sex differences in cancer

By GPT4 ChatGPT

DNA damage and repair

Population-level considerations

Only corresponding reference papers

The Life Span Study (LSS) of atomic bomb survivors from Hiroshima and Nagasaki provided critical insights into sex differences in solid cancer risk due to radiation exposure. Across the follow-up period from 1958 to 2009, 22,538 first primary solid cancer cases were identified, with 992 directly associated with radiation exposure. Key findings included: - Females exhibited a linear dose-response relationship with an excess relative risk (ERR) of 0.64 per Gy. - Males showed significant upward curvature in the dose-response, best described by a linear-quadratic model with an ERR of 0.20 at 1 Gy. - The rate of decrease in ERR with increasing age was more rapid in males compared to females. - A statistically significant dose-response relationship was observed even at low dose ranges (0–100 mGy). This extensive analysis revealed enduring solid cancer risks over six decades post-exposure, with noteworthy sex-dependent variations in the dose-response relationship and a reduction in risk with increasing age. Despite adjustments for smoking and other improvements in the study methods, discrepancies with prior reports suggest unresolved uncertainties affecting the guidance of radiation protection policies. Ongoing analysis, particularly for specific organs and continued cohort follow-up, is essential for a comprehensive understanding of radiation-related cancer risk and its significance to public health 【47† source】.

Sex hormone regulation of DNA repair

We demonstrate that the androgen receptor (AR) regulates a transcriptional program of DNA repair genes that promotes prostate cancer radioresistance, providing a potential mechanism by which androgen deprivation therapy synergizes with ionizing radiation. Using a model of castration-resistant prostate cancer, we show that second-generation antiandrogen therapy results in downregulation of DNA repair genes. Next, we demonstrate that primary prostate cancers display a significant spectrum of AR transcriptional output, which correlates with expression of a set of DNA repair genes. Using RNA-seq and ChIP-seq, we define which of these DNA repair genes are both induced by androgen and represent direct AR targets. We establish that prostate cancer cells treated with ionizing radiation plus androgen demonstrate enhanced DNA repair and decreased DNA damage and furthermore that antiandrogen treatment causes increased DNA damage and decreased clonogenic survival. Like the approach used to select patients with breast cancer for antiestrogen therapy based on estrogen receptor/progesterone receptor status, it may be possible to tailor the administration of androgen deprivation therapy (ADT) alongside radiotherapy based on the androgen receptor (AR) output of prostate tumors. Mutations in double-strand break repair proteins often lead to diseases characterized by cancer predisposition, sensitivity to ionizing radiation, and chromosomal instability. Among the critical nuclear hormone receptors expressed in breast cancer cells are the estrogen and retinoic acid receptors. Estrogen receptors (ER) and retinoic acid receptors (RAR) belong to a family of ligand-dependent transcription factors that include steroid, thyroid, and vitamin D receptors. In experiments with breast cancer cell lines, it has been observed that estrogen receptor-positive lines (MCF7 and T47D) and estrogen receptor-negative lines (MDA-MB-231 and MDA-MB-468) exhibit differential responses to treatment with 17β-estradiol (E2) or all-trans retinoic acid (RA), alone or in combination, prior to DNA damage induction with etoposide. In this mini-review, we examine evidence for sex differences in cellular responses to DNA damage, their underlying mechanisms, and how they might relate to sex differences in cancer incidence and response to DNA-damaging treatments. Sex differences in cancer incidence are widespread and include hematological malignancies, as well as cancers of the bladder, colon, skin, liver, and brain, with male-to-female incidence ratios ranging from 1.26:1 to 4.86:1. Considering the cellular response to DNA damage is crucial, as it is a potential common mechanism underlying sex differences in cancer incidence and response to treatment. There is also the complementary aspect to consider—evidence for sex differences in cancer in individuals with cancer predisposition syndromes from germline DNA repair defects. These sex differences in cancer mechanisms point to the biological complexity and the necessity for tailored approaches in understanding and treating different cancers. In a model of castration-resistant prostate cancer, research shows that second-generation antiandrogen therapy leads to the downregulation of DNA repair genes. Through RNA-seq and ChIP-seq, it has been determined which DNA repair genes are induced by androgen and are direct targets of the androgen receptor (AR). It has been established that prostate cancer cells treated with ionizing radiation, when combined with androgen, demonstrate enhanced DNA repair capabilities and decreased DNA damage. Conversely, antiandrogen treatment has been observed to cause increased DNA damage and reduced survival of clonogenic cells. Moreover, there is evidence that antiandrogen treatment results in decreased activity of classical nonhomologous end-joining, an important DNA repair pathway. This suggests that the AR may regulate a network of DNA repair genes, providing a potential mechanism by which androgen deprivation therapy could synergize with radiotherapy to treat prostate cancer.

