Commentary
Volume 10:05, 2024

Journal of Clinical Respiratory Diseases & Care: Open Access

ISSN:2472-1247 Open Access

The Relationship between Smoking, Alcohol Consumption and Betel Quid Chewing with Epigenetic Alterations in Cancers

Yueqi Lin*

Department of Chemical Biological Sciences, University of Sonora, Hermosillo, Sonora, Mexico

Introduction

The interplay between lifestyle factors and cancer development is a critical area of research, particularly as it relates to smoking, alcohol consumption, and betel quid chewing. These habits, often culturally and socially ingrained, have been linked to significant epigenetic changes that drive cancer progression. Epigenetic alterations, unlike genetic mutations, involve changes in gene expression without modifying the underlying DNA sequence. They include DNA methylation, histone modifications, and the dysregulation of non-coding RNAs, all of which can disrupt normal cellular processes and contribute to oncogenesis. This report explores the association between these lifestyle factors and the epigenetic aberrations observed in various cancers. Smoking is one of the most well-documented risk factors for cancer. Tobacco smoke contains a complex mixture of carcinogens, including polycyclic aromatic hydrocarbons (PAHs) and nitrosamines, which induce both genetic mutations and epigenetic changes. DNA methylation, in particular, is profoundly affected by smoking. Hypomethylation of oncogenes and hypermethylation of tumor suppressor genes have been observed in smokers, leading to dysregulated cellular growth and increased cancer risk. For example, hypermethylation of the *p16* and *MGMT* tumor suppressor genes is frequently found in lung and head-and-neck cancers associated with smoking. These epigenetic changes are often accompanied by histone modifications that further enhance the transcriptional repression of critical regulatory genes.

Description

The carcinogenic impact of smoking extends beyond direct DNA damage and epigenetic alterations. Tobacco use is associated with chronic inflammation and oxidative stress, which contribute to a pro-cancerous microenvironment. This sustained inflammation can lead to further epigenetic dysregulation, including the activation of pro-inflammatory genes through histone acetylation and the suppression of anti-inflammatory pathways via histone deacetylation. Additionally, smoking-induced epigenetic changes can persist long after cessation, indicating a "memory effect" that maintains an elevated cancer risk in former smokers. Alcohol consumption is another major lifestyle factor linked to cancer through epigenetic mechanisms. Ethanol metabolism produces acetaldehyde, a highly reactive compound that can form DNA adducts and interfere with DNA repair processes. This damage is often accompanied by global changes in DNA methylation patterns. Chronic alcohol consumption has been shown to cause hypomethylation of oncogenes and hypermethylation of tumor suppressor genes, similar to the effects of smoking. These alterations are particularly evident in cancers of the liver, esophagus, and oropharynx [1].

Alcohol also disrupts one-carbon metabolism, a pathway critical for maintaining proper DNA methylation. Ethanol metabolism depletes key nutrients, such as folate and S-adenosylmethionine (SAM), which serve as methyl donors in DNA methylation reactions. This depletion can lead to widespread hypomethylation, promoting genomic instability and tumorigenesis. Moreover, alcohol consumption is associated with histone modifications, such as increased acetylation of histones H3 and H4, which can activate oncogenic pathways. Non-coding RNAs, including microRNAs (miRNAs), are also affected by alcohol use, with altered expression profiles observed in alcohol-related cancers. These dysregulated miRNAs can act as oncogenes or tumor suppressors, further complicating the epigenetic landscape of cancer. Betel quid chewing, a common practice in many parts of Asia and the Pacific, has been strongly associated with oral and esophageal cancers. Betel quid contains areca nut, slaked lime, and, in many cases, tobacco, all of which contribute to its carcinogenic potential. Arecoline, a major alkaloid in areca nut, has been shown to induce both genetic and epigenetic changes. DNA methylation changes are prominent in betel quid users, with hypermethylation of tumor suppressor genes such as *p16* and *DAPK* frequently observed in oral cancers. These methylation changes are accompanied by alterations in histone modifications, including increased histone H3 acetylation and H3K4 methylation, which enhance the expression of pro-tumorigenic genes [2,3].

