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Carcinogenic Effects of Carbon-Based Compounds: A Comprehensive Review

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Abstract: *Carbon-based compounds encompass a structurally and functionally diverse group of chemicals — ranging from classical polycyclic aromatic hydrocarbons (PAHs) and benzene to engineered nanomaterials such as carbon nanotubes (CNTs), fullerenes (C_{60}), graphene, and carbon black. Many of these compounds share a common oncogenic capacity mediated through shared molecular mechanisms: reactive oxygen species (ROS) generation, covalent DNA adduct formation, chromosomal instability, epigenetic dysregulation, and persistent inflammatory signaling. This review consolidates the current scientific understanding of the carcinogenic potential of carbon-based compounds, examining their chemical classification, routes of exposure, molecular mechanisms of genotoxicity, associated cancer types, and regulatory classification. Evidence from epidemiological, *in vitro*, and *in vivo* studies is synthesized to provide a comprehensive mechanistic and translational picture. The review concludes by identifying critical research gaps and regulatory priorities, particularly concerning novel carbon nanomaterials whose carcinogenic profiles remain incompletely characterized.*

Keywords: polycyclic aromatic hydrocarbons, carbon nanotubes, benzene, DNA adducts, oxidative stress, carcinogenesis, genotoxicity, nanomaterials

I. INTRODUCTION

Carbon is the chemical backbone of life, yet many carbon-containing compounds rank among the most potent human carcinogens ever identified. The carcinogenic properties of carbon-based compounds were first formally recognized in the early 20th century when occupational exposure to coal tar and soot was linked to scrotal and skin cancers in chimney sweeps and industrial workers. Since those foundational observations, the field has expanded dramatically to encompass hundreds of compounds spanning simple aromatic hydrocarbons, complex fused-ring systems, and, most recently, engineered carbon nanomaterials.

The International Agency for Research on Cancer (IARC) has classified numerous carbon-based compounds as Group 1 (definite human carcinogens), Group 2A (probable), or Group 2B (possible) carcinogens. Benzene, for example, is an unambiguous Group 1 carcinogen causally linked to acute myeloid leukemia (AML) and other lymphohematopoietic malignancies (Eastmond et al., 2014). Benzo[*a*]pyrene (B[*a*]P), the archetypal PAH, is classified as Group 1, while multi-walled carbon nanotubes (MWCNTs) of a specific type were reclassified as Group 2B in 2014 (Barbarino & Giordano, 2021). Graphene oxide and fullerene C_{60} remain under active toxicological evaluation.

The broad relevance of carbon-based carcinogens is underscored by their ubiquity. PAHs are generated by incomplete combustion of organic matter and are present in tobacco smoke, automobile exhaust, charcoal-broiled foods, and industrial emissions (Moorthy et al., 2015). Benzene pervades urban air and occupational environments (Jin et al., 2024). Carbon nanomaterials are increasingly incorporated into consumer goods, medical devices, and industrial processes (Zhang et al., 2020). Understanding their carcinogenic mechanisms is therefore of profound public health importance.

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This review is organized around five major themes: (1) chemical classification and sources of carbon-based carcinogens; (2) routes of human exposure; (3) molecular mechanisms of carcinogenicity; (4) cancer types and epidemiological evidence; and (5) regulatory status and future research priorities.

II. CLASSIFICATION AND SOURCES OF CARBON-BASED CARCINOGENS

2.1 Polycyclic Aromatic Hydrocarbons

PAHs are a class of fused aromatic ring compounds formed during the incomplete combustion of organic material. The U.S. Environmental Protection Agency (EPA) has identified 16 priority PAHs, ranging from two-ring naphthalene to seven-ring coronene. Among these, B[*a*]P, dibenzo[*a,l*]pyrene, 7,12-dimethylbenz[*a*]anthracene (DMBA), and 3-methylcholanthrene are the most extensively studied for carcinogenic activity (Hooven et al., 2005).