Tumor suppressor effects of the X chromosome

X chromosome mutation buffering effects

The X chromosome has emerged as a key player in the genomic defense against cancer, particularly through its interaction with the tumor suppressor protein p53. Studies have shown that the X chromosome encodes numerous tumor-suppressor genes, which are integral to the functional integrity of p53 and the regulation of its network. The significance of the X chromosome in p53's tumor suppressive functions is highlighted by the observation that X chromosome-encoded genes significantly interact with the p53 signaling pathway. Furthermore, evidence suggests that the functional engagement of p53 in cancer defense is sex-specific, influenced by the genetic sex defined by either the XY male or XX female chromosome configuration. Recent findings have demonstrated that p53 can directly influence X chromosome silencing by upregulating XIST levels, which further emphasizes the chromosome's role in mutation buffering. Furthermore, the X chromosome is enriched with microRNAs that target the p53 signaling pathway, indicating a complex regulatory network. Particularly noteworthy is the identification of KDM6A, an X chromosome-encoded tumor suppressor gene, which illustrates the chromosome's protective effect against bladder cancer (BCa) in females. KDM6A acts as both a demethylase-dependent and demethylase-independent tumor suppressor gene, and its deletion leads to a reduction in sex differences in BCa susceptibility. This suggests that the tumor suppressive activities facilitated by the X chromosome, such as those of KDM6A, could be essential in understanding the sex-biasing effects of cancers. The loss of KDM6A holds prognostic value, as females without this gene develop aggressive squamous-like cancer and experience poorer outcomes. Therefore, the tumor suppressor effects of the X chromosome appear to be multifaceted, involving genetic and epigenetic mechanisms to buffer against mutations that could lead to oncogenesis. The complex interplay between the X chromosome and p53 provides a potential explanation for the observed sex disparities in cancer incidences and outcomes, and further research into this relationship could yield critical insights into cancer biology and treatment.

Sex chromosome loss in cancer

In the context of "Sex chromosome loss in cancer," several studies have revealed significant associations between the loss of sex chromosomes and various types of cancers. Loss of the Y chromosome has been reported in different renal cell tumors such as chromophobe carcinomas (60%), oncocytomas (51%), and clear cell carcinomas (39%) 【23†source】. Moreover, the loss of chromosome Y was linked with favorable disease outcomes in certain cancer types, reflecting that its presence or absence may influence tumor biology; however, this could vary across different cancers. For instance, in chronic myelomonocytic leukemia, Y loss was associated with better prognosis, while it signified worse outcomes in head and neck cancer and multiple myeloma. Similarly, the loss of the X chromosome (monosomy X) has been observed in myeloid-derived cells of patients with myelodysplastic syndromes (MDS) that evolved into acute myeloid leukemia (AML). It is suggested that the loss of the X chromosome might be involved in the expansion of a dysplastic clone, contributing to the illness's progression and potentially indicating the loss of a tumor suppressor gene. Although there currently is no identified tumor suppressor gene on the X chromosome, monosomy X could be associated with MDS and the neoplastic transformation of cells, particularly when it arises as the sole non-constitutional cytogenetic abnormality in bone marrow 【29†source】. Taken together, these studies highlight the tumor suppressor effects of the X chromosome and suggest that the loss of sex chromosomes, whether X or Y, may have distinct impacts on the pathogenesis and prognosis of various cancers. This underscores the potential significance of sex chromosome composition in tumor biology and the disease course, implicating the need for further research to understand its role in cancer.

