

# [Epigenetics mechanism that primarily occurs when a methyl](https://assignbuster.com/epigenetics-mechanism-that-primarily-occurs-when-a-methyl/)

Epigenetics Student’s NameInstitutional Affiliation Epigenetics Behavioral epigenetics is the field of study that attempts to examine the role of epigenetics in altering and shaping animal behavior. It is an experimental science that has the core objective of explaining how nurture shapes nature. Social-contextual and psychological factors can produce a variety of profound effects on plasticity, as well as brain development. Epigenetic processes like DNA methylation have the potential to regulate human’s genomes in response to stress and other environmental inputs (Ennis, 2017). Moreover, epigenetic regulation has the capability of driving long-term multi-generational trajectories in human behavioral development. In most cases, epigenetic processes usually lead to individual differences in behavior, appearance, cognition, and physiology. Various studies have recognized the fact that histone modifications and DNA methylation are essential for shaping human behavior and the learning process.

This paper’s core objective is to analyze how epigenetic processes affect DNA methylation and next generations particularly after a trauma has occurred in an individual’s life. DNA Methylation” DNA methylation is an epigenetic mechanism that primarily occurs when a methyl group is added to DNA” (Lu, Chang, & Richardson, 2015). It modifies the functions of genes, and this affects gene expression. In most incidences, the methyl groups customarily inhibit the transcription process. In human DNA, the 5-methylcytosine is found in a small section of the genomic DNA. Epigenetic gene regulation typically involves modifications or changes in DNA methylation and histones. In this context, the histones are the proteins that wrap DNA (Karpf, 2012). The epigenetic changes have the potential of influencing the growth of various neurons in the brain.

Moreover, the changes can modify the activities and the functions of the neuron in an individual’s mind. The epigenetic changes that occur in the neurons’ functions and structure can have significant impacts on an animal’s or a human’s behavior. DNA methylation is a typical example of an epigenetic mechanism that is primarily used by cells to influence and control gene expression. It is important to note that DNA methylation has the potential of changing the activities of DNA segment without altering or changing the sequence (Kundu, 2012).

The process of DNA methylation is usually essential for the normal development of an animal or a human. It is associated with processes such as X-chromosome inactivation. Other processes that have to be beneficial include the repression process and genomic imprinting.

In mammals, embryonic development demonstrates and shows the bimodal DNA methylation reprogramming that in most cases occurs in the primordial germ cells (Karpf, 2012). In such cells, the loss of DNA methylation is customarily related to the creation of new methylation pattern that is specific to female or the male gamete. After the fertilization process and the period of pre-implantation, the loss of DNA methylation is customarily essential in the setting up of the zygote’s totipotency. DNA methyltransferases or the DNMTs control the addition of methyl groups in various cells. The establishment, as well as the maintenance of DNA methylation patterns, requires three DNMTs which include the DNMT3b, DNMT3a, and the DNMT1. In specific incidences, the process may require additional enzymes that have specialized but related functions.

The enzymes that may be needed include the DNMT3L and the DNMT2. DNMT1 plays a leading role in maintaining a variety of established DNA methylation patterns. On the other hand, the DNMT3a and the 3b are crucial for mediating the establishment of the de novo and new variations of DNA methylation patterns. Apart from the DNA methylation, another process that is usually important is DNA demethylation, which refers to the removal of the methyl groups (Jost & Saluz, 2013). DNA demethylation is significant in epigenetic reprogramming of different genes and is involved in many disease mechanisms like the tumor progression. In most scenarios, the process of DNA demethylation can either be active or passive. Passive DNA demethylation occurs on synthesized DNA strands during the replication process.

Conversely, the active demethylation process primarily takes place through the removal of 5-methylcytosine. The 5-methylcytosine is vital in epigenetic modification of gene expression and phenotype (Kundu, 2012). The deficiency of 5-methylcytosine causes the decline in global DNA methylation or the DNA hypomethylation. The deficiency can also occur due to various environmental influences. The 5-methylcytosine has been viewed as a molecular marker in a variety of biological processes. The field of epigenetics has an essential role in showing how traumatic experiences can be transmitted or passed from one generation to another. Epigenetic processes in most incidences operate throughout an organism’s lifespan. Environmentally-driven DNA methylation changes typically appear long-lived and robust especially when they occur or happen during sensitive periods such as the early childhood periods or before birth (Moore, 2015).

Parental care can have a lead role in canalizing DNA methylation patterns with various generational consequences for both the behavior and the genome. Research conducted by biologists showed that adult male rodents that were raised by caring mothers in most cases had low DNA methylation levels.  Based on the research study, animals that have low DNA methylation levels usually exhibit stress resilience behavior. On the other hand, animals with high DNA methylation levels have low gene expression and in most cases exhibit increased anxiety-like behavior. The study also showed that the mother’s behavior and lifestyle primarily determined the amount of glucocorticoid receptor promoters. In other words, the study linked DNA methylation patterns with caregiver experiences (Michels, 2012).

