Cancer and Heredity Literature

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Abstract: Cancer is the general name for a group of more than 100 diseases. Although there are many kinds of cancer, all cancers start because abnormal cells grow out of control. Untreated cancers can cause serious illness and death. The body is made up of trillions of living cells. Normal body cells grow, divide, and die in an orderly fashion. During the early years of a person's life, normal cells divide faster to allow the person to grow. After the person becomes an adult, most cells divide only to replace worn-out or dying cells or to repair injuries. This paper collects literatures of cancer and heredity.
[Smith MH. **Cancer and Heredity Literature.** *Cancer Biology* 2011;1(3):77-83]. (ISSN: 2150-1041). http://www.cancerbio.net. 4

Keywords: cancer; biology; research; life; disease; heredity

1. Introduction

Cancer is the general name for a group of more than 100 diseases. Although there are many kinds of cancer, all cancers start because abnormal cells grow out of control. Untreated cancers can cause serious illness and death. The body is made up of trillions of living cells. Normal body cells grow, divide, and die in an orderly fashion. During the early years of a person's life, normal cells divide faster to allow the person to grow. After the person becomes an adult, most cells divide only to replace worn-out or dying cells or to repair injuries.

Literatures

Arar, N., I. Thompson, et al. (2000). "Risk perceptions among patients and their relatives regarding prostate cancer and its heredity." Prostate Cancer Prostatic Dis **3**(3): 176-185.

We performed a qualitative study to examine how prostate cancer (PC) patients and their spouses and relatives take into account family history when considering susceptibility to PC. Semi-structured interviews were conducted with 20 participants. All interviews were tape-recorded, transcribed and content analyzed. Patients' and spouses' views concerning the seriousness of PC were different. Wives viewed PC as a serious disease because it has affected their marital relationships; patients found PC to be less serious because it can be treated. All participants viewed PC as a male disease that can be passed on from fathers to sons. Furthermore, participants were aware of PC clustering in their families. However, this awareness did not encourage (healthy) male relatives to seek early detection. Additionally, participants perceived age, high-fat diet, and less exercise as important risk factors, while socioeconomic status, ethnic origin and a family history of PC were viewed as less important. We recommend that health educators pay special attention to these findings when planning to teach patients, spouses and their relatives about PC, its heredity and risks. Prostate Cancer and Prostatic Diseases (2000) 3, 176-185

Brewer, D. A., E. L. Bokey, et al. (1993). "Heredity, molecular genetics and colorectal cancer: a review." Aust N Z J Surg **63**(2): 87-94.

It is estimated that the hereditary polyposis and non-polyposis colorectal cancer (CRC) syndromes, which have an autosomal dominant pattern of inheritance, represent less than 10% of the total CRC burden. Thus, more than 90% of all cases of CRC have previously been considered to arise 'sporadically', with no identifiable genetic link. However, recent clinical evidence now suggests that a significant proportion of CRC seen in the general population may involve an inherited genetic susceptibility. Therefore, constructing an accurate family tree on all patients with a family history of CRC is an essential part of identifying families with an increased risk for CRC who could then be offered screening. Also, molecular genetic study of colorectal adenomas and carcinomas has led to a proposed genetic model of colorectal tumorigenesis which involves interactions between oncogenes and tumour suppressor genes. This information has important potential implications for screening, determining prognosis and for providing multiple targets for altering the sequence of malignant transformation.

Civitelli, S., B. Civitelli, et al. (1995). "[Heredity and colorectal cancer]." Minerva Chir **50**(3): 167-75.

Colorectal cancer is the second leading cause of death from malignancies in Western Countries. In spite of advances in treatment, little change in survival has been accomplished in last decades and this mandates greater importance to prevention and early

detection. Although dietary factors have received primary attention familial clustering suggests that susceptibility to KCR is inherited. Hereditary colorectal cancer can arise on Familial Adenomatous Polyposis (HCC) or not on polyposis (HNPCC) and members of these families are at high risk of such neoplasias. Anyway, even in "sporadic" forms of KCR first-degree relatives have a 2 to 3-fold increased risk of the same cancer. The most desirable screening protocol would be a simple procedure involving only a blood test to identify gene defect by molecular biology techniques. Unfortunately, this is not practically possible, for lack of specific genetic alterations, out of FAP, and only the study of family history can enable targeted surveillance and cost-effective management strategies.

de Sanjose, S., P. Viladiu, et al. (1998). "[Breast cancer and heredity: results of a population case-control study in Girona]." Med Clin (Barc) **110**(10): 370-2.