Two- and three-ring PAHs are generally considered non-carcinogenic; however, alkylated three-ring PAHs — particularly certain dimethylphenanthrenes — can induce DNA adducts and tumor formation in animal models (Le Goff et al., 2017). Carcinogenic potency increases substantially with ring number and structural features such as bay-region geometry, which facilitates metabolic activation to reactive diol epoxides (Xue & Warshawsky, 2005). Sources of human PAH exposure include tobacco smoke, vehicular exhaust, coal-tar pitch, aluminum production, carbon electrode manufacture, charcoal-broiled meats, and dietary contamination from environmental pollution (Munnia et al., 2005).

2.2 Benzene and Simple Aromatic Hydrocarbons

Benzene (C_6H_6) is the simplest aromatic hydrocarbon and one of the most widely used industrial chemicals globally. It functions as a solvent and chemical intermediate in the production of styrene, phenol, cyclohexane, dyes, and rubber (Shin et al., 2021). Human exposure occurs occupationally in petroleum refining, rubber production, printing, and shoe manufacturing, and environmentally through automobile exhaust, cigarette smoke, and indoor materials. Benzene is a definitive IARC Group 1 carcinogen, with causative links to AML, chronic lymphocytic leukemia (CLL), multiple myeloma (MM), and non-Hodgkin lymphoma (NHL) (Eastmond et al., 2014; Jin et al., 2024).

2.3 Carbon Nanomaterials

The past three decades have witnessed the rapid development and commercial deployment of carbon nanomaterials, including single-walled carbon nanotubes (SWCNTs), multi-walled carbon nanotubes (MWCNTs), fullerene C_{60}, graphene, graphene oxide (GO), and carbon black. These materials possess extraordiary physicochemical properties — exceptional tensile strength, electrical conductivity, and high surface-area-to-volume ratios — that have driven their adoption across electronics, biomedicine, materials science, and environmental remediation (Zhang et al., 2020).

SWCNTs consist of a single graphene sheet rolled into a cylinder (diameter ~0.4–2 nm), while MWCNTs comprise multiple concentric cylinders (diameter ~2–100 nm). Fullerene C_{60} is a spherical carbon allotrope of 60 carbon atoms arranged in pentagonal and hexagonal rings. Graphene is a single atomic layer of sp^2-hybridized carbon. Each of these materials exhibits distinct toxicological profiles, though all share the capacity to generate oxidative stress under biological conditions (Zhang et al., 2020; Leszczynski et al., 2012).

III. ROUTES OF HUMAN EXPOSURE

Human exposure to carbon-based carcinogens occurs via three principal routes: inhalation, ingestion, and dermal absorption. Inhalation represents the dominant pathway for occupational and environmental PAH exposure, as well as for carbon nanomaterial exposure during manufacturing and processing. Workers in aluminum smelting, coke production, and roofing industries face the highest occupational PAH burdens (Moorthy et al., 2015). CNT exposure is most relevant in nanotechnology manufacturing facilities, where aerosol release during synthesis, handling, and processing creates inhalation risk (Barbarino & Giordano, 2021).

Ingestion is a major route for dietary PAH exposure, particularly from charcoal-broiled meats, smoked fish, and contaminated drinking water. Among non-smokers without defined occupational exposure, diet may constitute the

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primary source of total PAH body burden (Munnia et al., 2005). Benzene exposure via ingestion is less significant but can occur through contaminated water supplies or food products stored in benzene-containing packaging.

Dermal absorption is relevant for workers in contact with coal tar, creosote, and mineral oils. PAH penetration through intact skin contributes meaningfully to total body burden in workers engaged in road paving, roofing, and chimney sweeping. Benzene can also be absorbed dermally from contaminated solvents and fuels, though this route is quantitatively less important than inhalation for most occupational scenarios.

IV. MOLECULAR MECHANISMS OF CARCINOGENICITY

4.1 Metabolic Activation and DNA Adduct Formation

The carcinogenicity of PAHs is not intrinsic to the parent compound but requires metabolic activation to electrophilic intermediates capable of covalently binding DNA. Three principal activation pathways have been characterized (Xue & Warshawsky, 2005):

Pathway 1 — Bay-region diol epoxide formation: Cytochrome P450 enzymes (CYP1A1, CYP1B1, and epoxide hydrolase) convert PAHs to highly reactive diol epoxides. For B[*a*]P, this yields (+)-*anti*-benzo[*a*]pyrene-7,8-diol-9,10-epoxide (BPDE), which forms stable adducts predominantly at the N^2 position of guanine residues in DNA. If unrepaired by nucleotide excision repair (NER), these bulky adducts cause miscoding during DNA replication, generating G→T transversions — a mutational signature prominently observed in *TP53* and *KRAS* in smoking-related lung cancers (Moorthy et al., 2015; Karahalil et al., 2012).

