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Review of Pharmacoepigenetics: Bridging Epigenetics and Personalized Medicine

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Abstract

Pharmacoepigenetics is an emerging field that explores how alterations in gene expression, independent of DNA sequence changes, influence individual responses to drugs. Unlike pharmacogenetics, which focuses primarily on genetic variation, pharmacoepigenetics integrates genetic, environmental, and lifestyle factors to explain interindividual differences in drug efficacy, toxicity, and resistance. Key epigenetic mechanisms including DNA methylation, histone modifications, non-coding RNAs, and RNA methylation play critical roles in regulating drug metabolism and therapeutic outcomes. This review highlights the growing clinical relevance of pharmacoepigenetics in cancer, neurological, and cardiovascular disorders, where epigenetic biomarkers and targeted therapeutics are reshaping treatment strategies. Epigenetic agents, such as DNMT, HDAC, and EZH2 inhibitors, can restore normal gene expression and enhance drug sensitivity. Despite these advances, challenges remain in biomarker validation, data interpretation, and establishing causal links between epigenetic modifications and treatment response. Integration of CRISPR-based epigenome editing, artificial intelligence, and multi-omics approaches is anticipated to accelerate the clinical translation of pharmacoepigenetic insights, enabling safer, more effective, and truly personalized therapies.

Keywords: Pharmacoepigenetics, DNA methylation, non-coding RNA, epigenetics biomarkers, personalized medicine

INTRODUCTION

Epigenetics is a branch of biological science that investigates the molecular mechanisms and regulatory interactions between genes and their expression products that collectively determine the manifestation of an organism's phenotype. When the term "epigenetics" was first used, it included all molecular processes that alter a genotype's expression to produce a specific phenotype. As molecular genetics advanced quickly over time, the term's definition became more precise. Currently, the study of heritable changes in gene function that result from mitotic or meiotic processes without involving any changes to the underlying DNA sequence is known as epigenetics [1]. DNA methylation, histone modifications, histone variations, and non-coding RNA regulation are examples of epigenetic alterations that are frequently discussed in the literature nowadays. These modifications are crucial for regulating gene expression without changing the DNA sequence [2]. Epigenetics acts as a dynamic link between the environment and the genome since such changes can happen in response to environmental factors such as food, oxidative stress, and exposure to toxins. However, several illnesses, including cancer, neurological disorders, and cardiovascular

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ailments, are associated with disruption of epigenetic control, underscoring its biological significance [3]. Pharmacoepigenetics is a term used in pharmacology to describe how individual variances in medication response are influenced by epigenetic variability. Drug absorption, distribution, metabolism, excretion (ADME), efficacy, and toxicity can all be impacted by epigenetic alterations that change the expression of genes related to drug metabolism, transport, and receptor activation. Pharmacotherapy and the epigenome have a reciprocal interaction since medications themselves can cause epigenetic changes [4]. The application of pharmacoepigenetic knowledge has enormous potential for individualized medical and pharmacy practice. Clinicians can customize treatments to each patient's unique molecular profile by integrating epigenetic biomarkers with genetic and environmental data, enhancing therapeutic benefit and reducing side effects. This strategy supports the objectives of precision medicine by advancing healthcare beyond the "one-size-fits-all" paradigm toward genuinely customized medication [5].

EPIGENETIC MECHANISMS RELEVANT TO PHARMACOLOGY

DNA Methylation

The epigenetic process of adding methyl groups to DNA nucleotides, mainly cytosine and adenine, is known as DNA methylation. DNAm is believed to have a significant impact on DNA's structure and activities, especially in the control of gene expression. Previous research showed that whereas DNAm levels of gene body CpGs might promote the expression of their target genes, DNAm levels of CpG sites close to transcription start sites could suppress gene expression levels [6]. Drug metabolism and response can be impacted by DNA methylation, which can also affect the expression of drug-metabolizing enzymes, receptors, and transporters [7]. One example of an epigenetic mark that is both mitotically stable and sensitive to environmental stimuli is DNA methylation [8]. DNA methylation of genes linked to medication response and its significance for clinical pharmacology Interindividual differences in medication response are largely caused by variations in the expression of genes linked to drug response, DNA methylation has attracted growing attention [9].

HISTONE MODIFICATION

Histones, which are proteins that encircle DNA, are essential for controlling gene expression. Acetylation, methylation, and phosphorylation are examples of chemical changes to histones that can change the chromatin structure and make DNA more accessible or densely packed. Gene expression may be repressed or activated by these modifications. Histone alterations can impact genes associated to drug resistance, cancer cell proliferation, and other drug-related processes in the context of pharmacoepigenetics. For instance, acetylation of histones can increase drug resistance gene expression in cancer cells, making chemotherapy more difficult [7–10].

