



Cancers in the Urban Environment: How Malignant Diseases are Caused and Distributed Among the Diverse People and Neighborhoods of a Major Global Metropolis, 2nd Edition. Thomas M. Mack. Academic Press, An imprint of Elsevier, 125 London Wall, London EC2Y 5AS, UK. 2021. xii + 1136 pages. Price: US\$ 175. ISBN: 9780128117453.

This book is an interesting publication which integrates elementary knowledge of cancer, its types and occurrence in a complex urban environment. It is a unique collection of statistical datasets of different cancers, which portrays their demographic and geographic patterns in a defined area. The methodology adopted is sound and most of the work presented is easy to understand for the medical and scientific fraternity. Surely, it will assist to recognize the specific patterns and causal hypothesis of cancers in an urban environment.

Urbanization has caused undeniable damage to the environment. The accumulated knowledge of urban ecology strongly suggests that the increasing anthropogenic activities due to urbanization deteriorate the quality of the surrounding environment. This exposes humans to a broad spectrum of pollutants, which may result in the generation of different life-threatening diseases, including cancer, a leading cause of human mortality. One such adverse environmental effect that has recently received a lot of attention and has been recognized as a serious public health hazard is air pollution. The increasing industrialization, intense consumption of energy and excessive vehicle exhaust emission have resulted in the release of a large number of pollutants in the air. According to a study on the global burden of diseases, air pollution is among the primary causes of premature mortality and morbidity. Along

with the other pollutants, particulate matter (PM) generated due to gas-to-particle translation or direct atmospheric release is a major contributor of different non-communicable disease forms, including cancer. PM is an intricate mixture of small particles and gaseous molecules such as smoke, nitrates, sulphates, organic chemicals, dust particles, etc. PM can be broadly divided into three subclasses, i.e. coarse particles (PM10), fine particles (PM2.5) and ultra-fine particles (PM0.1). The coarse particles, which are larger in size largely get attached to the respiratory tract and influence the pulmonary functions, whereas the smaller ones can translocate and affect other body parts. Moreover, the larger surface area and smaller size assist these tiny particles to activate a number of biological response mechanisms. These disturbances can further result in various alterations at the level of genome and epigenome. At the genomic level, this can be observed as perturbed DNA repair response and abnormal gene function, while at the epigenomic level it may disturb the DNA methylation/demethylation process, expression of microRNAs and post-translation histone modifications. The organic and metallic compounds present within PM can potentially generate free radicals, disturb the mitochondrial machinery and trigger a DNA damage response, which is regulated via phosphoinositide 3-kinases signalling pathways. This further deregulates the mitochondrial–nuclear crosstalk and alters the vital epigenetic programming. The complex signalling processes among mitochondria and nucleus are of bidirectional nature. While several regulatory mediators of mitochondrial mechanisms are encoded by the nucleus, the mitochondrial genome regulates the important molecules required for various key mechanisms like oxidative phosphorylation. The disturbances in this nuclear to mitochondrial (anterograde) and mitochondrial to nuclear (retrograde) signalling mechanisms can consequently result in an epigenomic imbalance. In this multifaceted process of mitochondrial-induced epigenetic modifications, the nuclear factor kappa-light-chain-enhancer of activated β (NF- $\kappa\beta$) cells acts as an inflammatory switch. Recent population-based studies strongly suggest that the individuals residing in high-pollution areas have disturbed epigenomic machinery in comparison to those living in low-pollution zones. Importantly, epigenetic modifications bridge the multifaceted set-up of gene–environment interfaces; therefore, such disturbances can

affect the tightly regulated transcriptomic machinery and alter the specific gene-expression patterns. This further interferes with distinct cellular functions, which may result in cancer. In addition, the intricate composition of PM comprises several chemicals that have a significant positive association with the occurrence of cancer processes or associated symptoms. These chemicals can also mimic the growth hormones that assist in higher proliferation of cancer cells. However, an important point to note herein is that the physico-chemical properties, composition and time of PM exposure have a direct correlation with the PM-associated disturbances. In view of all this, the International Agency for Research in Cancer, Lyon, France has classified PM2.5 as a class-I carcinogen. Based on the serious health effects of PM exposure, the World Health Organization, Geneva, has established air-quality guidelines which clearly recommend that the maximum annual average PM2.5 exposure should not surpass $10 \mu\text{g}/\text{m}^3$. Nevertheless, more than 91% of the total world's population lives in areas which cross these levels and do not favour a healthy lifestyle.

As the connection of different cancers with developing urban environments is a matter of serious concern, this book is well-timed and adequately presented. The contents of the book are suitably designed to provide simple access to a broad range of statistical datasets. The chapters are precise and define the topics with clarity, avoiding unnecessary details. The book provides an exhaustive account of the different malignancy patterns that occurred in the Los Angeles (LA) County, USA, over a given period. Details related to age, sex, social status, locality, ethnicity and time are appropriately included. In view of the fact that people living in the LA County are of mixed origin, the book offers a useful collection of information about different geographical patterns of cancer incidences observed in a large urban population. Overall, the contents can be divided into three parts. Chapters 1 and 2, which form the initial part briefly, define cancer, its types and environmental impact, while the middle part of the book that comprises chapters 3–20 discusses different resources, methods and errors in the calculation of disease risk at the local level. These chapters also provide an in-depth account of data interpretation that could be useful for researchers of cancer epidemiology. The last part of the book that starts from chapter 21 is the most important, which summarizes

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information for each type of cancer. This section attempts to explain different statistical datasets of cancer types in a defined area. It also provides useful information about the non-random occurrence of different cancer types with comprehensive demographical details. Perhaps, the common aim of all chapters is to improve the current knowledge about the implications of disturbed gene–environment interactions and provide the reader a simple understanding of the cancer occurrence. To sum up, the book is a thoughtful effort and can act as a valuable source of information for

the scientific and medical community to design and develop a causal hypothesis of cancer risk patterns in an urban environment. Of course, it is common belief that such molecular epidemiological datasets may not necessarily reflect the molecular underpinnings and often fail to establish dose–response relationship from an environmental health perspective. Nevertheless, in the present era, where higher rates of urbanization, deteriorating environmental conditions and frequency of non-communicable diseases are intimately linked, this book offer readers novel insights of cancer

epidemiology. It is a valuable collection of knowledge for young researchers, biomedical scientists, clinicians and public-health professionals.

PRADYUMNA KUMAR MISHRA*
ARPIT BHARGAVA

*Department of Molecular Biology,
ICMR-National Institute for Research
in Environmental Health,
Bhauri,
Bhopal 462 030, India
e-mail: pkm_8bh@yahoo.co.uk
