

Nutrition & Epigenetics



Definitions

Epigenetics refers to the study of changes in gene expression or cellular phenotype that do not involve alterations to the underlying DNA sequence.

Essentially, epigenetic changes are like "marks" on the genome that regulate how, when, and to what extent genes are expressed without changing the genetic code itself.

These changes can be influenced by various environmental factors, such as diet, stress, toxins, and lifestyle, which can turn genes on or off or adjust their activity levels.

Unlike mutations, epigenetic modifications are often reversible and can sometimes be passed on to future generations.

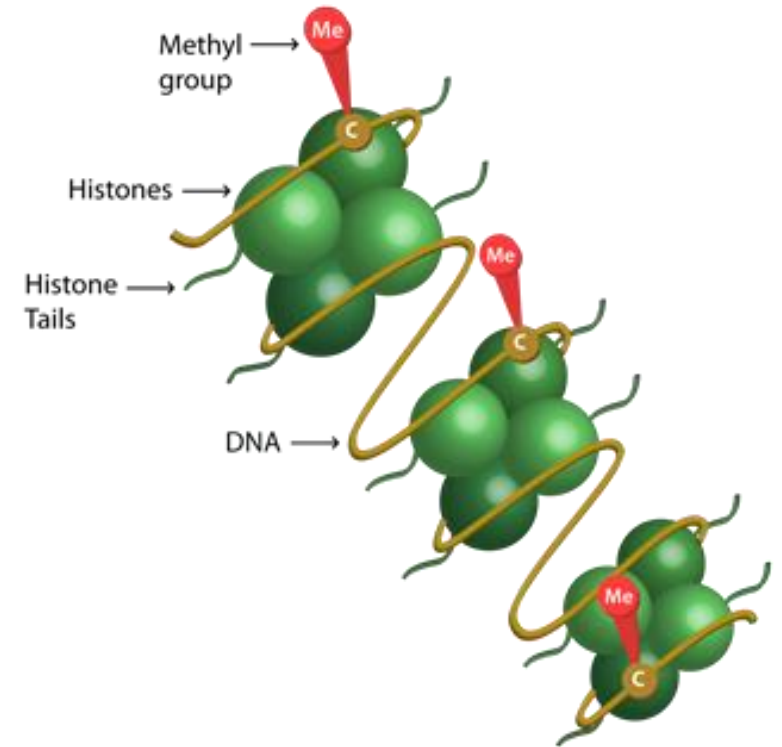
Mechanisms

DNA Methylation

This is adding a methyl group (CH_3) to the DNA molecule, usually at cytosine bases adjacent to guanine bases (CpG sites).

DNA methylation typically suppresses gene expression by preventing the binding of transcription factors or by recruiting proteins that compact the DNA, making it less accessible.

Methylation patterns can change in response to environmental factors, and they are often involved in processes like aging and the development of diseases such as cancer.



Histones

DNA is wrapped around histone proteins, forming a structure called chromatin.

Histones can undergo various chemical modifications, such as acetylation, methylation, phosphorylation, and ubiquitination.

These modifications influence how tightly or loosely DNA is wrapped around histones, thereby regulating gene accessibility.

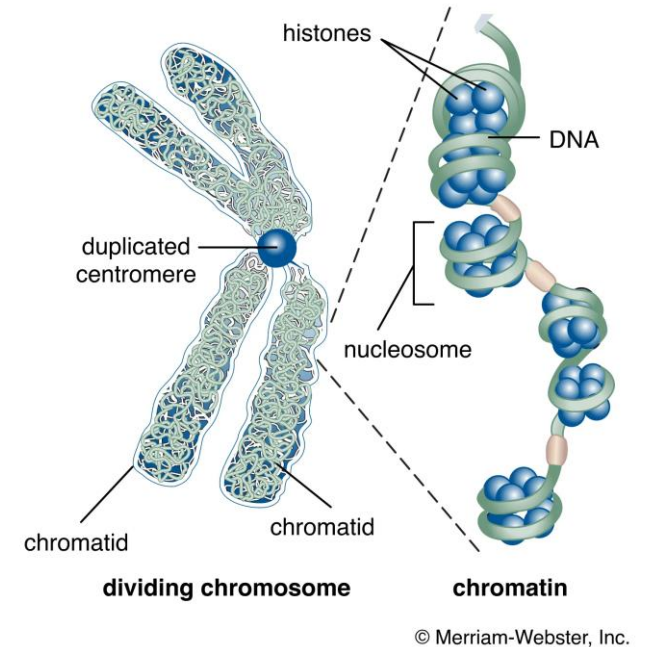
For example, acetylation of histones usually leads to a relaxed chromatin structure, allowing gene transcription, while deacetylation leads to tighter wrapping, reducing gene expression.