BACKGROUND: To characterise the relationship between breast cancer and different aspects of the reproductive life, use of drugs and alcohol by family history of breast cancer. PATIENTS AND METHODS: From the cancer registry of Girona, Spain, 330 women were identified with histologically confirmed breast cancer during 1986-1989. For each case, a control woman was selected from a random sample of the population living in the matched area to the case by age (+/- 5 yr.). The information was collected by a personal interview and included: family history of breast cancer, reproductive history, presence of acne during the teenage years, use of oral contraceptives and drugs for sleep and anxiety disorders, and alcohol consumption. RESULTS: 18.5% of breast cancer cases and 8.9% of all controls had a family history of breast cancer. Family history on a first degree relative (mother or sister) was present in 10.6% of the cases and 2.8% of controls, which represented an odds ratio for breast cancer of 3.7 (95% CI, 1.8-7.8) higher than the general population. Women with a first degree family history of breast cancer were at higher risk for breast cancer if they had a history of acne during the teenage period (OR = 2.4; 95% CI, 1.1-5.2) and if they referred long menstrual periods in the early years of menarche (OR = 3.1; 95% CI, 1.3-7.0). Women with no family history had a higher breast cancer risk if they had a late menarche, long menstrual periods, late first full term pregnancy, and history of acne during puberty. Alcohol consumption and use of drugs for anxiety and sleep disorders were associated with a decreased risk of breast cancer. CONCLUSIONS: First degree family history of breast cancer seems to be the best risk indicator for developing breast cancer. Long menstrual periods and presence of acne during puberty may indicate hormonal imbalance that act independently of the family history in breast cancer development.

Gaudilliere, J. P. (1994). "[Cancer between infection and heredity: genes, viruses and mice at National Cancer Institute (1937-1977)]." Rev Hist Sci Paris **47**(1): 57-89.

After World War II, in the United States,viral explanation of cancer replaced a vision of the disease emphasizing genetic factors. From the mid 1950s onwards, experimental oncologists favored the notion that cancer was initiated by infectious agents passed from one generation to the next. In order to analyze this displacement, the present paper focuses on the part played by new experimental systems, i.e. mice showing tumors induced by viruses. Since animal models are agencies which "represent" human diseases, and mediate between different social worlds, their uses often result in opposing views. Mouse models thus provided tractable resources which favored the alternation between heredity and infection. The paper describes the emergence, in the late 1930s, at the Jackson Memorial Laboratory, of an agent enhancing the formation of mammary tumors in mice. This laboratory was then involved in the production of marketable inbred mice as well as in research concerned with genetic factors that may cause cancer. After World War II, loose theories and conflicting results helped turn the agent into a virus. At the National Cancer Institute, the virus was associated with a whole range of particles causing leukemia in mice. Owing to the Virus Cancer Program, the value of mouse tumor viruses increased in the late 1960s. This research effort then aimed at finding human tumor viruses, and at crafting cancer vaccines. It was modeled after the experience of the NCI chemotherapy program stemming from war research. In addition to the fact that biomedical research became a state enterprise, the study emphasizes three parameters. First, loose practices--both theoretical and experimental--helped manage the variability of animal models. Secondly, the standardization and mass production of animals and reagents encouraged the stabilization of research programs. Thirdly, private biotechnology companies working under NCI contracts implemented preclinical work, and mediated between virology laboratories and clinical settings.

Knudson, A. G., Jr. (1974). "Heredity and human cancer." Am J Pathol **77**(1): 77-84.

The dominantly inherited tumors of man demonstrate that mutation can be a step on the carcinogenetic pathway. Nonhereditary tumors may involve the same mutation in somatic cells rather than

germ cells. In neither case is this mutation alone sufficient to produce a tumor, and evidence is presented that a second mutational step is required in the initiation process. Individuals who bear these mutations germinally are extremely susceptible to specific tumors, they may develop more than one tumor, and the average age at onset is earlier than usual. Recessive genes for cancer susceptibility and environmental carcinogens may interact with each other and with these dominant "cancer genes" to increase the probability that cancer mutations will occur.

Kune, G. A., S. Kune, et al. (1989). "The role of heredity in the etiology of large bowel cancer: data from the Melbourne Colorectal Cancer Study." World J Surg **13**(1): 124-9; discussion 129-31.