Pathway 2 — Radical cation formation: One-electron oxidation of PAHs by peroxidases and CYP enzymes generates radical cations that form depurinating adducts at N7-guanine and N3-adenine. These adducts are lost from DNA by cleavage of the glycosyl bond, leaving apurinic sites that generate cancer-initiating mutations. Cavalieri and Rogan (2014) demonstrated that the most potent carcinogenic PAHs — B[*a*]P, dibenzo[*a,l*]pyrene, and DMBA — predominantly form depurinating adducts, with the resulting apurinic sites driving mutations in Harvey-*ras* oncogenes in mouse skin papilloma models.

Pathway 3 — Ortho-quinone pathway: Aldo-keto reductases (AKRs) convert PAHdihydrodiols to catechols, which are oxidized to *o*-quinones. These quinones undergo redox cycling, generating ROS that cause oxidative DNA damage — particularly 8-oxo-7,8-dihydroguanine (8-oxoG), a highly mutagenic lesion that causes G→T transversions during replication (Xue & Warshawsky, 2005).

The biological consequences of these adducts depend on the efficiency of cellular DNA repair. Base excision repair (BER) handles oxidative lesions such as 8-oxoG, while NER removes bulky adducts. Polymorphisms in repair genes (e.g., *OGG1*, *XRCC1*, *XPD*) modulate individual cancer susceptibility (Karahalil et al., 2012).

4.2 Oxidative Stress and Reactive Oxygen Species

Oxidative stress — defined as an imbalance between ROS generation and antioxidant defense capacity — is a central mechanism linking carbon-based compound exposure to carcinogenesis. Toyokuni (2008) synthesized epidemiological and molecular evidence demonstrating that chronic oxidative conditions, whether induced by asbestos, iron overload, tobacco smoke, or chemical carcinogens, consistently precede carcinogenesis through accumulation of genotoxic damage.

For CNTs, oxidative stress is the dominant mechanistic pathway. Loft et al. (2014) demonstrated that CNT exposure depletes cellular antioxidants, elevates intracellular ROS production, and activates pro-inflammatory signaling in immune cells, epithelial cells, endothelial cells, and stromal cells. Pre-treatment with antioxidants attenuates these responses, confirming ROS dependency. CNT-mediated oxidative stress produces elevated lipid peroxidation products and oxidatively damaged DNA nucleobases — particularly in the lungs and liver — concomitant with depletion of glutathione (GSH), superoxide dismutase (SOD), and catalase (Loft et al., 2014).

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Benzene exerts its myelotoxic and leukemogenic effects partly through ROS generated by its reactive metabolites. Hydroquinone and 1,4-benzoquinone — the most toxic benzene metabolites — undergo redox cycling in bone marrow cells, generating superoxide and hydrogen peroxide that damage hematopoietic progenitor cell DNA (Winn & Badham, 2010). Mitochondrial dysfunction, including depolarization of the mitochondrial membrane potential and cytochrome C release, is an additional consequence of benzene metabolite-induced oxidative stress (Zhu et al., 2018).

4.3 Chromosomal Instability and Aneuploidy

A mechanistically distinct carcinogenic pathway for carbon nanomaterials involves physical disruption of the mitotic spindle apparatus. CNTs, owing to their high-aspect-ratio tubular morphology and structural similarity to cellular microtubules, can interact with centrosomes and spindle fibers, causing monopolar, tripolar, and quadripolar chromosomal divisions. The resulting aneuploidy — gain or loss of whole chromosomes — is a recognized mechanism of carcinogenesis independent of direct DNA sequence mutation (Reynolds et al., 2010).

Reynolds et al. (2010) reviewed *in vitro* genotoxicity studies demonstrating that CNT exposure causes DNA fragmentation, chromosomal mutations, and mitotic spindle disruption in pulmonary cells. This mechanism is particularly concerning because it can operate at sub-cytotoxic doses and may be missed by standard mutagenicity assays that focus on point mutations rather than chromosomal number errors.

