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Non-smoking causes of primary lung cancer

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Up to 25% of lung cancer cases worldwide can arise in never smokers. As fewer people smoke in the West, it is now important to consider the non-smoking causes of primary lung carcinoma, as steps will be needed to identify susceptible individuals early, so prevention and/or early diagnosis can be made. We will consider if there is a predisposition for individual (histological) types of carcinoma, polymorphisms may account for differences in response to exposure and if there is a familial predisposition to develop lung cancer. Second-hand smoke alone is not thought to induce cancer, without other factors. Different polymorphisms are described for smokers and recently in never smokers. Epigenetic factors have also been discovered. Occupational factors are considered, such as asbestos and if there needs to be fibrosis for the carcinoma to develop. Nickel, Chromium, Beryllium, Radon and chlorinated solvents will be mentioned. Lifestyle effects, such as cooking (fumes in poorly-ventilated areas from oils), and coal fume exposure, which is possibly due to a polymorphism of glutathione S-transferase. Lifestyle effects such as obesity, dietary factors, including the intake of vegetables with antioxidant properties (carrots, greens), smoking marijuana, bidis and the effect of social class will be discussed, as well as cancers arising post chemotherapy, single nucleotide polymorphisms can affect response to chemotherapy and post radiotherapy.

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A phenotype-based approach to validate biomarkers and identify molecular targets in cancer

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Current methods to predict anti-cancer drug efficacy in humans is often inaccurate in clinical trials. Our novel "disease in a dish" approach overcomes challenges associated with identifying key, disease-relevant targets, in order to rationally develop (combinations of) targeted therapeutics. The ineffectiveness of treatment is in large part attributable to both inter- and intra tumor heterogeneity. Our approach uncovers the molecular basis of aggressive tumor cell phenotypes, directly from individual clinical bio specimens. We segregate tumor cell subsets that are more competent than others for mediating aggressive behavioral properties (form tumors, metastasize, and/or resist therapy). Collectively, these properties have been attributed to "cancer stem cells" or CSC. We have developed novel processes and methods to extract CSC from clinical bio specimens in primarily cultures, and have been able to validate that CSC subsets exhibit distinctive behavioral properties, which can then be directly associated with molecular differences in the same bio specimen.

1. Personalized targeted therapy is important for the effective treatment of cancers; however, it requires the identification and validation of relevant molecular drivers.
2. Our approach introduces phenotypic bioassays for both driver discovery and target validation.
3. Advanced stage disease does not prohibit (and may be advantageous) for associating specific biomarkers with functional phenotypes.
4. Biological discovery that emphasizes the design of appropriate functional bioassays to characterize aggressive cancer cell phenotypes and molecular biology may enable us to rationally halt tumor progression.

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