Family history data of colorectal cancer, heart disease, and stroke were obtained on near relatives (parents, siblings, and children) in 702 colorectal cancer cases and 710 age-/sex-matched community controls as part of a large, comprehensive, population-based epidemiological and clinicopathological study of colorectal cancer conducted in Melbourne (the Melbourne Colorectal Cancer Study). There was a statistically significant higher family history rate of colorectal cancer in cases than in controls (relative risk = 2.13; 95% confidence interval = 1.53-2.96; p less than 0.001). This family history effect was more pronounced for colon cancer than for rectal cancer and there was an earlier age of detection of colorectal cancer in those with a family history of this cancer when compared with those without such a history. Dietary risk factors for colorectal cancer, which were previously described in the Melbourne study, were separate and independent from the family history effects. It is concluded that a family history of colorectal cancer is an important indication to screen individuals for this cancer, and also that while heredity has a definite role in the etiology of colorectal cancer, this hereditary effect is either likely to be small, or else likely to be important in only a proportion (perhaps 20%) of cases.

Lagergren, J., W. Ye, et al. (2000). "Heredity and risk of cancer of the esophagus and gastric cardia." Cancer Epidemiol Biomarkers Prev **9**(7): 757-60.

The importance of genetic factors in the etiology of esophageal cancer is uncertain. We addressed the question of heredity in a population-based, nationwide case-control study conducted in Sweden during 1995 through 1997. The study involved 189 patients with esophageal adenocarcinoma, 262 with cardia adenocarcinoma, 167 with esophageal squamous cell carcinoma, and, for comparison, 820 control subjects. Familial occurrence of cancer was explored at face-to-face interviews. Logistic regression, with multivariate adjustment for potential confounders, was used to calculate odds ratios (ORs), which estimated relative risk. Occurrence of esophageal cancer among first-degree relatives did not increase the risk of adenocarcinoma or squamous cell carcinoma of the esophagus. Neither were there any significant associations with familial occurrence of gastric cancer or other gastrointestinal tumors. The risk of cardia adenocarcinoma was moderately increased among persons with first-degree relatives with gastric cancer (OR, 1.6; 95% confidence interval, 1.0-2.6). Familial occurrence of any cancer was not associated with increased risks of any of the three studied tumors. In conclusion, heredity does not seem to contribute importantly to the occurrence of esophageal cancer of any histological type. A weak association between familial gastric cancer and the risk of cardia cancer may represent a genetic link.

Levine, E. G., R. A. King, et al. (1989). "The role of heredity in cancer." J Clin Oncol **7**(4): 527-40.

Heredity is generally felt to play a minor role in the development of cancer. This review critically examines this assumption. Topics discussed include evidence for heritable predisposition in animals and humans; the potential importance of genetic-environmental interactions; approaches that are being used to successfully locate genes responsible for heritable predisposition; comparability of genetic findings among heritable and corresponding sporadic malignancies; and future research directions. Breast, colon, and lung cancer are used to exemplify clinical and research activity in familial cancer; clinical phenotypes, segregation and linkage analyses, models for environmental interactions with inherited traits, and molecular mechanisms of tumor development are discussed. We conclude that the contribution of heredity to the cancer burden is greater than generally accepted, and that study of heritable predisposition will continue to reveal carcinogenic mechanisms important to the development of all cancers.

Lynch, H. T., R. E. Harris, et al. (1977). "Role of heredity in multiple primary cancer." Cancer **40**(4 Suppl): 1849-54.

The occurrence of multiple primary malignant neoplasms characterizes virtually all varieties of hereditary cancer. This report focuses on this phenomenon in 11 families with the Cancer Family Syndrome (heritable adenocarcinomas of the colon and endometrium) and a single extended kindred with site-specific colon cancer. Of the 316 relatives with cancer in the 12 families, 68 (21.5%) had two or more primary malignancies and 59

(86.8%) of these multiple primaries involved the colon and/or endometrium. A pooled analysis of this resource revealed a consistent 3% risk for a second primary cancer in each year of survival following first onset. If a second primary occurs, the risk for a third is extremely high (6.9% per year), but shows a nonlinear trend with increasing survival following second onset. The high risk for development of extraprimary malignancies in patients from these kindreds indicates that careful consideration should be given to total removal of their principal target organs following the initial manifestation of cancer.

Marcus, J. N., P. Watson, et al. (1994). "Pathology and heredity of breast cancer in younger women." J Natl Cancer Inst Monogr(16): 23-34.

