

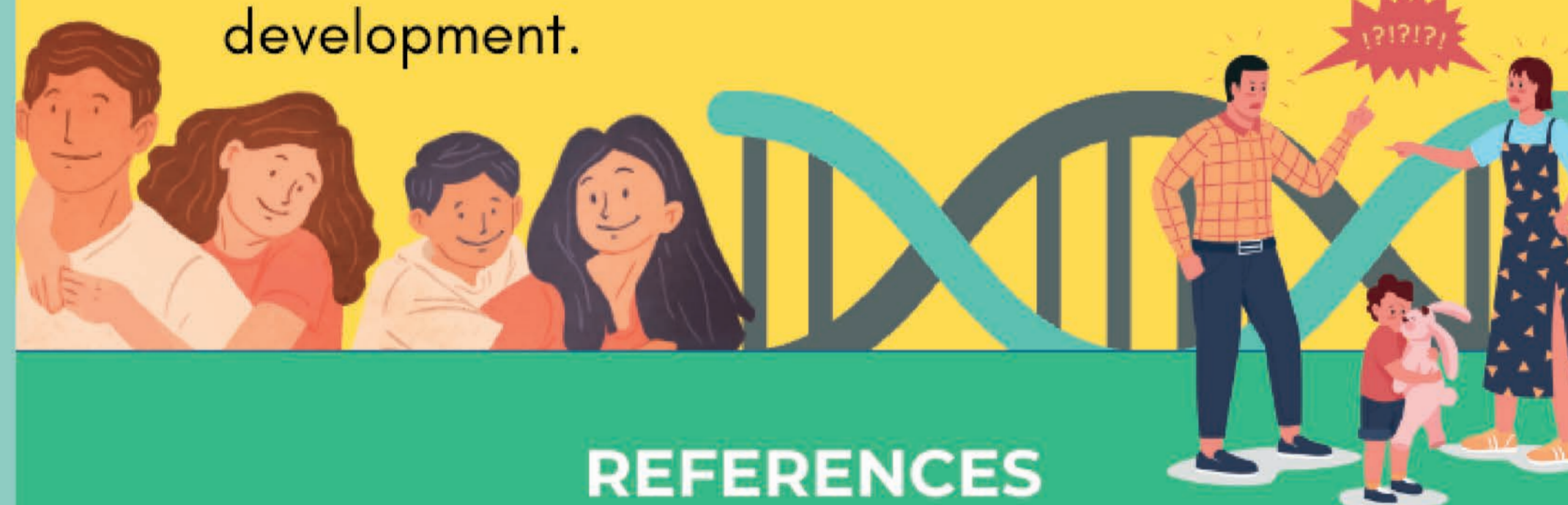
Inheriting Trauma:

An Exploration into the Epigenetic Effects of Intergenerational Trauma on Early Childhood Development

Sarah Jason-Vikram, ryshona@usf.edu

INTRODUCTION

- Intergenerational trauma is the transmission of trauma effects from one generation to the next
- Epigenetic mechanisms mediate this process, regulating gene expression without altering the underlying DNA sequence
- Trauma can cause changes in gene expression patterns that affect both the individual who experienced the trauma and their offspring
- This review explores the relationship between epigenetics, child development, and intergenerational trauma
- The findings suggest that interventions targeting these mechanisms may be a promising avenue for preventing or mitigating the effects of trauma on future generations and child development.



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Key Takeaways

What steps can be taken to effectively recognize and address intergenerational trauma, and how can this help promote healing and resilience in affected individuals and communities?

- Intergenerational trauma can be passed down through biological and environmental pathways.
- Nurturing environments and positive experiences can promote healthy gene expression.
- Combatting intergenerational trauma starts with raising emotionally and physically healthy children in a positive environment.
- Advocate for policies that support healthy childhood development.
- Remember, a **happy and healthy childhood** can break the cycle of intergenerational trauma!

The glucocorticoid receptor (GR) gene is responsible for metabolism, immune system response, and general development and is also implicated in stress response via the HPA axis. If the GR gene is turned off through methylation, the developing fetus will go on to develop a higher reactivity to stress. It also puts them at risk for future metabolic, stress, and immune disorders.

They theorize that a methyl group was added to the PIM3 gene, which silenced it. The PIM3 gene is involved in the body's ability to burn energy. This methyl group made the PIM3 gene less active across the lifetime of the child, thus slowing down their metabolism and increasing their body mass.

A neuroscientist at the University of Maryland stated that prenatal stress affects males more, and postnatal stress affects females more. This could be attributed to the protective factors of the female placenta during gestation (Bale, 2022).

A spike in S100B levels indicates a concussion (Fennel, 2021). Astonishingly, an elevated level of S100B is also found in children who have experienced emotional trauma. This 'injury,' causes children to have difficulty verbalizing their moods and emotions which manifests in the child withdrawing socially, crying excessively, participating in selective mutism, or having temper tantrums.

Childhood trauma can also affect the hippocampus, leading to increased stress and learning impairments, and the amygdala, resulting in heightened reactivity to trauma.

Studies have shown that neglectful parents came from families where they were neglected (Holland, 2021). As such, they do not have the capabilities to fulfill their children emotionally.

The impact of childhood trauma can have long-lasting effects, including an increased risk of heart disease and lung cancer, and a 20-year difference in life expectancy.

