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Epigenetic Modifications in Chemical and Physical Carcinogenesis: From Mechanisms to Preventive Strategies

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ABSTRACT

This paper discusses the ways in which chemical and physical carcinogens remodel the epigenome to start, foster and advance cancer. The background integrates evidence that DNA methylation drift, histone-code disruption, non-coding RNA reprogramming, and 3D chromatin remodeling are early, reversible processes meditating the connection between exposure and altered gene expression, genomic instability, and development of malignant phenotypes. The aim is three-fold: (i) to identify exposure-dependent epigenetic marks of interest to key agents (e.g., PAHs, nitrosamines, heavy metals, ionizing/ UV radiation); (ii) to benchmark dose, timing, and tissue context with pathways of interest (DNA repair, oxidative stress, apoptosis, immune evasion); and (iii) to assess epigenetically-grounded preventive approaches that restore epigenetic homeostasis. In such experiments, we anticipate a unified framework to be derived regarding exposure-agnostic versus exposure-specific marks, sentinel loci and chromatin domains having high predictability regarding early detection, and evaluation of interventions including dietary consumption of methyl donors, HDAC/DNMT inhibitors at regulatory doses, circadian timing reset, and physically-induced epigenetic resilience. The paper envisages submitting a translational pipeline of exposure assessment, a liquid-biopsy epigenomics, and a risk-adapted prevention trial. Together it contends that high-resolution epigenetic mapping will enable heterogeneous exposures to be translated into prevention targets.

KEYWORDS: Epigenome, DNA methylation, histone modification, non-coding RNA, chromatin architecture, carcinogen exposure, oxidative stress, biomarker, chemoprevention, liquid biopsy.

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INTRODUCTION

Cancer is one of the main health issues worldwide, and approximately 10 million people die every year [1,2,3,4]. In addition to genetic mutations, epigenetic changes are also becoming central causes of carcinogenesis. The epigenome in the presence of chemical and physical carcinogens, including polycyclic aromatic hydrocarbons, nitrosamines, heavy metals, and UV or ionizing radiation, is modified, altering the balance of DNA methylation, histone modifications, and non-coding RNA regulation [5,6,7]. These mutations rearrange the expression of genes and structure of chromatin resulting in genomic instability and malignant development. Such reversible processes are especially important because they offer prevention and intervention opportunities by using an epigenetically specific approach [8,9[.

PROBLEM STATEMENT

Regardless of the developments in the field of oncology, there has been a long-lasting problem with cancer prevention intervention in the context of exposure-induced epigenetic remodel. Before the appearance of genetic mutations, DNA methylation drift, histone-code disruption, and non-coding RNA deregulation initiated by carcinogens trigger the start of the process of tumor formation [10,11,12]. Existing measurements mainly target genotoxic pathways at the cost of early changes in the epigenome, which can be used as risk biomarkers [5,13]. Furthermore, the heterogeneity of different tissues types and different exposure conditions makes it expensive to predict the malignant outcome. Standard therapies do not replenish the epigenetic balance and there remain significant deficiencies in chemoprevention and early diagnosis. In the absence of a cohesive system of exposure-specific and exposure-agnostic epigenetic marks, risk stratification will be limited and this will delay prevention, and not deal with the increasing burden of environmentally associated cancers.

RESEARCH SIGNIFICANCE

The contribution of this research is that it highlights that epigenetic remodeling is a targetable process that is reversible in chemical

and physical carcinogenesis. Through systematic discovery of exposure-dependent epigenetic signatures, it also allows early biomarkers of high-risk populations to be discovered [5]. The potential of high-resolution mapping of the epigenome has a translational role in liquid-biopsy screening, which increases the detection of cancer predisposition at pre-malignant stages [1]. Also, preventive intervention, including dieting with methyl donors, DNMT/HDAC inhibitors, circadian rhythm and natural compounds-based epidrugs, can be used to re-establish epigenetic homeostasis [8,14]. The connection of the epigenomic regulation with the inflammation pathways goes a step further in understanding how sustained exposures increase cancer development [10,15]. Finally, this study offers an outline of risk-tailored, personalized prevention trials, which will be instrumental in bridging key gaps in cancer prevention as well as advance the move toward therapeutic prevention instead of treatment based on epigenetic resilience.