The pathology of early-age onset breast cancer is considered here from three perspectives: 1) benign proliferative disease, 2) the cancers themselves, and 3) familial and hereditary breast cancer. Hereditary breast cancer, a subset of familial breast cancer featuring a strong autosomal dominant pedigree pattern and multiple primary cancers, has a strong predilection for younger women, accounting for about one half of breast cancers under age 30. With respect to benign proliferative disease, the increased relative risk of breast cancer associated with proliferative disease with atypia, about fourfold to fivefold for all ages, is doubled by the presence of a family history of breast cancer and amplified by young age. With respect to the carcinomas, the relative incidences of medullary carcinoma and ductal carcinoma in situ are increased in young women, while lobular and tubular carcinomas are decreased. Invasive breast cancer is higher grade and more proliferative in younger women, as measured by thymidine-labeling index, DNA flow cytometric S-phase fraction, and proliferation-associated proteins. The increased fraction of ductal carcinoma in situ and higher grade invasive cancers may help to account, respectively, for increased recurrence rates with conservative therapy, and more aggressive natural history in younger women. Familial breast cancers show trends for increased medullary type, but the effect is not independent of age. Weak associations of family history with tubular carcinoma have been reported, but data for associations with lobular carcinoma in situ and invasive lobular carcinoma are conflicting. Hereditary breast cancer as a class has higher tumor proliferation rates, an effect independent of age. Knowledge of the pathology and biomarker characteristics of BRCA1 gene-linked hereditary breast cancers, which account for a substantial fraction of breast cancers in younger women, should shed light on the nature of the responsible gene(s) and guide approaches to therapy and prophylaxis.

Ondrusek, N., E. Warner, et al. (1999). "Development of a knowledge scale about breast cancer and heredity (BCHK)." Breast Cancer Res Treat **53**(1): 69-75.

An 11-item questionnaire, the Breast Cancer and Heredity Knowledge Scale (BCHK), was developed to test general knowledge about breast cancer and hereditary breast cancer (HBC) among women at low to moderate risk of HBC. The BCHK measures knowledge about breast cancer incidence and prognosis, risk factors, screening, disease presentation and treatment, and HBC. Scale items were generated from focus group interviews, previously published breast cancer knowledge scales, and consultation with a multidisciplinary research team, including health professionals and women with breast cancer or a family history of breast cancer. A 27-item draft scale was tested on 36 breast clinic patients and 17 women from the general public. Results were used to develop the final 11-item scale. Development of the scale and its potential uses are discussed.

Orrom, W. J., W. S. Brzezinski, et al. (1990). "Heredity and colorectal cancer. A prospective, community-based, endoscopic study." Dis Colon Rectum **33**(6): 490-3.

The frequency of colorectal neoplasia was assessed through colonoscopy in 114 patients with a family history of colorectal cancer. In over 90 percent of patients, a first-degree relative was affected. Twenty-one percent of patients who were studied endoscopically were positive for neoplastic disease, including two invasive cancers. Twenty-eight percent of patients had adenomas beyond the splenic flexure. Multiple primary relatives further increased risk with 36 percent positive for neoplasia. Neoplasia was common in young patients, with 25 percent under the age of 40 years positive for adenomas. These findings are identical to recent pedigree studies and further support a genetic basis for common colorectal cancers. First-degree relatives of patients with colorectal cancer should be considered at high-risk for colorectal neoplasia. Screening and surveillance with colonoscopy is recommended.

Page, W. F., M. M. Braun, et al. (1997). "Heredity and prostate cancer: a study of World War II veteran twins." Prostate **33**(4): 240-5.

BACKGROUND: Increased risk of prostate cancer among men with a family history of the disease has been observed in several epidemiological studies, and family studies have identified hereditary prostate cancer characterized by early onset and autosomal dominant inheritance. METHODS: In this study, we examine prostate cancer heritability among twins in

the NAS-NRC Twin Registry, with cases ascertained from a number of sources: recent telephone interviews, Medicare and Department of Veterans Affairs hospitalizations, previous mail questionnaires, and death certificates. A total of 1,009 prostate cancer cases were identified among the cohort of 31,848 veteran twins born in the years 1917-1927. RESULTS: Probandwise concordance for prostate cancer was substantially higher among monozygous twin pairs, 27.1%, than among dizygous twin pairs, 7.1% (P < 0.001). CONCLUSIONS: These data suggest that genetic influences account for approximately 57%, and environmental influences for 43%, of the variability in twin liability for prostate cancer.