INTERGENERATIONAL TRAUMA IS HERITABLE THROUGH :

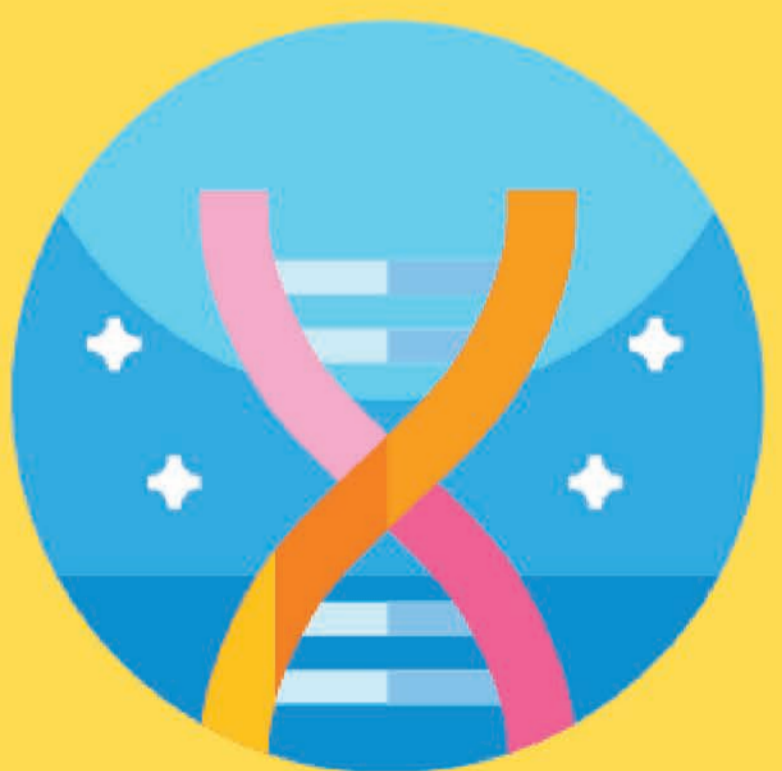
STRESS



- The **Hypothalamic-Pituitary-Adrenal (HPA)** axis is a stress response pathway.
- Childhood stress and trauma alter the HPA axis.
- Long-term **dysregulation of the HPA axis** induced by childhood stress/trauma has been associated with an **increased risk of adverse health outcomes**.
- Exposure to stress as a child can alter gene expression too.

GENE EXPRESSION AND METHYLATION

- Methylation is a **heritable mechanism** of epigenetics.
- Methylation affects the function and expression of genes, which refers to the turning on or the **turning off of genes**.
- When a **methyl group is added** to a gene, the **gene is turned off or silenced**.



PREGNANCY

- Pregnant mothers **prenatally treated for depression or anxiety** birthed infants with **increased methylation at the glucocorticoid receptor (GR) gene and increased reactivity to stress**.
- The GR gene is **implicated in stress response via the HPA axis**.
- If the **GR gene is turned off** through methylation, the developing **fetus** will go on to develop a **higher reactivity to stress**.



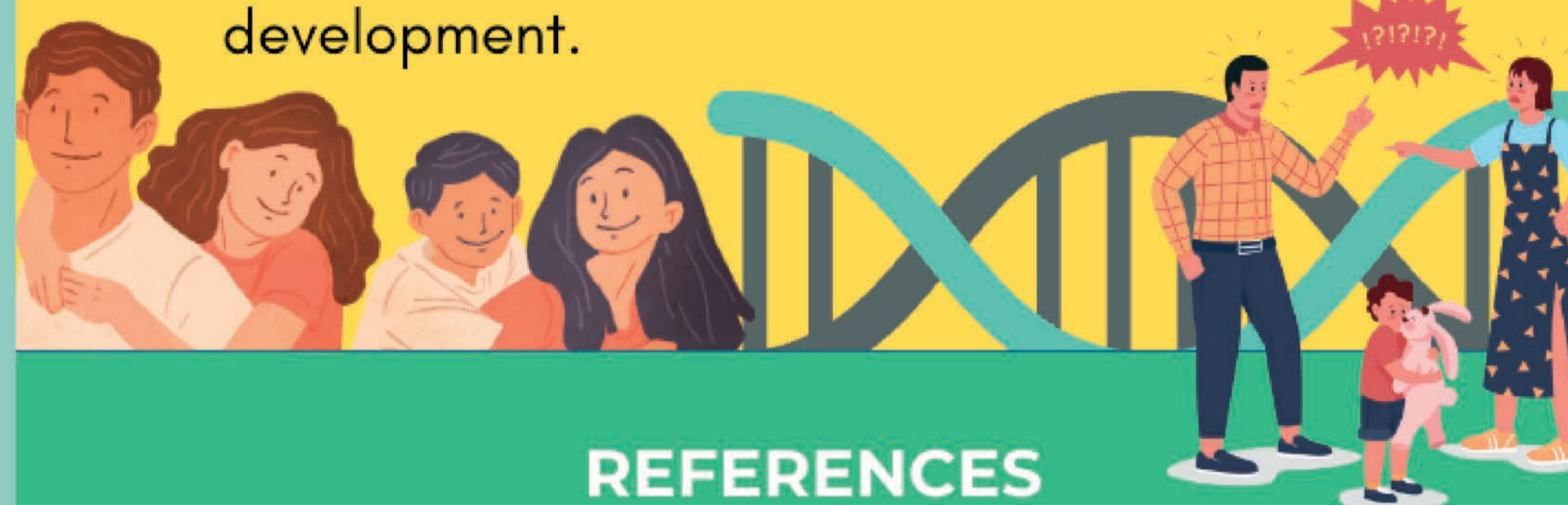
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