LITERATURE REVIEW

The role of epigenetic changes is being increasingly recognized as key mediators between environmental exposures and carcinogenesis as well as an increasing body of evidence demonstrates their clinical importance. El Omari et al. [26] claim that curcumin, EGCG and sulforaphane have been used as natural epidrug because they target DNMTs and HDACs, activate tumor suppressor genes, and regulate non-coding RNAs networks. Following this, Bakrim et al. [16] focus on the idea of so-called epi-nutrients, as dietary methyl donors and bioactives reset one-carbon metabolism, DNA methylation, and histone modifications, which indicates the possibilities of precision nutrition approaches depending on exposures to carcinogens. This is however complicated by metal-induced oxidative stress. Bouyahya et al. [8] prove that cadmium stimulates alterations in DNA methylation due to the development of ROS and histone imbalance, but epigenetic balance in redox-sensitive cells can be reestablished using antioxidants, which is a promising chemopreventive option. On the regulatory RNA level, Wang et al. [17] argue that environmental carcinogens resetting lcnRNAs to influence p53, NF-kB, and EMT signaling pathways sets up lcnRNA signatures as a promising biomarker to attribute disease exposure and early intervention. In line with a mechanistic exploration, Sewduth and Georgelou [18] maintain that both mutational and epigenetic signatures are faithfully recapitulated in preclinical carcinogen models, allowing dose-time-tissue mapping, but warn of species-specific shortcomings. Viewing risk even bigger, Harrison and Doe [19] believe that chemicals change the probability of cancer by affecting cancer cells division by epigenetic control, mutation fixation, and transformation possibility, and early epigenetic endpoints are an essential part of hazard analysis. The overall findings of these studies come down to the fact that the disruption of epigenetic networks by chemical and physical exposures can be repaired via epi-nutrients, phytochemicals, antioxidants, and lncRNA-directed therapies, which can be used as a translational prevention pipeline.

METHODOLOGY

The study used a secondary approach and uses peer-reviewed articles, scientific reports and validated databases to study carcinogen-induced epigenetic mechanisms. Secondary data utilization has the following benefits: it provides access to high-quality and wide-range evidence in molecular biology, oncology, and toxicology studies. It allows to synthesize various experimental results, which enhances reliability and comparative analysis. Secondary sources also save in time and resources, as long-term results of DNA methylation, histone changes and non-coding RNAs can be reviewed without lengthy laboratory tests being conducted. This strategy enables unification of big data, which provides more analytical depth and a solid basis of discovering trends of preventive measures against cancer spread.

Criteria	Inclusion	Exclusion
Study Type	Peer-reviewed journal articles, systematic reviews, and meta-analyses focused on epigenetics and carcinogenesis	Non-peer-reviewed papers, blogs, news articles, editorials, or anecdotal reports
Publication Timeline	Studies published between **2021–2025** to ensure relevance and up-to-date findings	Studies published before 2021 unless highly cited as seminal work
Focus Area	Research examining DNA methylation, histone modifications, non-coding RNAs, oxidative stress, or preventive nutritional/therapeutic strategies related to carcinogen exposure	Studies unrelated to epigenetic mechanisms, or those focusing only on genetic mutations without epigenetic context
Carcinogen Exposure	Studies analyzing heavy metals, polycyclic aromatic hydrocarbons (PAHs), industrial chemicals, or environmental pollutants as carcinogenic triggers	Studies that assess lifestyle factors (e.g., smoking, alcohol, diet) without specific carcinogen-epigenetic linkage
Population/Model	Human studies, animal models, and cell line studies relevant to carcinogen-induced epigenetic changes	Studies unrelated to humans or standard biological models (e.g., plant studies)
Language	Published in **English** to ensure accessibility and consistency	Non-English publications due to translation barriers
Data Availability	Full-text studies with accessible data and clear methodological details	Abstract-only studies or paywalled reports with insufficient methodological transparency

