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P8. EFFECTS OF OCCUPATIONAL CANCERS ON GENE REGULATION

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Cancer is the second leading cause of death worldwide, right behind cardiovascular disease. Occupational and environmental cancers are about 10% of all cancer cases. More than 152.000 deaths occur annually by reason of occupational cancers. Particular groups of working population such as miners, dye workers, smelter workers, chemical workers and more of the same is under much risk than the general population, because of high and frequent exposure to some specific carcinogens such as radium, radon, benzidine, arsenic, asbestos, benzene, chloroethers, UV etc. As a result of this high exposure, some types of cancer may develop especially as lung, bladder and skin cancers and leukemia.

Numerous experimental models have shown that environmental exposure may alter and disrupt the regulation of genome. DNA methylation and promoter hypermethylation is very important for DNA repair, apoptosis, cell cycle control, angiogenesis and carcinogen metabolism genes. Depletion of S-adenosylmethionine (SAM) and promoter hypermethylation of p53 occurs in selenium and arsenic related liver and colon cancers. Exposure to asbestos causes miRNA dysregulation in lung cancer. Airborne benzene exposure is related to hypermethylation of LINE-1, Alu and p15. Ionizing radiation induce the transcriptional changes and transcriptional silencing in genes such as cell cycle regulation/proliferation, ubiquitin cycle and DNA repair. Additionally, heavy metals such as cadmium, nickel, and zinc inhibit DNA methyltransferase (DNMT) activity and this causes a decrase in the level of DNA methylation.

The effects of occupational cancers on the genome are still ill-defined and a lot of research is done about this area.

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