Anticancer immunity

The section on "Anticancer immunity" in the paper titled 'The spectrum of sex differences in cancer' delves into sex differences in innate and adaptive immune responses, highlighting potential implications for cancer prognosis and therapy outcomes. **Innate Immunity:** The paper discusses how innate immunity, encompassing the body's first line of defense against pathogens, displays sex differences encoded at the genetic level. For example, innate recognition of nucleic acids by pattern recognition receptors (PRRs) varies between sexes due to sex chromosome-linked gene expression 【47†source】. Toll-like receptor 7 (TLR7), found on the X chromosome, may escape X inactivation, leading to higher expression levels in females along with higher interferon-α (IFNα) production. Sex differences are also observed in the activity of various innate immune cells. For instance, phagocytic activity is typically higher in neutrophils and macrophages from females, and antigen-presenting cells (APCs) from females are more efficient at presenting peptides than those from males. Certain cytokines and chemokines display sex-biased production upon immune cell activation, influenced by sex hormones like androgens and estrogens 【47†source】 【51†source】 . **Adaptive Immunity:** Adaptive immunity, involving specific responses to foreign antigens, is shaped by sex through the influence on the development, function, and distribution of lymphocyte subsets, including B cells and T cells. Females generally have higher B cell counts and exhibit stronger antibody responses than males. CD4+ T cell counts and the CD4/CD8 ratio are higher in females, whereas men display higher frequencies of CD8+ T cells. After stimulations such as mitogens, female immune cells often show greater activation and proliferation. Moreover, the differentiation and cytokine production of CD4+ T cell subsets, such as T helper 1 (TH1) and T helper 2 (TH2) cells, exhibit sexual dimorphism, potentially influencing immune responses during infections and vaccination 【51†source】 . **Sex Differences and Cancer:** The paper underscores that these sex-based immunological differences contribute to variations in disease incidence, susceptibility to infectious diseases, response to vaccines, and also the incidence of autoimmune diseases and malignancies between males and females. In broad terms, adult females typically mount stronger immune responses than males, leading to faster clearance of pathogens but also a heightened susceptibility to autoimmune diseases. The sex differences in immunological responses are argued to be due to hormonal, genetic, and environmental influences, which can change across an individual's lifespan 【47†source】 . To conclude, the paper suggests that understanding sex differences in immune responses, particularly innate and adaptive immunity, is vital for recognizing potential disparities in cancer outcomes and responses to treatment in males and females, thereby pointing to the importance of considering sex as a biological variable in immunological studies and cancer research.

Role of sex chromosomes in immunity

Sex chromosomes play a critical role in the immune system and in modulating responses to cancer. Females have two X chromosomes, one from each parent, while males have one X and one Y chromosome. Dosage compensation between the sexes is usually achieved by the inactivation of one X chromosome in females, which gets packaged into transcriptionally inactive heterochromatin, known as a Barr body 【19†source】 . This inactive X chromosome can sometimes escape silencing, affecting gene expression and immune function. Skewed X chromosome inactivation (XCI) has also been observed, where defects lead to the progressive silencing of the defective chromosome, potentially offering a biological advantage but also leading to immunological consequences 【23†source】 . The distribution of active X chromosomes in females can introduce functional diversity in immune responses, as mosaicism can provide an advantage when there are deleterious mutations on X chromosome genes. Furthermore, some X-linked genes have homologs on the Y chromosome and can escape XCI, playing a role in differences observed in immune responses between men and women, and between individual women 【19†source】 【23†source】 . Furthermore, X-linked microRNAs, which are more densely packed on the X chromosome when compared to autosomes, have been implicated in immunity, suggesting a potential contribution to gender-specific immune responses and possibly influencing the genetics of autoimmune diseases (AID) in adults. Various diseases such as Wiskott-Aldrich syndrome, X-linked neutropenia, and Chronic Granulomatous Disease exhibit major autoimmune features and are influenced by the expression patterns of genes on the X chromosome 【27†source】 . In conclusion, sex chromosomes and their associated epigenetic modulations, such as XCI escape, skewed XCI, and x-linked microRNAs, are key factors in understanding the divergent immune responses observed between males and females.

This variance may contribute to differences in the development and treatment of AIDs, and potentially, to anticancer immunity.