4.4 Inflammatory Signaling and Tumor Promotion

Chronic inflammation is a well-established tumor-promoting environment. CNTs induce a sustained inflammatory response in the lung resembling the pathological cascade triggered by asbestos — a paradigmatic carcinogen. Barbarino and Giordano (2021) reviewed evidence that CNT exposure causes persistent pulmonary inflammation, fibrosis, mesothelial hyperplasia, and ultimately mesothelioma in *in vivo* models, with molecular alterations — including activation of NF-κB, AP-1, and STAT3signaling — consistent with those observed in asbestos-induced mesothelioma.

PAHs also exerttumor-promoting effects beyond their role as initiators. Hooven et al. (2005) demonstrated that PAHs cause changes in cellular gap-junction communication analogous to those induced by the tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA), suggesting a promotional mechanism that operates in addition to direct genotoxicity. This dual initiator-promoter capacity may explain the potency of PAH-rich mixtures such as tobacco smoke and coal tar.

4.5 Epigenetic Mechanisms

Emerging evidence implicates epigenetic dysregulation as an additional carcinogenic mechanism for carbon-based compounds. Benzene exposure has been associated with altered DNA methylation patterns in peripheral blood cells of occupationally exposed workers, including gas station attendants and traffic police (Winn & Badham, 2010). Aberrant promoter methylation of tumor suppressor genes — including *RASSF1A* and *RARβ2* — has been detected in bronchial biopsies and serum of smokers exposed to PAH-rich tobacco smoke, with prognostic relevance for lung cancer relapse (Gratchev et al., 2015).

Tobacco smoke-derived PAH metabolites induce oxidative stress that alters histone modification patterns, disrupts chromatin remodeling, and modulates microRNA expression profiles. Gratchev et al. (2015) identified significant differences in the expression of miR-218, miR-130a, and miR-143 between smoking and non-smoking lung cancer patients, suggesting that PAH-induced epigenetic reprogramming contributes to the tumor microenvironment. These epigenetic changes are potentially heritable through cell division, providing a mechanism for stable transmission of a pro-tumorigenic phenotype even after removal of the initial carcinogenic stimulus.

V. ASSOCIATED CANCER TYPES AND EPIDEMIOLOGICAL EVIDENCE

5.1 Lung Cancer

Lung cancer is the malignancy most strongly and consistently associated with PAH exposure. Excessive exposure to PAHs — particularly B[*a*]P from tobacco smoke and occupational sources — is a leading etiological factor for lung cancer, the malignancy with the highest cancer mortality in the United States (Moorthy et al., 2015). PAHs induce

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CYP1A1/1B1 and related metabolic enzymes via the aryl hydrocarbon receptor (AhR), generating diol epoxides, radical cations, and *o*-quinones that form DNA adducts, drive *TP53* mutations, and alter gene expression profiles toward tumorigenesis. Polymorphisms in *CYP1A1*, *GSTM1*, *GSTT1*, and DNA repair genes modulate lung cancer susceptibility across populations of different ethnicity, gender, and age (Moorthy et al., 2015).

CNT-exposed workers face an additional lung cancer risk. *In vivo* studies have documented pulmonary fibrosis, granulomatous inflammation, and neoplastic changes following CNT inhalation, with the inflammatory and oxidative milieu providing a tumor-promoting microenvironment (Loft et al., 2014; Barbarino & Giordano, 2021).

5.2 Mesothelioma

Mesothelioma — a rare and highly aggressive malignancy of the pleural, peritoneal, or pericardial mesothelium — has been specifically associated with long-fiber, rigid MWCNTs that exhibit biopersistence and frustrated phagocytosis analogous to crocidolite asbestos. IARC's 2014 classification of MWCNT-7 as a Group 2B carcinogen was based on *in vivo* evidence of mesothelial hyperplasia and mesothelioma following intraperitoneal injection in rodent models (Barbarino & Giordano, 2021). The molecular alterations observed — including *BAP1* inactivation, NF2 loss, and CDKN2A deletion — mirror those found in asbestos-induced mesothelioma.