Non-Coding RNA

By definition, non-coding RNAs (ncRNAs) are transcripts that function in both translation and splicing events but do not produce proteins. few lncRNAs with small open reading frames (ORFs) that code for biologically active polypeptides. Depending on their size and function, these ncRNAs can be divided into various classes. Long non-coding RNAs (lncRNAs, >200 nucleotides) and small non-coding RNAs (<200 nucleotides), which include piwi-interacting RNAs (piRNAs), endogenous small interfering RNAs (siRNAs), microRNAs (miRNAs), and Y-RNAs, including ribosomal RNAs (rRNAs), transfer RNAs (tRNAs), circular RNAs (circRNAs), small nuclear RNAs (snRNAs), small nucleolar RNAs (snoRNAs), microRNAs (miRNAs), long non-coding RNAs (lncRNAs), transcription initiation RNAs (tiRNAs), and piwi-interacting RNAs (piRNAs). Figure 1 shows how ncRNAs are categorized [10]. ncRNAs, particularly miRNAs and lncRNAs, are promising indicators for predicting individual medication responses due to their tissue-specific expression and capacity to modify drug-metabolizing genes. As a result, they play a crucial role in the development of personalized medicine [11].

RNA Epigenetics (m6A Modification)

Drug efflux and chemoresistance are facilitated by several membrane transporter proteins, particularly members of the ABC family like ABCB1 and ABCC10. According to recent research, RNA

m6A alterations control the production of ABC proteins by either directly affecting tumor transcripts or indirectly affecting upstream signaling cascades. For example, m–A increased the transcription of ABCB1 by upregulating estrogen-related receptor gamma (ERRγ). Exosomal FTO promoted ABCC10 expression through the FTO/YTHDF2/ABCC10 axis, while METTL3 m-A-dependently increased translation of ABCD1, both of which contributed to drug resistance. These results demonstrate that RNA epigenetic modifications as an extra layer of pharmacoepigenetic regulation affecting drug responsiveness. By integrating this information into patient profiles, it may be possible to develop personalized treatment plans that maximize medication effectiveness and overcome resistance (Figure 1) [12].

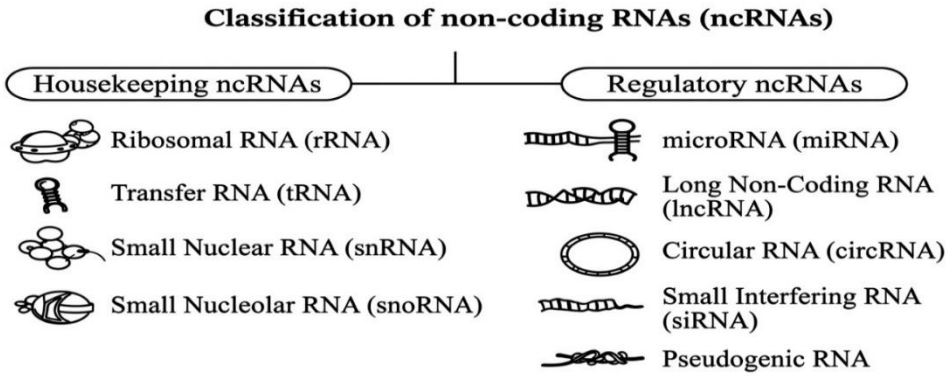

Figure 1. Classification of non-coding RNAs (ncRNAs). ncRNAs are broadly classified into housekeeping ncRNAs, including rRNA, tRNA, snRNA, and snoRNA, and regulatory ncRNAs, including miRNA, lncRNA, circRNA, and siRNA, based on their functional roles in cellular processes and gene regulation.