Acetylation (Adding Acetyl Groups): Think of this as putting "sticky notes" on specific pages of the book.

These sticky notes make the pages easier to open and read.

When the sticky notes (acetyl groups) are added to the pages (histones), it becomes easier for the "librarian" (the cell's machinery) to access and read the instructions on those pages (genes).

This means that the information in the book (genes) can be read and used more often.



Example in the Body:

When you exercise, your body may add these "sticky notes" to genes that produce energy or build muscle proteins.

This makes it easier for your cells to read these genes, helping your body to adapt and become stronger.



Deacetylation (Removing Acetyl Groups): Now, imagine someone starts removing the sticky notes, making the pages harder to open.

The book is still there, but it's more challenging for the librarian to get to certain sections. When the sticky notes are removed, the pages (genes) are tightly closed and not read as often, reducing activity.

Example in the Body: In response to stress, your body might remove the sticky notes from genes involved in inflammation control. This can reduce the production of proteins that help manage inflammation, potentially leading to prolonged inflammation and stress responses.

Histone Deacetylases (HDACs)

HDACs are like workers in the library who go around and remove the sticky notes from the book's pages. When HDACs remove these sticky notes (acetyl groups) from the histones (the proteins around which DNA is wrapped), the DNA becomes more tightly coiled, making it harder for the cell's machinery to access and read the genes. This leads to reduced expression or activity of those genes.

Example in the Body

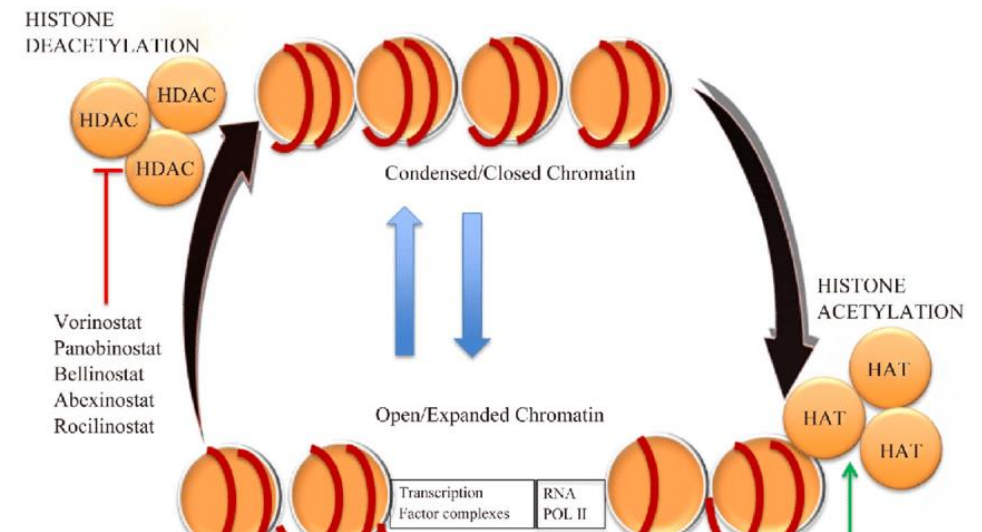
Imagine you are experiencing chronic stress. Your body might release signals that activate HDACs to remove the acetyl groups from genes that help manage inflammation. Those genes become less active with fewer sticky notes (acetyl groups), which may result in the body not producing enough anti-inflammatory proteins, potentially leading to prolonged inflammation.

Similarly, HDACs can be activated or influenced by various factors, such as:

Diet: Certain nutrients or lack thereof can influence HDAC activity.