5.3 Leukemia and Lymphohematopoietic Cancers

Benzene is the prototypical hematological carcinogen. Epidemiological evidence from cohort studies of petroleum workers, rubber industry workers, and shoe manufacturers demonstrates dose-dependent increases in AML, CLL, MM, and NHL (Eastmond et al., 2014; Mehlman, 2006). The Chinese Benzene Cohort Study, one of the largest occupational cohorts, provided quantitative dose-response data demonstrating leukemia risk even at exposures below 1 ppm — well within the range encountered in many occupational settings (Jin et al., 2024). The critical carcinogenic events occur in bone marrow hematopoietic stem cells and progenitor cells, where benzene metabolites generate ROS-mediated DNA damage and chromosomal aberrations that activate oncogenes or inactivate tumor suppressor genes (Eastmond et al., 2014).

5.4 Skin and Bladder Cancer

Occupational exposure to coal tar, creosote, and mineral oils — all rich in PAHs — is causally associated with skin squamous cell carcinoma, particularly in workers in roofing, road paving, and chimney sweeping. The carcinogenic PAHs in these mixtures form DNA adducts in keratinocytes, initiating the sequence of mutations that leads to malignant transformation.

Bladder cancer risk is elevated in workers exposed to aromatic amines — particularly 4-aminobiphenyl (4-ABP) and benzidine — which are structurally related to PAHs and metabolized through similar activation pathways to form DNA adducts in urothelial cells (Ghanghro, 2022). 4-ABP, classified as a Group 1 carcinogen by IARC, was formerly used in the rubber and dye industry; its metabolite N-hydroxyl ABP forms DNA adducts, and elevated 4-ABP-DNA adduct levels have been documented in hepatocellular carcinoma patients (Ghanghro, 2022).

VI. REGULATORY STATUS AND RISK ASSESSMENT

The IARC Monographs Programme provides the most authoritative classification of carbon-based carcinogens. Benzene and B[*a*]P are Group 1 definite human carcinogens. MWCNT-7 is classified as Group 2B (possibly carcinogenic to humans), while other CNT types remain unclassified due to insufficient evidence, partly reflecting the physicochemical heterogeneity of the CNT class (Barbarino & Giordano, 2021). Graphene oxide and fullerene C_{60} lack formal IARC classification, though *in vitro* studies indicate dose-dependent cytotoxicity and oxidative stress potential (Leszczynski et al., 2012; Zhang et al., 2020).

Occupational exposure limits (OELs) have been established for benzene (0.5 ppm in many jurisdictions) and B[*a*]P (as a surrogate for PAH mixtures), but regulatory frameworks for carbon nanomaterials lag behind their commercial deployment. The European Union's REACH regulation currently treats fullerenes under the same guidelines as bulk carbon, despite evidence that nano-scale C_{60} exhibits biological activities distinct from graphite (Zhang et al., 2020).

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This regulatory gap represents a significant public health concern given the accelerating production and use of carbon nanomaterials.

Quantitative risk assessment for benzene-induced leukemia employs the linearized multistage (LMS) model. Jin et al. (2024) applied this model to data from the Chinese Benzene Cohort Study, demonstrating that leukemia risk is detectable at cumulative exposures below 3 mg/m³·year — the current Chinese OEL — reinforcing calls for further tightening of exposure standards.

VII. FUTURE RESEARCH DIRECTIONS

Several critical gaps in knowledge warrant priority research attention. First, the carcinogenic classification of most CNT types remains uncertain due to limited human epidemiological data and the challenge of characterizing exposure in nanotechnology workforces. Prospective cohort studies of CNT-exposed workers, with biomarker-based exposure assessment, are urgently needed (Barbarino & Giordano, 2021).

Second, the carcinogenic potential of graphene and graphene oxide requires more systematic evaluation. While graphene-induced oxidative stress is demonstrably lower than that of CNTs or fullerenes (Leszczynski et al., 2012), the respiratory hazard of graphene nanoplatelets — which exhibit aerodynamic behaviors distinct from other nanomaterials — has been highlighted as a specific concern requiring dedicated *in vivo* studies.