IMPACT OF EPIGENETIC MODIFICATIONS ON DRUG RESPONSE

- *Drug Metabolism:* Drug metabolism-related enzyme expression can be controlled by epigenetic changes. For instance, epigenetic modifications can cause the upregulation or downregulation of CYP450 enzymes, which oversee the metabolism of numerous medications. Drug efficacy and toxicity can vary depending on how these enzymes are expressed, which can change drug concentrations in the blood. The rate at which a drug is metabolized and excreted can be affected by DNA methylation or histone changes in the promoter regions of CYP genes, which can either increase or decrease their production [7].
- *Drug Sensitivity and Resistance:* Drug resistance, especially in the treatment of cancer, is significantly influenced by epigenetic modifications. Through epigenetic modifications that alter drug targets or activate survival pathways, tumor cells can develop resistance to chemotherapy. For instance, the silencing of genes that typically control cell death can result from the hypermethylation of tumor suppressor genes or the modification of histone marks, enabling cancer cells to withstand treatment. By correcting the epigenetic changes that cause resistance, an understanding of these pathways can aid in the development of therapies to overcome drug resistance [7].
- *Toxicity and Side Effects:* Drug toxicity and adverse effects may potentially be influenced by epigenetic variables. For example, medications that alter DNA methylation or histone acetylation may unintentionally impact genes related to immunological response or tissue repair, resulting in negative consequences. Clinicians may be able to anticipate and lessen these side effects more successfully if they comprehend how these epigenetic changes affect the toxicity of particular medications [13].

PHARMACOEPIGENETICS IN DISEASE THERAPY

Cancer

To provide individualized cancer treatment, pharmacoepigenetics in cancer focuses on how epigenetic changes affect drug response and therapeutic resistance. Even though epigenetic processes,

like DNA methylation, histone modifications, and non-coding RNA control, can change gene expression without altering the DNA sequence, they are crucial for the development of tumors and the variation in treatment sensitivity. According to Ocaña-Paredes et al. (2024), these reversible alterations either activate oncogenic pathways or silence tumor suppressor genes, determining the tumor's sensitivity to chemotherapy or targeted therapies. Finding these epigenetic biomarkers increases oncology precision and aids in treatment outcome prediction [14]. Similarly, Smith (2023) highlighted the connection between patient-specific treatment response and genetic diversity through pharmacoepigenetics. Because of this knowledge, epigenetic medications, or "epidrugs," have been developed that can alter the behavior of cancer cells [15]. All things considered, pharmacoepigenetics is a fundamental component of precision oncology, combining genetic and epigenetic knowledge to customize cancer treatment. Even while there are still obstacles to overcome, such identifying stable biomarkers and reducing off-target effects, research is strengthening its role in creating more individualized and successful cancer treatments.

Neurological Disorders

Pharmacoepigenetics in neurological illnesses investigates how changes in epigenetic regulation, such as DNA methylation, histone modification, and non-coding RNA expression, affect the course of the disease and the results of treatment, providing new opportunities for precision therapy. Research indicates that altered epigenetic landscapes lead to neuronal malfunction and maladaptive plasticity in diseases including Alzheimer's, Parkinson's, Huntington's, and other uncommon neurodevelopmental disorders [16]. When combined, pharmacoepigenetics holds the potential to move neurological therapy away from symptomatic management and toward mechanism-based, customized interventions. However, there are still issues with long-term epigenetic stability, cell-type specificity, and drug delivery across the blood–brain barrier [17].

Cardiovascular Diseases

Cardiovascular disease (CVD) mortality and morbidity are major social burdens in the developed countries. Heart failure, congenital heart disease, atherosclerotic cardiovascular disease, cardiomyopathy, and hypertensive heart disease are examples of CVDs that are increasingly being treated as much more complex conditions [18]. MicroRNA, DNA methylation, and tone alterations are examples of epigenetic changes. In fact, there is an increasing amount of epigenetic study in this area, and it has been discovered that miRNAs and histone changes play a significant role in cardiac disease. Since aberrant methylation of CpG islands is a feature of many cardiovascular ailments, several medications (such as hydralazine and procainamide) that may directly inhibit DNA methyltransferase or reduce its gene expression are now being studied. Preclinical research has verified the antiproliferative, anti-inflammatory, and cardioprotective actions of histone deacetylase inhibitors [19].

EPIGENETIC BIOMARKERS IN PHARMACOEPIGENETICS

Prognostic and Diagnostic Biomarker

Prognostic biomarkers make it possible to track the progress of anticancer treatment, evaluate the tumor's stage and possible aggressiveness, and determine the likelihood of disease remission in each particular case. By identifying polymorphisms, mutations, changes in DNA methylation or gene expression, or by identifying the presence of particular microRNA (miRNA) molecules or CTCs in peripheral blood, prognostic biomarkers are linked to a particular type of tumor (Figure 2) [20]. For many years, biomarkers have been utilized for the clinical diagnosis of various diseases. For instance, amylase is used to diagnose pancreatitis, creatinine is used to diagnose renal insufficiency, and troponin level is used to diagnose myocardial infarction [21].