Environmental Factors: Toxins or pollutants might increase HDAC activity, affecting gene expression.

Medications: Some drugs are designed to inhibit HDACs to help treat conditions like cancer, where reducing HDAC activity can make it easier to activate tumor-suppressing genes.



To help prevent excessive HDAC activity, include a variety of plant-based foods in your diet, such as cruciferous vegetables (like broccoli and kale), berries, and soy products, which contain compounds that can inhibit HDACs.

Incorporate foods rich in antioxidants and anti-inflammatory agents, like green tea, turmeric, garlic, and nuts, to support healthy gene expression.

Omega-3 fatty acids from fatty fish, flaxseeds, and walnuts also protect against inflammation and chronic disease.

A balanced diet emphasizing these whole, unprocessed foods can promote overall health and reduce the risk of conditions linked to HDAC activity.

More Polyphenols Specifics

Increasing Histone Acetylation: Polyphenols, such as those in green tea (EGCG) and red grapes (resveratrol), add acetyl groups to histones, relaxing DNA structure and making genes more accessible, which can help activate protective genes.

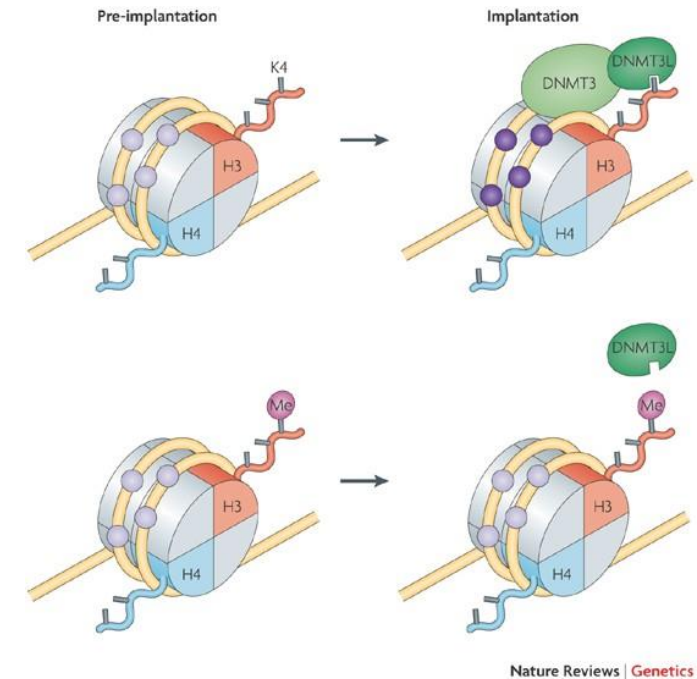
Inhibiting Histone Deacetylases (HDACs): Some polyphenols, like quercetin in apples and sulforaphane in broccoli, block enzymes that remove acetyl groups from histones, keeping DNA open and allowing beneficial genes to be more active.



Modifying Histone Methylation: Polyphenols, like curcumin in turmeric, can change the way methyl groups attach to histones, which can turn off harmful genes (like those promoting cancer) and activate helpful ones.

Providing Antioxidant Protection: Polyphenols protect histones and DNA from oxidative damage, helping maintain healthy gene expression patterns.

Overall, polyphenols help regulate gene expression in ways that support health by modifying histones and protecting them from damage.



Non-coding RNAs

(ncRNAs), such as microRNAs (miRNAs) and long non-coding RNAs (lncRNAs), do not code for proteins but play crucial roles in regulating gene expression at the transcriptional and post-transcriptional levels.

MicroRNAs can bind to messenger RNA (mRNA) molecules, leading to their degradation or preventing their translation into proteins.

Long non-coding RNAs can interact with DNA, RNA, or proteins to influence chromatin structure and gene expression patterns.

These mechanisms work together in complex networks to control gene activity in response to internal and external environmental signals, helping to adapt and respond to various conditions while maintaining overall genomic stability.

When non-coding RNAs do not regulate genes properly, it can disrupt critical processes in the body, contributing to various diseases.

