at the gene promoter in human TP53 that is highly enriched with H3K9me3.

The study investigated the effects of prenatal restraint stress on anxiety and depression related behavior in male and female Spragie-Dawley rats. They analyzed the hippocampus and frontal cortex gene expression levels and found that PS significantly increased anxiety related behavior in male but not female offspring. The paper also provided me with some information for how my experiment should be set up like how rats should be kept, and how stress conditions should be set.