Third, the epigenetic mechanisms of PAH and benzene carcinogenicity are incompletely mapped. High-resolution epigenome-wide association studies (EWAS) in occupationally exposed populations could identify methylation biomarkers predictive of cancer risk, enabling earlier intervention and improved surveillance.

Fourth, the interaction between carbon-based carcinogens and the tumor microenvironment — including effects on tumor-infiltrating lymphocytes, PD-L1 expression, and immune evasion — represents a frontier of translational relevance, given the growing clinical importance of immune checkpoint inhibition in carcinogen-associated cancers.

VIII. CONCLUSION

Carbon-based compounds span a spectrum of carcinogenic potency, from definitively established human carcinogens such as benzene and B[*a*]P to nanomaterials whose carcinogenic profiles are still being elucidated. Despite their structural diversity, these compounds converge on shared molecular mechanisms: metabolic activation to electrophilic intermediates, covalent DNA adduct formation, ROS-mediated oxidative DNA damage, chromosomal instability, persistent inflammation, and epigenetic dysregulation. The cancer types most consistently linked to carbon-based compound exposure — lung cancer, mesothelioma, leukemia, and bladder cancer — reflect the routes of exposure and the tissue-specific metabolism of these agents.

The rapid expansion of carbon nanomaterial production demands urgent acceleration of toxicological research, particularly for CNTs, graphene, and fullerenes. Regulatory frameworks must evolve to reflect the nano-specific hazard profiles of these materials rather than treating them as equivalent to bulk carbon. Biomarker-based surveillance of exposed populations, combined with mechanistic studies integrating genomic, epigenomic, and proteomic approaches, offers the most promising path toward accurate risk characterization and effective cancer prevention in the era of carbon nanotechnology.

REFERENCES

1. Barbarino, M., & Giordano, A. (2021). Assessment of the carcinogenicity of carbon nanotubes in the respiratory system. *Cancers*, *13*(6), 1318. https://doi.org/10.3390/cancers13061318
2. Cavalieri, E., & Rogan, E. (2014). The molecular etiology and prevention of estrogen-initiated cancers. *Molecular Aspects of Medicine*, *36*, 1–55. https://doi.org/10.1016/j.mam.2013.08.002
3. Eastmond, D. A., Keshava, N., & Sonawane, B. (2014). Lymphohematopoietic cancers induced by chemicals and other agents and their implications for risk evaluation: An overview. *Mutation Research – Reviews in Mutation Research*, *762*, 1–17. https://doi.org/10.1016/j.mrrev.2014.04.001
4. Ghanghro, L. (2022). Chemical risk factors of primary liver cancer: A short comment. *Hepatic Medicine: Evidence and Research*, *14*, 1–3. https://doi.org/10.2147/hmer.s350076