In addition to these direct effects, betel quid chewing promotes chronic inflammation and oxidative stress, further driving epigenetic dysregulation. The chronic exposure to inflammatory mediators, such as cytokines and reactive oxygen species (ROS), can activate epigenetic enzymes like DNA methyltransferases (DNMTs) and histone deacetylases (HDACs), leading to aberrant gene expression. Non-coding RNAs also play a critical role in betel quid-induced carcinogenesis. Altered expression of miRNAs, such as miR-21 and miR-155, has been reported in oral cancers associated with betel quid use, highlighting the complex regulatory networks involved in this process. The combined effects of smoking, alcohol use, and betel quid chewing can have a synergistic impact on epigenetic regulation, further amplifying cancer risk. Epidemiological studies have consistently shown that individuals engaging in multiple high-risk behaviors exhibit a significantly greater incidence of cancers, particularly in the head-and-neck and gastrointestinal regions. This synergy likely arises from the additive or interactive effects of the epigenetic changes induced by each factor. For instance, smoking and alcohol together can exacerbate DNA methylation abnormalities, while betel quid compounds these effects through additional mechanisms involving histone modifications and miRNA dysregulation [4,5].

Conclusion

Interestingly, the reversibility of epigenetic changes offers potential opportunities for intervention and therapy. Epigenetic alterations, unlike genetic mutations, can be modulated by pharmacological agents. Drugs targeting DNMTs, such as azacitidine, and HDAC inhibitors, like vorinostat, have shown promise in reactivating silenced tumor suppressor genes and restoring normal gene expression in cancer cells. Lifestyle modifications, including smoking cessation, reduced alcohol consumption, and avoidance of betel quid, can also mitigate epigenetic damage and lower cancer risk. Emerging evidence suggests that these behavioral changes may partially reverse some epigenetic alterations, particularly in the early stages of carcinogenesis. smoking, alcohol consumption, and betel quid chewing are strongly associated with epigenetic aberrations that contribute to cancer development. These lifestyle factors induce complex changes in DNA methylation, histone modifications, and non-coding RNA expression, disrupting the regulatory networks that maintain cellular homeostasis. The cumulative and synergistic effects of

***Address for Correspondence:** Yueqi Lin, Department of Chemical Biological Sciences, University of Sonora, Hermosillo, Sonora, Mexico, E-mail: linyueqi@gmail.com*

Received: 01 October, 2024, Manuscript No. Jcrdc-24-153704; **Editor Assigned:** 03 October, 2024, PreQC No. P-153704; **Reviewed:** 18 October, 2024, QC No. Q-153704; **Revised:** 24 October, 2024, Manuscript No. R-153704; **Published:** 31 October, 2024, DOI: 10.37421/2472-1247.2024.10.330

these behaviors further amplify cancer risk, highlighting the importance of targeted prevention and intervention strategies. Understanding the epigenetic mechanisms underlying these associations provides valuable insights into the pathogenesis of cancer and offers potential avenues for therapeutic development. By addressing the epigenetic consequences of these high-risk behaviors, it may be possible to reduce the burden of cancer and improve outcomes for affected individuals.

Acknowledgement

None.

Conflict of Interest

None.

References

1. Hardy, Tabitha M. and Trygve O. Tollefsbol. "Epigenetic diet: Impact on the epigenome and cancer." *Epigenomics* 3 (2011): 503-518.
2. Bishop, Karen S. and Lynnette R. Ferguson. "The interaction between epigenetics, nutrition and the development of cancer." *Nutr* 7 (2015): 922-947.
3. Legaki, Evangelia and Maria Gazouli. "Influence of environmental factors in the development of inflammatory bowel diseases." *World J Gastrointest Pharmacol Ther* 7 (2016): 112.
4. Leung, Amy, Candi Trac, Juan Du and Rama Natarajan, et al. "Persistent chromatin modifications induced by high fat diet*♦." *J biol chem* 291 (2016): 10446-10455.
5. Lee, Ho-Sun. "Impact of maternal diet on the epigenome during in utero life and the developmental programming of diseases in childhood and adulthood." *Nutr* 7 (2015): 9492-9507.

How to cite this article: Lin, Yueqi. "The Relationship between Smoking, Alcohol Consumption and Betel Quid Chewing with Epigenetic Alterations in Cancers." *J Clin Respir Dis Care* 10 (2024): 330.