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If certain microRNAs that usually help control cell growth are not functioning correctly, they might fail to suppress the activity of genes that promote uncontrolled cell division, potentially leading to the development of cancer.

Additionally, imbalances in specific non-coding RNAs have been linked to neurodegenerative diseases like Alzheimer's, where abnormal levels of ncRNAs may affect the expression of genes involved in neuron survival and function.

To keep non-coding RNAs balanced, focus on a diet rich in antioxidants (like berries and green tea) and anti-inflammatory foods (such as fatty fish, turmeric, and olive oil) to protect against cellular stress and inflammation.

Include fiber-rich foods like whole grains and vegetables to support a healthy gut microbiome, which can positively influence ncRNA regulation.

Limit intake of processed foods and sugars to reduce inflammation and oxidative stress, which can disrupt ncRNA function.



Incorporate regular physical activity to promote healthy gene expression and manage stress through techniques like mindfulness and yoga to prevent imbalances in ncRNA levels.

Ensure adequate sleep to maintain proper gene regulation and avoid environmental toxins that could damage ncRNAs.

Together, these diet and lifestyle choices help support the healthy functioning of ncRNAs, contributing to overall wellness and disease prevention.



The Role Of Exercise

Exercise influences epigenetic markers, which are changes that affect gene activity without altering DNA. Regular physical activity can modify these markers, particularly in genes related to metabolism and inflammation, leading to health benefits.

DNA Methylation Changes: Exercise can reduce DNA methylation in genes involved in metabolism, enhancing the body's ability to use fats and sugars efficiently and lowering the risk of obesity and diabetes.



Histone Modifications: Exercise increases histone acetylation, relaxes DNA, and promotes the expression of genes that reduce inflammation and support muscle growth and recovery.

Reduced Inflammatory Gene Activity: Physical activity can decrease the expression of genes that cause inflammation, helping protect against diseases like heart disease, arthritis, and cancer.



Role of Sleep & Stress

Stress management and adequate sleep can significantly impact epigenetic regulation, affecting overall health.

Stress Management: Chronic stress can cause harmful epigenetic changes that activate inflammation-related genes and suppress protective genes.

Techniques like meditation and mindfulness can reduce these changes, lower stress-related health risks, and enhance the expression of genes that support mental health.



FKBP5 Gene: This gene plays a crucial role in controlling how the body responds to cortisol, a hormone released during stress. Normally, FKBP5 helps regulate cortisol levels, ensuring that the body can return to a balanced state after a stressful event.

Stress Impact:

Chronic Stress: Prolonged exposure to stress can lead to changes in the methylation patterns of the FKBP5 gene. Specifically, chronic stress can reduce methylation in certain regions of this gene, leading to its overexpression.

Result: The body's response to cortisol becomes dysregulated when FKBP5 is overexpressed due to reduced methylation. This can result in an exaggerated stress response, where cortisol levels remain elevated for longer periods, increasing the risk of anxiety, depression, and other stress-related disorders.

Nutrients like folate (found in leafy greens and legumes), vitamins B12 (in meat, fish, and dairy) and B6 (in poultry, fish, and potatoes), choline (in eggs and soybeans), and methionine (in meat, fish, and nuts) support methylation, helping regulate the FKBP5 gene.

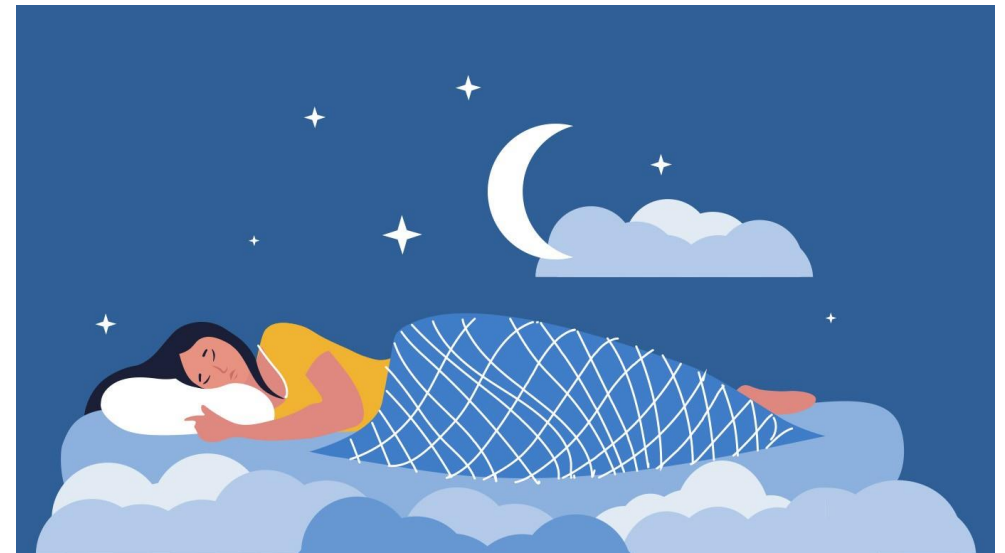
These nutrients help maintain proper gene methylation patterns, balancing the body's stress response and reducing the risk of stress-related disorders.