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Impact Factor: 8.2

5. Gratchev, A. N., Kzhyshkowska, J. G., Buldakov, M. A., Kokova, D. A., Cherdyntseva, N. V., Grigoryeva, E. S., &Cherdyntsev, E. S. (2015). Smoking-related DNA adducts as potential diagnostic markers of lung cancer: New perspectives. *Experimental Oncology*, *37*(1), 5–12. https://doi.org/10.31768/2312-8852.2015.37(1):5-12
6. Hooven, L. A., Baird, W. M., & Mahadevan, B. (2005). Carcinogenic polycyclic aromatic hydrocarbon–DNA adducts and mechanism of action. *Environmental and Molecular Mutagenesis*, *45*(2–3), 106–114. https://doi.org/10.1002/em.20095
7. Jin, K., Wu, B., Li, M., Huang, D., Xing, C., Zhu, F., Wang, X., Cheng, X., & Li, M. (2024). Leukemia risk assessment of exposure to low-levels of benzene based on the linearized multistage model. *Frontiers in Public Health*, *12*, 1355739. https://doi.org/10.3389/fpubh.2024.1355739
8. Karahalil, B., Wilson, D. M., & Bohr, V. A. (2012). Impact of DNA polymorphisms in key DNA base excision repair proteins on cancer risk. *Human & Experimental Toxicology*, *31*(10), 981–1005. https://doi.org/10.1177/0960327112444476
9. Le Goff, J., Pampanin, D. M., Meier, S., Brooks, S. J., Sydnes, M. O., &Grøsvik, B. E. (2017). DNA adducts in marine fish as biological marker of genotoxicity in environmental monitoring: The way forward. *Marine Environmental Research*, *124*, 1–13. https://doi.org/10.1016/j.marenvres.2017.01.002
10. Leszczynski, J., Urbaszek, P., Gajewicz, A., Leszczynska, D., Rasulev, B., Puzyn, T., &Dinadayalane, T. C. (2012). Advancing risk assessment of engineered nanomaterials: Application of computational approaches. *Advanced Drug Delivery Reviews*, *64*(15), 1663–1693. https://doi.org/10.1016/j.addr.2012.05.014
11. Loft, S., Cao, Y., Roursgaard, M., Jacobsen, N. R., Hemmingsen, J. G., Kermanizadeh, A., … Møller, P. (2014). Role of oxidative stress in carbon nanotube-generated health effects. *Archives of Toxicology*, *89*(9), 1525–1539. https://doi.org/10.1007/s00204-014-1356-x
12. Mehlman, M. A. (2006). Dangerous and cancer-causing properties of products and chemicals in the oil refining and petrochemical industries. Part XXX. *Annals of the New York Academy of Sciences*, *1076*(1), 110–119. https://doi.org/10.1196/annals.1371.065
13. Moorthy, B., Carlin, D. J., & Chu, C. (2015). Polycyclic aromatic hydrocarbons: From metabolism to lung cancer. *Toxicological Sciences*, *145*(1), 5–15. https://doi.org/10.1093/toxsci/kfv040
14. Munnia, A., Ibanez, R., Amiano, P., Barricarte, A., Agudo, A., Tormo, M. J., … Peluso, M. (2005). Reliability of bulky DNA adducts measurement by the nuclease P1 ^{32}P-post-labelling technique. *Biomarkers*, *10*(1), 56–70. https://doi.org/10.1080/13547500500050580
15. Reynolds, S. H., Sargent, L. M., & Castranova, V. (2010). Potential pulmonary effects of engineered carbon nanotubes: *In vitro* genotoxic effects. *Nanotoxicology*, *5*(2), 196–210. https://doi.org/10.3109/17435390.2010.500444
16. Shin, S. S., Nam, D. J., Ryoo, J.-H., Lee, H. C., & Yang, E. H. (2021). Association between urinary trans,trans-muconic acid and diabetes: A cross-sectional analysis of data from Korean National Environmental Health Survey (KoNEHS) cycle 3. *Annals of Occupational and Environmental Medicine*, *33*, e35. https://doi.org/10.35371/aoem.2021.33.e35
17. Toyokuni, S. (2008). Molecular mechanisms of oxidative stress-induced carcinogenesis: From epidemiology to oxygenomics. *IUBMB Life*, *60*(7), 441–447. https://doi.org/10.1002/iub.61
18. Winn, L. M., & Badham, H. J. (2010). *In utero* and *in vitro* effects of benzene and its metabolites on erythroid differentiation and the role of reactive oxygen species. *Toxicology and Applied Pharmacology*, *244*(3), 273–279. https://doi.org/10.1016/j.taap.2010.01.002
19. Xue, W., & Warshawsky, D. (2005). Metabolic activation of polycyclic and heterocyclic aromatic hydrocarbons and DNA damage: A review. *Toxicology and Applied Pharmacology*, *206*(1), 73–93. https://doi.org/10.1016/j.taap.2004.11.006
20. Zhang, C., Liang, Q., Shao, B., Liu, Y., Peng, Z., Zhang, W., … Liu, Z. (2020). Advances in the application, toxicity and degradation of carbon nanomaterials in environment: A review. *Environment International*, *134*, 105298. https://doi.org/10.1016/j.envint.2019.105298

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International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
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21. Zhu, H., Zhang, C., Yang, X., Yu, X., Gao, J., Sun, S., Yan, H., & Zhang, Q. (2018). PINK1/Parkin-mediated mitophagy was activated against 1,4-benzoquinone-induced apoptosis in HL-60 cells. *Toxicology in Vitro*, *51*, 1–8. https://doi.org/10.1016/j.tiv.2018.03.002