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Pancreatic cancer and genetics: Where are we heading to?

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The incidence of pancreas cancer ranges between 1 to 10 cases per 100,000 persons. The incidence rate is higher in Western countries like the United States. Approximately 40,000 persons died from pancreas cancer in 2014 in the United States, making this the 4th most common cause of cancer death in women and men. Only 20% of patients with PC present at diagnosis time with localized disease that can be treated surgically, but the overall 5 years survival is 6% even for the patient who underwent surgical resection. In patients with first degree relative with PC the risk for developing PC increased by factor of 2, 6 and 30 for 1, 2 or 3 first degree relatives respectively. Breast and ovarian cancer syndrome is caused by mutation in different genes such as: BRCA1, BRCA2, PALB2 and ATM. BRCA1 and BRCA2 also increase the risk for prostate cancer and PC by 4-7 folds for patients carrying the mutation. Furthermore carriers of DNA mismatch repair (MMR) genes: MLH1, MSH2, MSH6 and PMS2 have increased risk to develop PC 8 times more compared to the general population. Other Genes that play role in other familial syndromes also were found to increase the risk of PC such as: APC, CDK2NA (P16), PRSS1; STK11and TP53. Identifying germ-line mutations that increase the risk of pancreas cancer has substantial benefits for both patient and their unaffected relatives. Pancreas cancer patients with BRCA1 and BRCA2 mutations are more likely to benefit from PARP inhibitors and platinum-based chemotherapy compared with those without mutations.

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Why the hamster pancreatic cancer model is most valuable for clinical studies

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Despite advances in genetic and clinical studies, the etiology, histogenesis, early diagnosis, prevention and therapy of the disease have remained a challenge. The recent shift from chemical carcinogenesis to molecular carcinogenesis and the introduction of the transgenic mouse model, which has dominated recent pancreatic cancer research, have buried the valuable information gained from the hamster pancreatic cancer model. It is time to reemphasize the important role of the hamster model for clinical studies by highlighting the comparative data on clinical symptoms, the morphology, biology, antigenicity, genetic, metabolic alterations and histogenesis of the tumors. In addition, references will be made to numerous studies related to early diagnosis, prevention, therapy and the nutritional role in pancreatic carcinogenesis.

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