Powell, I. J., J. Carpten, et al. (2001). "African-American heredity prostate cancer study: a model for genetic research." J Natl Med Assoc **93**(4): 120-3.

A genome-wide scan of high-risk prostate cancer families in North America has demonstrated linkage of a particular marker to Chromosome 1q (HPC1). An even greater proportion of African-American families have shown linkage to HPC1. Therefore, investigators at the National Human Genome Research Institute (NHGRI) in collaboration with Howard University and a predominantly African-American group of urologists established the African-American Hereditary Prostate Cancer (AAHPC) Study Network to confirm the suggested linkage of HPC in African Americans with a gene on Chromosome 1. Blood samples from recruited families were sent to Howard University for extraction of DNA. The DNA was sent to NHGRI at NIH where the genotyping and genetic sequence analysis was conducted. Genotype data are merged with pedigree information so that statistical analysis can be performed to establish potential linkage. From March 1, 1998, to June 1, 1999, a total of 40 African-American families have been recruited who met the study criteria. Preliminary results suggest that racial/ethnicity grouping may affect the incidence and extent of linkage of prostate cancer to specific loci. The importance of these findings lays in the future treatment of genetic-based diseases.

Powell, I. J., J. Carpten, et al. (2001). "African-American heredity prostate cancer study: a model for genetic research." J Natl Med Assoc **93**(12 Suppl): 25S-28S.

A genome-wide scan of high-risk prostate cancer families in North America has demonstrated linkage of a particular marker to Chromosome Iq (HPC11. An even greater proportion of African-American families have shown linkage to HPC 1. Therefore, investigators at the National Human Genome Research Institute [NHGRI] in collaboration with Howard University and a predominantly African-American group of urologists established the African-American Hereditary Prostate Cancer (AAHPC) Study Network to confirm the suggested linkage of HPC in African Americans with a gene on Chromosome 1. Blood samples from recruited families were sent to Howard University for extraction of DNA. The DNA was sent to NHGRI at NIH where the genotyping and genetic sequence analysis was conducted. Genotype data are merged with pedigree information so that statistical analysis can be performed to establish potential linkage. From March 1, 1998, to June 1, 1999, a total of 40 African-American families have been recruited who met the study criteria. Preliminary results suggest that racial/ethnicity grouping may affect the incidence and extent of linkage of prostate cancer to specific loci. The importance of these findings lays in the future treatment of genetic-based diseases.

Rapola, J. (1980). "Heredity of cancer." J Toxicol Environ Health **6**(5-6): 983-7.

Hereditary factors are linked with cancer in four ways: by the association of specific chromosomal abnormalities with certain types of cancer, the increase of cancer incidence in some hereditary disorders, the increase of susceptibility to cancer in certain genotypes, and by direct inheritance of some rare malignant neoplasms.

Snelders, S. (2008). "The plot against cancer: heredity and cancer in German and Dutch medicine, 1933-1945." Gesnerus **65**(1-2): 42-55.

In the Third Reich hereditarian approaches and their eugenic implications seemed to offer possibilities for fundamental progress in the fight against cancer. This did not lead to an exclusive emphasis on genetics in theory or practice. The concept of a hereditary predisposition for cancer, the Krebs-disposition or Krebsbereitschaft, led to flexible multifactor approaches, including proposals for both eugenic and social-hygienic measures. These approaches were not typical of German medicine alone. In the Netherlands hereditarian approaches did not play a central role in the 1930s. They lacked institutional support in a country where health policies were characterised by indirect strategies working through intermediaries such as general practitioners and home nursing organisations. However, potentially the elements for similar anti-cancer policies as in Germany were present. The German occupation offered opportunities to develop these elements (concepts, institutions, personnel). This development was blocked because of the political radicalisation during the war and the German defeat.

Trano, G., H. H. Wasmuth, et al. (2009). "Awareness of heredity in colorectal cancer patients is insufficient among clinicians: a Norwegian population-based study." Colorectal Dis **11**(5): 456-61.