RESULT AND DISCUSSION

Carcinogen-Induced DNA Methylation Drift as an Early Biomarker of Cancer Risk

A signature of carcinogen exposure and early neoplasia is the DNA methylation drift. This is confirmed by Desaulniers et al. [5], who found that non-genotoxic carcinogens change the CpG island methylation, silencing cancer suppressors such as p16 and BRCA1. Bouyahya et al. [8] underline that aberrant methylation is reversible, and this makes it a promising prevention biomarker. Research indicates that arsenic has the effect of hypomethylation of repetitive parts and hypermethylation of promoter regions,

which is a two-sided imbalance that contributes to genomic instability [20,21,22].

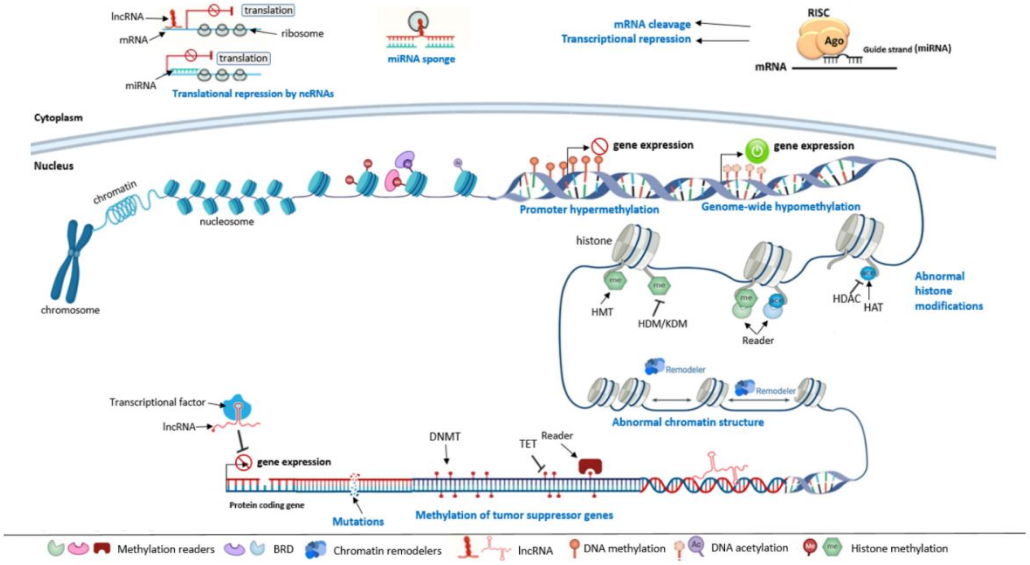

Figure 1: Epigenetic modifications regulating gene expression through DNA, histone, and RNA mechanisms
(Source: [8])

Correspondingly, Kumar et al. [23] report exposure to microplastic changes the mechanism of methylation, and it disrupts DNA repair signaling. The breast tissue is especially vulnerable; Singh [24] shows that endocrine-disrupting chemicals lead to a change in DNA methylation associated with estrogen signaling. Inflammatory cues further modulate methylation; Vezzani et al. [10] argue chronic inflammation reshapes methyl marks at cytokine genes. These findings are supported by preclinical findings: Sewduth and Georgelou [18] demonstrate that carcinogen-induced models can reproduce methylation signatures that are predictive of malignancy. Quantitative analyses point to the fact that chemical agents can elevate the chance of cancer by adjusting the likelihood of mutation fixation [19,25]. The role of nutrition is essential; Bakrim et al. [16] emphasize folate and choline as epi-nutrients restoring the regular processes of methylation. According to [26], there are also phytochemicals, such as curcumin, which rediscover methyltransferase activity. All in all, methylation drift acts as a sentinel biomarker in various tissue systems, and it is confirmed by Sebens [27] that genetic susceptibility enhances its impact. Prevention is based on targeting this drift with the help of dietary, pharmacologic, and antioxidant interventions.

