

The Impact of Cancer Screening on the Disease Progressions

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DESCRIPTION

Cancer interception is the process of actively inhibiting the development of cancer by preventing premalignancy from progressing to invasive disease. The insufficient understanding of the earliest molecular events associated with lung carcinogenesis, the lack of preclinical models of pulmonary premalignancy, and the difficulty of developing highly sensitive and specific methods for early detection are the rate-limiting steps for effective lung cancer interception. Developments in next-generation genomics, computational techniques, and a renewed focus on precision medicine and immuno-oncology have all aided recent advancements in clinical diagnosis. The present level of knowledge in the fields of molecular defects in lung cancer progression, preclinical human models of lung cancer pathogenesis, and breakthroughs in early lung cancer detection are described in this paper.

The purpose of cancer screening is to improve the chances of a cure by discovering the malignancy, or its precursor lesion, before symptoms appear, when therapy is most beneficial. Wilson and Jungner established a framework for analysing the function of lung cancer screening that includes six critical criteria to examine when evaluating the significance of screening for a specific condition. Knowledge about the disease's natural development, the performance characteristics of existing screening tests, and the availability of viable treatment techniques for early-stage disease are among these aspects.

The necessity for an identifiable latent or preclinical stage of disease is one of Wilson and Junger's core principles. The most effective cancer screening methods are now being used in cervical and colon cancers, where studies have shown a distinct period of asymptomatic preclinical disease. The evidence for carcinogenesis in these cancers are taken together, points to a gradual development to more invasive and advanced stages of cancer. Screening for these two malignancies enables for the early detection and treatment of precursor lesions (e.g., cervical intraepithelial neoplasia with cervical cancer screening and colonic polyps with colorectal cancer screening).

Lung carcinogenesis is a multi-step process in which genetic mutations and epigenetic modifications impact cellular activities such as proliferation, differentiation, invasion, immunological response, and metastasis. Molecular findings update a progressive lung carcinogenesis model in which the establishment of a cancerized field-the accumulation of molecular abnormalities from repetitive injury-leads to genetically and epigenetically changed cells that play a crucial role. Early lung cancer lesions are polyclonal, and late-stage disease has genetic variants, indicating that tumour progression is mediated by large-scale genomic rearrangements.

Due to a lack of methodologies for individualized risk assessment, existing patient selection criteria for screening, based on the NLST and NELSON, necessitate screening a wide proportion of the population. The widespread use of LDCT in the "high-risk" population increases the potential of false-positive detection, resulting in numerous diagnostic and treatment challenges for doctors, as well as confusion, worry, and injury to patients from unnecessary procedures. According to studies, using age and smoking history alone is less effective than using "personalised" risk models that incorporate additional characteristics (e.g., COPD, family history, race), which increases the prediction of future lung cancer development.

Understanding the molecular basis for lung cancer aetiology and techniques for molecular early detection are rising rapidly, as are technology for disease monitoring, noninvasive prognostic assessment, and therapy response prediction. The use of LDCT screening in clinical practice is becoming more common, but a better knowledge of molecular risk factors and noninvasive diagnostic tests to aid in the management of indeterminate pulmonary nodules is still required.

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