It is worth mentioning that experience-induced changes that occur in the process of DNA methylation are avenues through the alterations of brain functions are produced by a host of childhood experiences. Maternal care plays a lead role in promoting epigenetic changes of different epicenters and genes of maternal behavior, stress regulation, addiction, and cognitive control. The epigenetic regulation of a variety of gene loci has in most scenarios been associated with a broad spectrum of childhood experiences. For instance, the antisocial or the negative behavior exhibited by girls with some history of early life exposure to sexual harassment and abuse associated with the process or the mechanism of DNA hypermethylation. Likewise, there is a link between serotonin transporter hypermethylation and the ability of isolated infants to respond to high-stress levels (Roth, 2013). Children raised or reared in organizations that provide temporary care in most cases exhibit higher forms of DNA methylation than other children. Adult and adolescent methylation levels have an essential role in acting as the central pathways for the process of cell-signaling in a person’s brain.

The levels have also been associated with the parents’ stress levels. The intrauterine environment is significant control to an individual’s growth and development. Disturbances that occur to a person during this period can have adverse effects on behavioral development. Moreover, such disorders can have long-term impacts on an offspring’s mental health. Changes that occur in the process of DNA methylation can alter or influence developmental trajectories. The stress experienced by a mother can have various epigenetic consequences on the offspring.

Significantly, restraint stress in most situations produces high amounts of DNMT 1 and 3a. A study conducted in the recent past revealed that rodents that were stressed during the critical phase of prenatal development exhibited a host of negative behaviors such as social interaction deficits, hyperactivity, memory capacity, and altered prepulse inhibition. The restraint stress experienced by different pregnant rodents like rats is related to modifications that occur to the genes that have a lead role in protecting and shielding the developing fetus from a variety of stress hormones (Roth, 2013). In utero experiences also have significant impacts on circuitry and the epigenetic gene regulation associated with motivation and addiction. Additionally, the consumption of foods rich in fats during the pregnancy period has been shown to produce children that in adulthood would prefer sugar and fat. The methylation status responds to a wide range of environmental influences or prenatal factors. For instance, children born and raised by mothers who experienced high depression, stress, and anxiety levels especially during the third trimester in most incidences exhibit increased Nr3cl methylation (Sarkar, 2013). Depression and stress experienced by a mother affect the methylation state of different genes.

The consumption of an unbalanced diet during pregnancy produces GR gene methylation. Besides, maternal tobacco and alcohol use are some of the factors that can alter the methylation, as well as the functioning of genes that have a lead role in ensuring the placenta’s growth.  Within the intrauterine environment, various epigenetic alterations may affect a person’s health and increase his or her risks of developing different disorders and diseases. The adversity that an individual experiences later in life can render long-term health and psychological costs. Alterations that occur to the DNA located in the hippocampus are indicators of chronic stress and memory loss (Roth, 2013).

For instance, repeated exposure to social instability and predatory stress typically produces rodents that exhibit memory and learning deficits, glucocorticoid abnormalities, and increased anxiety-like behavior. Social defeat stress is associated with a long-term decline in DNA methylation in animals and humans that develop social avoidance behavior. Several research studies have noted that stress-resilient and stress-susceptible male rates display behavioral differences after being exposed to mild stressors. PTSD and Epigenetics A study conducted by Rachel Yehuda revealed that the descendants or the offspring of the individuals who survived the Holocaust had a variety of stress hormone profiles compared to other individuals. It also reported that the survivors of this traumatic event have low cortisol amounts or levels, a hormone that is usually essential in helping the body to return to its normal state after a given traumatic event such as the Holocaust (Mandal, 2017).

Individuals who suffer PTSD in most incidences have low cortisol amounts. The study also noted that the survivors of this traumatic event experienced PTSD, which was later transmitted to the offspring or the descendants. It further reported that the Holocaust survivors had low amounts of an enzyme that is essential for breaking down cortisol.

Other epigenetic studies have also reported that the impacts of a variety of childhood experiences tend to be enduring and may be transmitted to the later generations.  Like the Holocaust survivors, the descendants had low cortisol amounts, and this was particularly severe in offspring whose mothers experienced PTSD after the traumatic event. The cortisol-busting enzyme has a leading role in protecting the fetus from the maternal circulating cortisol. In situations where the pregnant Holocaust survivors had low amounts of the cortisol-busting enzyme in the placenta, a higher proportion of the cortisol could enter the fetus’ body. Epigenetic changes usually serve to biologically prepare the descendants or the offspring for an environment that is primarily the same to that of the parents. Due to the low cortisol amounts and the high enzyme levels, many Holocaust survivors’ descendants would be ill-adapted or prepared to survive such conditions. In fact, the low-stress hormones would make the descendants more susceptible to high-stress levels and PTSD. Moreover, previous studies have reported that the offspring or the descendants of the Holocaust survivors are in most situations vulnerable to the various impacts of stress and have high chances of experiencing symptoms of PTSD.