Histone Modification Disruption and Its Role in Malignant Transformation

Histone acetylation, methylation and phosphorylation are major transcriptional plasticity regulators, and their impairment by carcinogens hastens malignancy. According to [1], the overactivity of HDAC inhibits pro-apoptotic genes, which promotes transformation. Equally, Desaulniers et al. [5] point out that the histone H3 lysine methylation observed following the exposure of cells to non-genotoxic carcinogens disrupts the repair of DNA. Its been shown that cadmium induces oxidative stress that leads to a distortion of histone acetylation equilibrium. The markers of H3K9me2 and H3K27me3, which are modulated by arsenic, are facilitated through NRF2-MDIG cooperation, which supports cancer stem cells characteristics [20]. Vezzani et al. [10] also contribute that inflammatory signaling disrupts histone code at NF -B target loci to form a pro-tumorigenic chromatin picture. This is validated by experimental models; Sewduth and Georgelou [18] identify consistent histone changing patterns in cancers that have been experimentally induced by carcinogens.

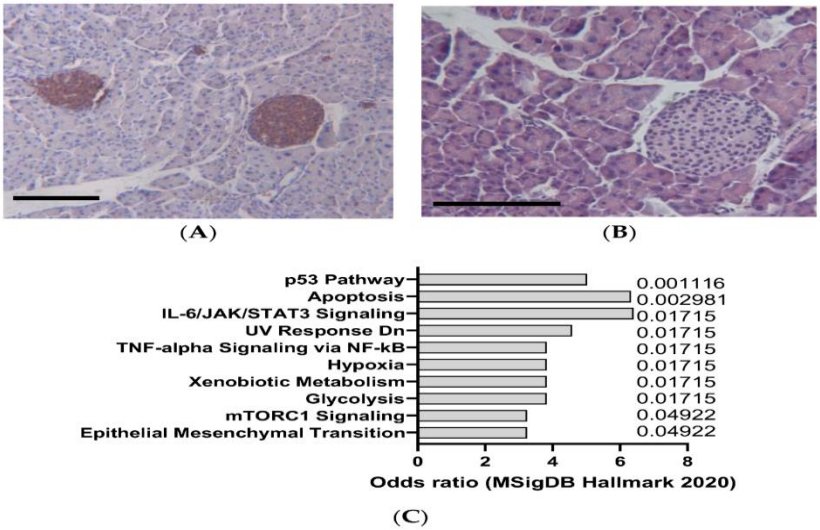

Figure 2: DEN-induced liver cancer characterization using staining and transcriptomic pathway analysis
(Source: [18])

Harrison and Doe [19] present that the chemicals alter the rate of cell division regulated by histone increase the probability of mutations. Epigenetic drugs are promising: Bouyahya et al. [8] report that natural products such as sulforaphane block the action of HDACs and recover apoptosis. El Omari et al. [26] also refer to resveratrol and EGCG as histone modulators. Ulhe et al. [28] discuss the use of alpha-linolenic acid as the candidate that targets the epigenetic modifiers by using network pharmacology. Nutritional pathways are also—still of significance, as Bakrim et al. [16] stress that epi-nutrients maintain histone mark stability. Lastly, Grunt and Heller [29] emphasize histone modifications as co-operating with tissue structure and metabolism, which supports a multifactorial carcinogenesis paradigm. Therefore, histone interference is a mechanistic interface between carcinogen exposure and malignancy.