OBJECTIVE: The assessment of family history and medical data is crucial in identifying families with Lynch syndrome (LS). Among consecutive colorectal cancer (CRC) patients, we aimed at identifying all patients with a hereditary predisposition, and to study a possible discrepancy with assessments made by the responsible clinicians. METHOD: All consecutively diagnosed patients with CRC from two Norwegian hospitals were included, and information on family history was collected in a detailed interview. We assessed information in medical records, and tumours were examined for LS-associated histopathological features. RESULTS: Among 562 patients, there was no documentation of family history in 388 (69.0%) medical records, and in 174 (31.0%) patients, there was no clinical assessment of the information that was collected on family history. Based on detailed interviews and extended pathological examination, we found that 137 (24.4%) of the 562 patients could be classified as possible LS according to the Revised Bethesda Guidelines (RBG); and that 46 (33.6%) of these patients could be identified by family history alone. CONCLUSION: Family history and relevant information in patient records can identify patients with possible LS. However, clinicians often fail to include information on hereditary factors and to assess relevant data in medical records. Familial CRC is therefore not acknowledged, and genetic counselling is not offered.

Velu, T., N. Ravelingien, et al. (1994). "[Genes, heredity and colorectal cancer]." Rev Prat **44**(20): 2694-9.

All cancers result from the accumulation of mutations of proto-oncogenes and tumor suppressor genes. Sporadic and familial colorectal cancers result from the accumulation of the following genes, in a relatively stereotyped chronological order: the tumor suppressor gene apc whose mutations are responsible for the familial adenomatous polyposis; the proto-oncogene K-ras which is mutated in 50% of large adenomas (> 1 cm) and adenocarcinomas; the tumor suppressor gene dcc; and the tumor suppressor gene p53 whose inactivation in a factor of bad prognosis. While some of them are induced by mutagens, others result from an instability of the genome. Two types of instability are observed in both sporadic and familial colorectal cancer. The first type, which is found in 25-50% of cases, appears as cytogenetic abnormalities with aneuploidy and allelic losses. The second type of instability is induced by mutations of the hMSH2 or hMLH1 genes which code for proteins involved in the mechanism of DNA repair.

Virtanen, A., M. Gomari, et al. (1999). "Estimation of prostate cancer probability by logistic regression: free and total prostate-specific antigen, digital rectal examination, and heredity are significant variables." Clin Chem **45**(7): 987-94.

BACKGROUND: Despite low specificity, serum prostate-specific antigen (PSA) is widely used in screening for prostate cancer. Specificity can be improved by measuring free and total PSA and by combining these results with clinical findings. Methods such as neural networks and logistic regression are alternatives to multistep algorithms for clinical use of the combined findings. METHODS: We compared multilayer perceptron (MLP) and logistic regression (LR) analysis for predicting prostate cancer in a screening population of 974 men, ages 55-66 years. The study sample comprised men with PSA values >3 microg/L. Explanatory variables considered were age, free and total PSA and their ratio, digital rectal examination (DRE), transrectal ultrasonography, and a family history of prostate cancer. RESULTS: When at least 90% sensitivity in the training sets was required, the mean sensitivity and specificity obtained were 87% and 41% with LR and 85% and 26% with MLP, respectively. The cancer specificity of an LR model comprising the proportion of free to total PSA, DRE, and heredity as explanatory variables was significantly better than that of total PSA and the proportion of free to total PSA ($P < 0.01$, McNemar test). The proportion of free to total PSA, DRE, and heredity were used to prepare cancer probability curves. CONCLUSION: The probability calculated by logistic regression provides better diagnostic accuracy for prostate cancer than the presently used multistep algorithms for estimation of the need to perform biopsy.

Voitenko, V. P. (1985). "Heredity, age and cancer." IARC Sci Publ(58): 35-42.

A factor analysis of mortality from gastric cancer in the populations of 41 countries has been made. It is concluded that the interrelation between age and cancer has both a biological and a chronological component. On the one hand, tumour development is linked to the molecular-genetic and systemic-physiological mechanisms of ageing. On the other, increasing mortality from cancer with age reflects the number of years for which the organism was exposed to the carcinogenic action. Each of these mechanisms is illustrated by the factor model of mortality from gastric cancer. Hereditary effects on both mechanisms that relate age and cancer are discussed.

Watanabe, S. and H. Ochi (1987). "[Heredity in clinical cancer]." Gan No Rinsho **33**(5 Suppl): 610-4.

Familial clustering of cancer causes a problem whether significant differences exist from one person to the next in cancer susceptibility. Such clustering could occur simply by the chance occurrence of common environmental exposure, but in rare families, clustering is caused by discrete genetic factors, some being recessive and some being dominant inheritance with mendelian segregation. In this review article, heritable cancers are listed and discussed. Recent laboratory developments, such as the study on chromosome fragile sites and restriction enzyme-fragment length-polymorphism, to find the predisposition for cancer was introduced. Other analysis for individual difference by the genetic epidemiology was also summarized.

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