Predictive Biomarkers for Drug Response

A predictive biomarker is defined by the FDA-NIH Biomarker Working Group as a biomarker that is used to identify people who are more likely than comparable individuals without the biomarker to experience a positive or negative effect from exposure to a medical product or an environmental agent [22]. Two kinds of predictive biomarkers have entered the clinic over the last few decades: Biomarkers

that are pharmacogenetic (PGx) and companion diagnostic (CDx) [23]. Predictive biomarkers aid in the therapeutic decision-making process by offering information on the likelihood of receiving a response to treatment. Somatic mutations of both point mutation and chromosomal aberration types in the following genes-EGFR, KRAS, BRAF, PDGFRA, KIT, HER2, BCR-ABL, and EML4-ALK are the most significant predictive biomarkers with established therapeutic utility in molecular diagnostics in oncology (Table 1 and Figure 2) [24].

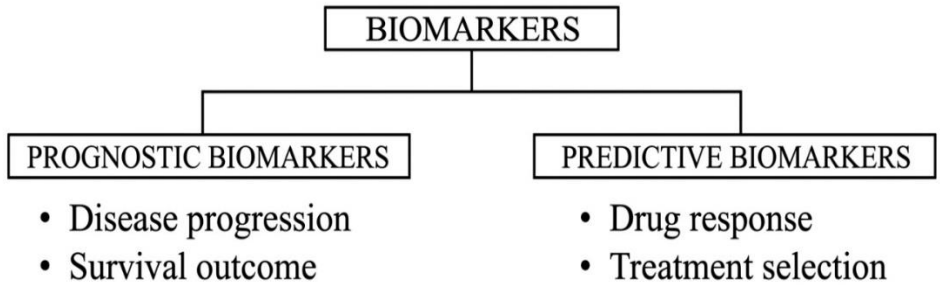

Figure 2. Classification of epigenetic biomarkers in pharmacoepigenetics. Epigenetic biomarkers are categorized into prognostic biomarkers, which indicate disease progression and clinical outcomes, and predictive biomarkers, which assess therapeutic response and support personalized drug selection.

Table 1. Classical and emerging epigenetic drugs with their mechanisms of action (compiled and adapted from [14, 25, 26, 27] and recent studies).

Drug	Class	Molecular target	Mechanism of action
Azacitidine	DNMT inhibitor	DNMT1	DNA hypomethylation leading to reactivation of genes.
Decitabine	DNMT inhibitor	DNMT	Incorporates into DNA and inhibits methylation.
Vorinostat	HDAC inhibitor	HDAC	Increases histone acetylation leading to gene activation.
Romidepsin	HDAC inhibitor	Class I HDAC	Induces apoptosis via chromatin remodeling.
Tazemetostat	EZH2 inhibitor	EZH2	Inhibits H3K27 methylation and reduces gene silencing.
Givinostat	HDAC inhibitor	HDAC	Modulates histone acetylation and gene regulation.
Revumenib	Menin inhibitor	Menin-KMT2A complex	Blocks menin interaction restoring transcription.
Dordaviprone	Epigenetic modulator	DRD2 / ISR pathway	Activates stress response and indirect epigenetic effects.
Vafidemstat	LSD1 inhibitor	LSD1	Inhibits histone demethylation altering gene expression.
Mevrometostat	EZH2 inhibitor	EZH2	Selectively inhibits oncogenic gene silencing.

DNA Methyltransferase

DNA methyltransferase (DNMT) inhibitors, which target the enzymes that methylate cytosine residues inside CpG dinucleotides in DNA, are becoming more widely acknowledged as crucial medicines in cancer therapy. Azacitidine and decitabine are two FDA-approved DNMT inhibitors that are used to treat acute myeloid leukemia, chronic myelomonocytic leukemia, and myelodysplastic syndromes. These inhibitors incorporate into DNA as nucleoside analogs, resulting in DNA hypomethylation. They mostly have a cytostatic impact at therapeutic levels, allowing previously silenced genes to be reactivated [14]. The primary disadvantage of DNMTi is that they are not appropriate for precisely targeting a specific methylated CpG. Thousands of transposable elements, such as endogenous retroviruses (ERVs) and latent cancer testis antigens (CTAs), which are typically repressed by DNA methylation in most somatic cells, are expressed by DNMTi in addition to their capacity to reactivate genes such as tumor suppressors [25].