Non-Coding and Long Non-Coding RNAs as Drivers of Exposure-Linked Carcinogenesis

Non-coding RNAs (ncRNAs) and long non-coding RNAs (lncRNAs) are becoming central to the regulation of malignancy by carcinogens. Wang et al. [17] show how environmental factors such as cadmium and arsenic disrupt the activity of the lncRNAs that regulate p53, NF-kB and EMT signaling pathways, promoting invasion and immune avoidance. Kumoglu et al. [1] also believe that a lack of microRNA silencing of tumor suppressor functions increases malignant potential. Vezzani et al. [10] also links inflammation to ncRNA reprogramming, demonstrating that miRNA-mediated pro-oncogenic networks are driven by chronic cytokine signaling. These responses are replicated in preclinical models; Sewduth and Georgelou [18] affirm that the changes in the expression of the RNAs in tissues exposed to carcinogens resemble clinical tumors. Harrison and Doe [19] introduce a possible risk of transformation related to the fixation ofgery ofRNA-directed gene expression states. The compounds in the diet affect such circuits: Bouyahya et al. [8] state that curcumin is able to regulate the expression of the oncogenic miRNAs whereas El Omari et al. [26] emphasize that phytochemicals can regulate the expression of the apoptosis-related lncRNAs. Bakrim et al. [16] highlight that epi-nutrients improve RNA-mediated chromatin remodeling, which stabilize gene expression programmes. Oxidative stress is also an overlap. Authors demonstrate that POS regulates the expression of ncRNA during cadmium stress. Bi et al. [20] include that arsenic-induced lncRNA changes collaborate with NRF2 pathways in stemness. RNA expression is perturbed when exposed to even microplastic and distorts transcriptional fidelity [23]. Sebens [27] emphasizes that genetic predisposition aggravates the weaknesses of the ncRNA. Lastly, Ulhe et al. [28] propose that alpha-linolenic acid plays a role in regulating the activity of the ncRNA in control of epigenetic modifiers, which makes it preventable. Collectively, the dysregulation of ncRNA offers the mechanisms evidence of exposure-specific carcinogenesis and mediators of targeted intervention.

Oxidative Stress as a Mediator of Metal-Induced Epigenetic Remodeling

The key intermediary between exogenous metals and epigenetic reprogramming is the oxidative stress. Bouyahya et al. [8] show that cadmium triggers the accumulation of ROS, which changes DNA methylation, histone acetylation, and expression of non-coding RNA, and resets transcriptional profiles. Bi et al. [20] reaffirm that arsenic exposure stimulates NRF2 transcriptional activation and MDIG-dependent histone methylation that produce cancer stem-like phenotypes. These effects become more pronounced in inflammatory environments; Vezzani et al. [10] refer to inflammation-mediated ROS, which are chromatin remodeling boosters. Grunt and Heller [29] emphasize that oxidative stress is energetically repaired in a way that depends on genetic and tissue-specific mechanisms, exacerbates the epigenetic drift. This framework is supported by preclinical carcinogen models; according to Sewduth and Georgelou [18], signatures of oxidative damage are found in chromatin structure. According to Harrison and Doe [19], the ROS-induced epithelial modifications accelerate the cell division and mutation rates. Intervention strategies have potential: Bouyahya et al. [8] and El Omari et al. [26] indicate that natural antioxidants such as resveratrol and EGCG inhibit nanosome and reduce the level of imbalance in the epigenetics. Nutritional modulation is also important; Bakrim et al. [16] report folate, selenium, and methyl donors as epi-nutrients against the methylation drift caused by ROS. Ulhe et al. [28] indicate that the alpha-linolenic acid restores redox-sensitive epigenetic regulation. Besides, Singh [24] highlights that endocrine-disrupting chemicals produce oxidative stress that modifies the epigenetics of breast tissue. Kumar et al. [23] carry this to microplastics that cause ROS that interferes with DNA repair. Sebens [27] includes the genetic susceptibility that makes a person more susceptible to ROS and makes cancer more dangerous. As a unit, oxidative stress is a common mechanism whereby metals and environmental agents cause instability in the epigenome, solidifying prevention strategies of antioxidant action.