Role of Sleep

Adequate Sleep: Proper sleep maintains circadian rhythms that regulate genes involved in metabolism, immunity, and cellular repair. Lack of sleep can disrupt these rhythms, increasing inflammation and the risk of diseases.

Managing stress and getting enough sleep help maintain healthy gene expression, reduce inflammation, and support overall well-being.



Why Epigenetics Is Not Simple

Understanding how diet, epigenetic changes, and health interact is challenging for several reasons:

It's hard to separate the effects of genes you inherit from those of epigenetic changes, which are influenced by diet and other environmental factors.

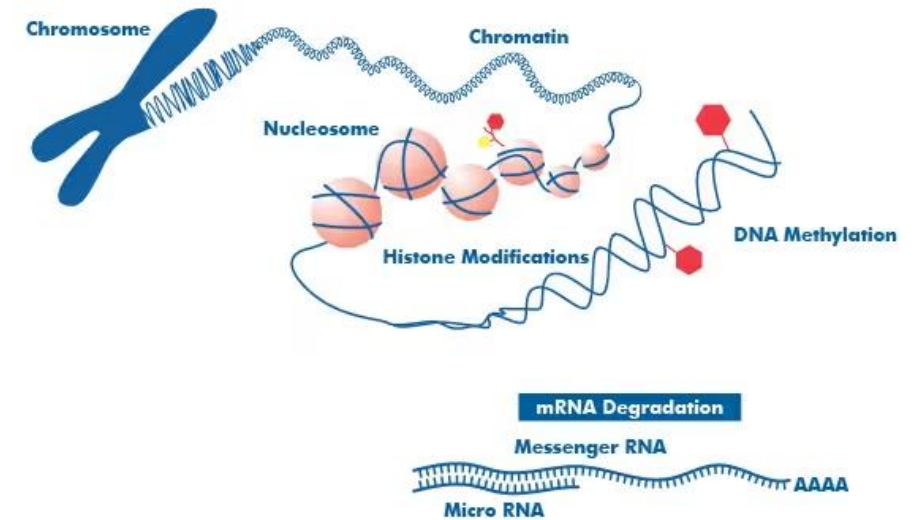
People respond differently to the same foods due to their unique genetic and epigenetic makeup, making it difficult to find universal patterns.

Diet is complex, containing many nutrients that can have mixed or opposing effects on gene regulation, so isolating the impact of one nutrient is tough.

Epigenetic changes can be temporary and reversible, influenced by many factors, so knowing if they have long-term health effects is challenging.

Long-term studies are needed to understand these effects, which are expensive, time-consuming, and hard to control.

Bringing together data from many scientific fields is complicated and requires advanced tools and collaboration to understand the full picture.



Diet can lead to epigenetic changes that impact an individual's health and their descendants, potentially increasing or decreasing the risk of certain diseases across multiple generations.

For example, when a parent consumes a diet high in certain nutrients, like folate or antioxidants, or low in calories, it can cause changes in DNA methylation or histone modifications, which regulate gene activity without altering the DNA sequence.

If these changes occur in the reproductive cells (sperm or eggs), they can be passed down to their children. This could lead to an increased or decreased risk of conditions like obesity, diabetes, and cardiovascular diseases, depending on which genes are affected.