HISTONE DEACETYLASE INHIBITOR

Over the past five years, many histone deacetylase inhibitors (HDACi) have been evaluated in clinical trials. Both histone and nonhistone targets may be hyperacetylated by these drugs, which may have a variety of effects on immune responses, cancer cells, and their surroundings [26, 27]. The FDA has approved HDAC inhibitors, including vorinostat, romidepsin, belinostat, and Panobinostat [14]. Histone deacetylase inhibitors generally have three structural characteristics: a zinc binding moiety, an opposing capping group, and a straight chain alkyl, vinyl, or aryl linker connecting the two. The ideal connection between the two functional groups is often an aliphatic chain with six carbons [28]. Understanding the molecular mechanisms of HDAC inhibitors, particularly their effects on chromatin structure and gene expression, is crucial for improving cancer therapy strategies and overcoming resistance and adverse effects [14].

EZH2 INHIBITOR

One way to provide targeted epigenetic regulation is to inhibit enhancer of zeste homolog 2 (EZH2). The polycomb repressive complex 2 (PRC2) catalytic components, EZH2, is a histone methyltransferase. EZH2 functions by trimethylating histone 3 lysine 27 (H3K27) in a PRC2-dependent manner. Methylation of H3K27 is a significant epigenetic event that results in gene suppression. Since EZH2 is a master regulator, any dysregulation of it could encourage the growth of cancer [27]. The U.S. Food and Drug Administration (FDA) has approved Tazemetostat (Tazverik), a first-in-class oral EZH2 inhibitor for FL and epithelioid sarcoma (ES). Tazemetostat inhibits EZH2 and restores normal epigenetic regulation, making it an important therapeutic option in cancers driven by EZH2 overactivity [29].

CHALLENGES AND LIMITATIONS IN PHARMACOEPIGENETICS

With the resources at our disposal and our current understanding of pharmacoepigenetics, theories regarding the relationship between epigenetics and traditional non-epigenetic drug response can be generated. Nevertheless, as demonstrated by the clozapine example, some of the data required to rebuild or forecast pharmacoepigenetic connections is now unavailable. Unavailable data is not the only problem in the field. Although pharmacoepigenetics is difficult to investigate in the context of cancer, epigenetic pathways are crucial for the development of cancer. Epigenetic markers can be influenced by medications, environmental variables, and disease states; similarly, marker levels can change drug responsiveness and disease prognosis. Furthermore, epigenetic states are dynamic and alter over the course of a person's lifetime. Therefore, it is not always easy to determine the origin and effect of reported connections between pharmacological reactions, medical problems, and epigenetic markers [30]. Even though a lot of information is already accessible, further research is needed to completely understand how pharmacoepigenetic factors affect drug response. We may fully realize the potential of pharmacoepigenetics as a potent tool for comprehending biological mechanisms and creating successful therapies by using cutting-edge computational techniques to find or forecast pharmacoepigenetic correlations combined with biological validation [4].

FUTURE PERSPECTIVE

The integration of CRISPR-based epigenome editing techniques, big-data analytics, artificial intelligence (AI), and multi-omics technologies will influence the future of pharmacoepigenetics. The development and integration of multi-omics data increases treatment efficacy and reduces side effects by enabling more precise and personalized therapeutic methods [31]. A deep neural network model that incorporates multi-omics data is used to predict cellular responses to known anti-cancer treatments [32]. Healthcare could undergo a radical change because to precision medicine and artificial intelligence (AI). AI enables the system to reason and learn, empowers physician decision-making with enhanced intelligence, and generates insights through sophisticated computation and inference [33]. By combining the accuracy of CRISPR with the ability to rewrite epigenetic marks, CRISPR/Cas9-based epigenome editing provides a customizable and reversible gene regulation technique without altering DNA sequences. CRISPR/dCas9 effectors are delivered temporarily for programmable epigenome editing [34]. Advances and Future Prospects of Epigenome Editing-Based Therapy. Although CRISPR-

based gene editing holds great promise for customized therapy, there are certain hazards and challenges that must be carefully evaluated [35].

CONCLUSION

Pharmacoepigenetics investigates how individual medication responses are influenced by epigenetic mechanisms as DNA methylation, histone modification, non-coding RNA control, and RNA methylation. It explains differences in pharmacological efficacy, toxicity, and resistance by bridging genetic predisposition and environmental conditions. Liquid biopsies and epigenetic biomarkers help track illness, forecast results, and direct individualized therapies. Its relevance in precision medicine is highlighted by applications in neurology, cardiovascular medicine, and oncology. DNMT, HDAC, and EZH2 inhibitors are examples of epigenetic medications that have the potential to reactivate suppressed genes and improve treatment sensitivity. Clinical translation will be accelerated by developments in multi-omics, AI analytics, and CRISPR-based epigenome editing. Pharmacoepigenetics is an important step toward safer and more effective tailored medicines, despite obstacles including tissue specificity and data limitations.

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