The offspring may also be vulnerable to age-related metabolic syndromes including insulin resistance, hypertension, as well as obesity (Sahu, 2012). Epigenetic processes in most instances alter gene expression without producing or making significant changes to the DNA sequence. Different animal studies have revealed that epigenetic changes that usually stem from stress exposure can be transmitted to the offspring. In one of the studies, it was reported that the methylation of one of the stress-related genes or the FKBP5, was primarily correlated with depression, stress levels, and PTSD (Meyers, 2012).

In other words, the Holocaust survivors customarily exhibit high methylation of the FKBP5 compared to other individuals. Based on these observations, it is evident that parental trauma plays an essential role in acting as a contributor to the offspring’ biology. The decreased expression of GAD 1 and reelin and the increased expression of the DNMT 1 are common among individuals with schizophrenia.

Deficits of GAD 1 and reelin levels alter methylation levels. The process of DNA methylation typically shows enhanced relationships between life stress, sexual harassment and early abuse and the diagnosis of PTSD (Nigg, 2017). Genetic changes that stem from the trauma suffered from various events can be transmitted or passed on to the offspring. In other words, an individual’s life experiences can have significant impacts on the subsequent generations. A study conducted among Jewish men and women who had experienced torture or who were interned in the Nazi concentration camp showed that such experiences could be transmitted to the children. It analyzed their children’s genes and reported that they experienced increased stress disorders compared to the children who lived outside Europe during the war period. The gene changes that occurred in the children were attributed to their parents’ exposure to the Holocaust.

Based on the study, environmental influences such as stress, diet, alcohol, and smoking can affect the offspring’s genes and behavior. In animals, specific fears can be inherited. An individual’s lifestyle and the environment can interact with the genome, and this can result in epigenetic changes. It is noteworthy that the modifications or the alterations may manifest themselves in subsequent generations and later in an individual’s lifespan. For instance, human epigenetic studies have proved that a person’s risk of developing different behavioral disorders and a host of chronic diseases can increase due to their exposure to postnatal, as well as prenatal influences or environmental factors. The studies have had a lead role in showing that children born and reared during the painful periods of the Dutch famine that occurred in the early 1940s have high rates and incidences of heart diseases and eating disorders.

This can be attributed to their mothers’ exposure to famine during early pregnancy (Razin, Cedar, & Riggs, 2012). Likewise, individuals that were exposed to various famine conditions in early childhood have displayed high incidences and rates of disorders like schizophrenia. Research also shows that a mother’s exposure to various environmental factors such as pollution could have significant impacts on the child’s asthma susceptibility.

The mother’s intake of vitamin D could be essential in changing DNA methylation that in most cases influences the functioning of the placenta. Although humans’ epigenetic marks are usually more stable during adulthood, they are dynamic and can be modified by the environmental influence and lifestyle choices. It has become apparent that epigenetic effects occur in the course of an individual’s lifespan and that the epigenetic changes that occur can be reversed (Tollefsbol, 2012). The environment acts a powerful influence on disease susceptibility and epigenetic tags. For instance, air pollution has the potential to alter the methyl tags on DNA, and this can increase a person’s risk of developing neurodegenerative diseases.

Diet modifies the epigenetic tags in various ways (Roth, 2013). Low carb and a high-fat diet are essential in opening up chromatin, and this can improve a person’s mental ability through the HDAC inhibitors. Certain studies have also reported that the foods consumed by humans have specific compounds that can help in protecting individuals against cancer. The compounds have the potential of adjusting methyl marks on tumor suppressor genes and the oncogenes. ConclusionEpigenetic studies have in the past few years offered useful insights into the role or the function of DNA methylation in acting as a source for the interactions between the static genome and the environment. Whereas humans usually inherit different genes from their parents, the environment and diet can influence epigenetic changes.  The field of epigenetics has an essential role in showing how traumatic experiences can be transmitted or passed from one generation to another.

Epigenetic processes in most incidences operate throughout an organism’s lifespan. Environmentally-driven DNA methylation changes typically appear long-lived and robust. Genetic changes that stem from the trauma suffered from various events can be transmitted or passed on to the offspring.

In other words, an individual’s life experiences can have significant impacts on the subsequent generations. Postnatal and prenatal environmental factors can influence or affect an adult’s risk of developing behavioral disorders and a host of chronic diseases.