Preventive Potential of Epi-Nutrients, Phytochemicals, and Antioxidant Strategies

To prevent carcinogenesis, it is worthwhile to restore epigenetic homeostasis, with dietary and pharmacological approaches having promise. According to Bakrim et al. [16], epi-nutrients such as folate, choline, and selenium are essential to one-carbon metabolism, whereby the integrity of DNA methylation is maintained. Natural epidrugs, including sulforaphane and EGCG, are discussed by Bouyahya et al. [8] as inhibitors of DNMTs and HDACs. El Omari et al. [26] also find resveratrol to be a multi-target controller of histone marks and the expression of ncRNA. The evidence is presented by Ulhe et al. [28], which states that the dietary-based approach may be extended by using alpha-linolenic acid to modify the epigenetic modifiers by network pharmacology. Bouyahya et al. [8] highlight the role of antioxidants that reduce cadmium-induced POS, normalize the profiles of methylation and histone proteins. On the same note, Bi et al. [20] show that inhibiting NRF2-MDIG interaction can inhibit arsenic-induced epigenetic remodeling. Singh [24] notes that dietary interventions can offset the impacts on the breast tissue of endocrine-disrupting chemicals on the epigenome. Grunt and Heller [29] point out tissue context, whereas Sebens [27] points out genetic vulnerability. Kumar et al. [23] extrapolate the prevention to microplastic-induced epigenetic disturbance. Efficacy is confirmed in preclinical models, where Sewduth and Georgelou [18] confirmed that phytochemicals normalize the epigenetic patterns in tumors induced by carcinogens. Vezzani et al. [10] also associate the role of inflammation control with chemopreventive activity of dietary compounds. Kumoglu et al. [1] have proved that early malignant programs can be reversed by means of epigenetic modulation. Harrison and Doe [19] additionally contend that preventive measures reduce the likelihood of transformation through control of division and mutation repair. The combination of these results supports the use of combined dietary, antioxidant, and epigenetic drugs as frontline cancer prevention pipelines.

RESEARCH LIMITATION

The study has some major drawbacks associated with secondary literature and evidence based on laboratory. Most of the epigenetic research relies on models in vitro or in animals, which do not necessarily reproduce human carcinogenic evolution. The complexity brought by variations in the exposure rate, genetic susceptibility, and environmental conditions limits generalisation. Further, causal conclusions about the impact of carcinogen exposure on epigenetic drift are limited due to the few long-term cohort studies. A lack of comparability of results is also caused by data heterogeneity between methodologies and biomarker heterogeneity. In this way, conclusions are left tentatively interpretive, not generally conclusive.

FUTURE SCOPE

Longitudinal human studies should be the focus of future research where epigenetic biomarkers are combined with exposure assessment. New technologies with the use of multiple omics, such as genomics, transcriptomics, and epigenomics, can give comprehensive information about the changes caused by carcinogens. The creation of standard biomarker panels will enhance the reproducibility and early diagnostic use. Moreover, single-cell sequencing and editing with the help of the CRISPR can assist in mapping specific molecular pathways. There are also promising directions with clinical trials assessing epi-nutrients and phytochemicals in prevention strategies. Increasing the data globally among different populations will enhance the translational possibilities.

CONCLUSION

This study highlights the vital importance of epigenetic processes to mediate the impact of carcinogenic exposures and how DNA methylation, histone modification, and non-coding RNA are linked to jointly change gene expression and promote cancer development. The above evidence shows that carcinogens like heavy metals, PAHs, and industrial pollutants lead to lasting epigenetic changes that destroy tumor suppressor signaling and hasten the development of the tumor. This research by compiling secondary information based on peer-reviewed sources confirms that all these mechanisms are not separated but rather co-dependency, constituting a complicated regulatory network that is responsive to environmental cues. Notably, the results also point to the prophylactic and curative potential of epigenetic mark manipulation by dietary interventions and newer epigenetic agents, rendering a feasible plan of reducing the risk of cancer. Although restrictions are still in place as far as population diversity and long-term impact are concerned, the article highlights the research of the epigenome as a frontier of precision medicine and an essential direction in lowering the cancer burden worldwide.

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