Similarly, a mother's diet during pregnancy can directly influence the developing fetus's epigenome.

For instance, inadequate nutrition or excess sugar and fat intake can alter the gene expression in the child's metabolic regulation, immune function, and brain development.

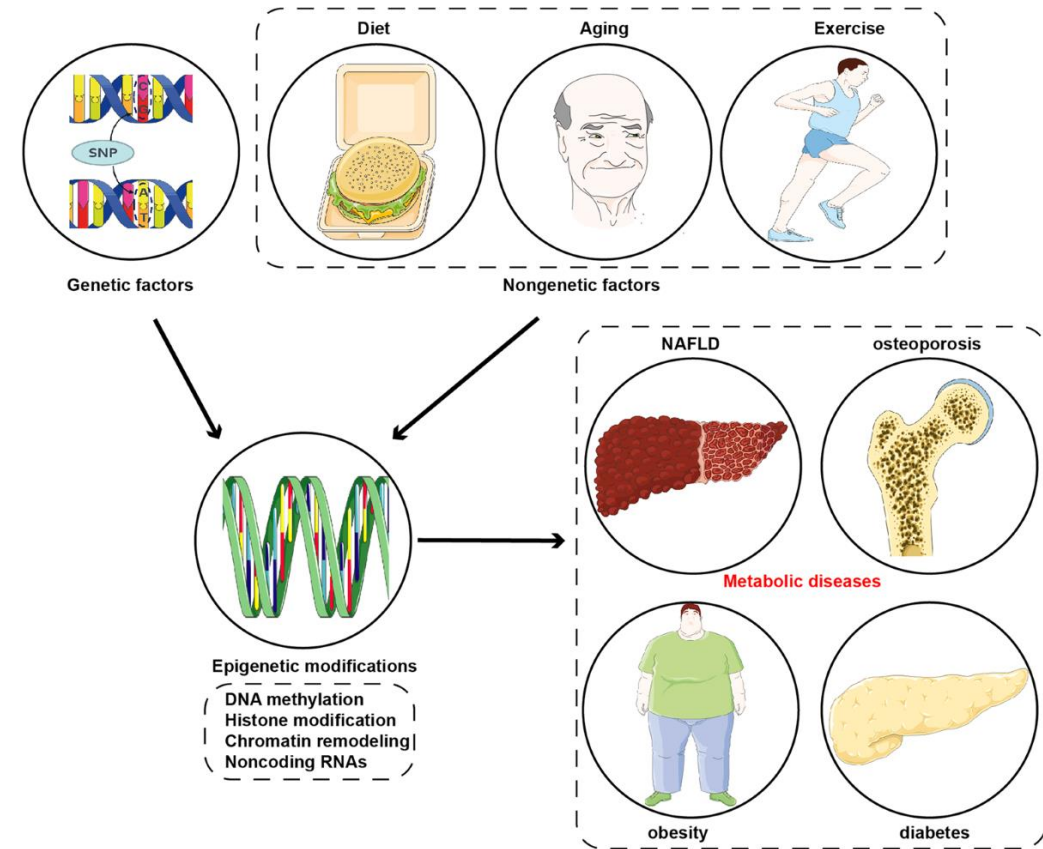
These changes can affect the child's long-term health, potentially leading to issues like insulin resistance, heart disease, or cognitive impairments later in life.

These epigenetic modifications can sometimes persist into the next generation. If a mother experiences nutritional deficiencies or excesses during pregnancy, her child's germ cells (which will become her grandchildren) can also be epigenetically modified.

This means that a poor diet in one generation can lead to health problems in multiple future generations due to inherited gene expression patterns.

Overall, the diet of one generation can set the stage for health outcomes in children, grandchildren, and beyond,

This shows how vital nutritional choices are not only for individual well-being but also for the long-term health of future generations.



When working with clients with a genetic predisposition to a health condition, you want to use this information to encourage them to make changes.

But this info also needs to be considered when preventing degenerative diseases.

Or further degeneration — we don't know how much DNA has been influenced or changed by any client.

But focusing on how to influence epigenetics will help.

