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THE INTERPLAY OF ENVIRONMENTAL AND GENETIC FACTORS ASSOCIATED WITH PROSTATE CANCER

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Abstract: Prostate cancer (PCA) is among the top four most common cancers in both sexes combined and the second most common cancer in men. Exposure to environmental hazards has been associated with different cancers in humans. Genetic risk factors have also shown to contribute to the cause of PCA. Since there is not a unique environmental or genetic risk factor that can be responsible or associated with PCA alone, it is vital to identify the genetic risk as well as the environmental risk factors that can contribute together to initiate the prostate tumor to grow. Several studies and literature reviews have been conducted to investigate the roles of environmental and genetic risk factors to determine their association on PCA. Based on our reviews and studies, we have found an extensive amount of information about susceptible genetic and environmental risk factors for PCA that has assisted us to examine the consequences of the interplay of these factors on the cause of PCA initiation. We tried to tabulate all the identified genetic (i.e., susceptible genes or alleles) and environmental (e.g., cigarette smoking, air pollution, pesticide exposure, or heavy metal poisoning) risk factors for comparative studies side by side to understand the interaction and interplay of these risk factors. So far, we found that obesity, diabetes, and genetic polymorphisms in some key genes have phenotypic correlation in PCA. Also, environmental risk factors (e.g., cigarette smoking) do have detrimental effects in the genetic or epigenetic level on the adverse outcome of PCA. There really are gene- environment interactions and interplays associated with PCA, "the greatly overexpressed KRAS in the malignantly transformed prostate cell due to chronic Cadmium or Arsenic exposure" is just another example. In addition, the prostate is exposed to environmental and endogenous stress and increasing age is another significant risk factor for PCA. DNA methylation, genomic imprinting, and histone modifications are examples of epigenetic factors known to undergo change in the aging and cancerous prostate. Our study can provide an overall and clear view of how aging, environmental, and genetic/epigenetic factors interact and interplay for the occurrence of PCA. Furthermore, an expression quantitative trait loci (eQTL) analysis on gene expression and SNP data from huge microarray and NGS datasets would be performed to identify more de novo genetic alleles that might play a bigger role on PCA initiation. A list of the Bioinformatics software used for this project would be also surveyed and evaluated.

Keywords: Microarray, next-generation sequencing (NGS), single nucleotide polymorphism (SNP), gene expression, quantitative trait loci (QTL), gene-environment interaction, epigenetic, aging, prostate cancer (PCA)

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