Role of sex hormones in immunity

The interplay between sex hormones and the immune system is increasingly recognized as a vital aspect of cancer biology. Within the framework of anticancer immunity, it is critical to acknowledge the nuanced impact that these hormones can have on the body's natural defenses against malignancies. Estrogens, for instance, have been found to exert substantial influence on cytokine production. Cytokines are signaling molecules that mediate and regulate immunity, inflammation, and hematopoiesis. The ability of estrogens to modulate both the production and function of various cytokines underscores their potential role in orchestrating specific immune responses. Understanding this mechanism is essential as it sheds light on why certain cancers may exhibit sex differences in both susceptibility and treatment efficacy. Additionally, estrogen receptor (ER) signaling plays a notable role in immune cell regulation, particularly affecting plasmacytoid dendritic cells (pDCs). These cells are instrumental in the body's antiviral responses and are also implicated in tumor surveillance. The direct pathway through which ER signaling influences pDC differentiation and function may offer valuable insight into the manner in which hormonal fluctuations impact anticancer immunity. In contrast to estrogens, testosterone has been shown to suppress the production of immunoglobulins, which are crucial components of the adaptive immune response. This suppression of antibody synthesis by testosterone may reveal why male immune responses are different from those of females, particularly in the context of cancer, where such differences could be consequential. The influence of ovarian hormones, including both estrogens and progesterone, extends to the modulation of B cell immunity. B cells are a type of white blood cell responsible for producing antibodies. The role of these hormones is especially pertinent when considering mucosal immunity within the female genital tract, as well as the hormonal variations that occur throughout the menstrual cycle and their implications for immune surveillance and responsiveness. Testosterone also plays a pivotal role in immune system regulation through its effect on macrophage behavior, specifically by modifying the expression of Toll-like receptors responsible for detecting pathogens. This alteration in innate immune responses provides another facet of how sex hormones might influence immune system efficacy against infections and by extension, affect cancer progression and the response to therapy. Lastly, the impact of estrogens on the differentiation of dendritic cells from bone marrow precursors brings to light another critical pathway in anticancer immunity. Given the central role of dendritic cells in presenting antigens and initiating the adaptive immune response, their estrogen-driven differentiation could be instrumental in determining how effectively the immune system identifies and targets cancer cells. Collectively, these insights contribute to a more comprehensive understanding of the spectrum of sex differences in cancer, underscoring the intricate relationship between sex hormones and immune system dynamics.

Impact of sex bias on tumor immunity and immunotherapy

Sex differences exert a substantial influence on immune responses within the tumor microenvironment and the subsequent efficacy of cancer immunotherapies. The interplay between immune selection, estrogens in myeloid cells, and the mechanisms of immunotherapy reveal the need for personalized cancer treatments that take into account sex-based biological factors. Particularly, the benefits of immunotherapy in combination with chemotherapy for women in certain types of cancer like advanced lung cancer underscore the necessity of sex-specific therapeutic strategies. This integrated approach is essential for both developing more effective treatments and for rigorous evaluation of efficacy in clinical settings. To complete the subsection for your paper, I will review the remaining PDF documents for additional insights to incorporate into the summary. assistant: The review article by Conforti et al., "Cancer immunotherapy efficacy and patients' sex: a systematic review and meta-analysis," discusses the influence of a patient's sex on the efficacy of immune checkpoint inhibitors used as cancer treatments. The systematic review and meta-analysis aimed to assess the heterogeneity in efficacy between men and women by analyzing data from randomized controlled trials of inhibitors of PD-1, CTLA-4, or both. The study found that immune checkpoint inhibitors improve overall survival for patients with advanced cancers such as melanoma and non-small-cell lung cancer, but the magnitude of benefit is sex-dependent. Specifically, the pooled overall survival hazard ratio (HR) for male patients treated with immune checkpoint inhibitors was 0.72, while for female patients, it was 0.86, pointing to a greater benefit for men. This significant difference in efficacy between men and women (p=0.0019) emphasizes the need for future research to include more women in trials, focus on improving immunotherapy effectiveness in women, and perhaps explore different immunotherapeutic approaches for men and women. Conforming to these findings, the summary for your "Anticancer Immunity" section could incorporate the evidence of sex-related immune system differences that impact the natural course of diseases, including cancer, and the need to consider these differences in the development and administration of immunotherapies. There's acknowledgment that sex (the biological differences between men and women) and gender (behavioral differences) potentially affect immune responses to both pathogens and tumors, leading to differing outcomes between the sexes in terms of infection severity, prevalence, response to vaccines, and autoimmune diseases prevalence. As women generally mount stronger immune responses, this may explain the lower severity and prevalence of many infections in women and their greater response to vaccines. However, women also constitute the majority of patients with systemic autoimmune diseases globally. At the cellular level, there are substantial differences in the innate and adaptive immune systems based on sex, reflecting complex interactions between genes, hormones, environmental factors, and the microbiome composition. Bringing together these findings, the subsection on "Impact of sex bias on tumor immunity and immunotherapy" should stress the sex-based differences in immune responses, the modulation of these responses by sex hormones, and their effects on cancer immunotherapy efficacy. Moreover, it should advocate for more inclusive and sex-balanced clinical trials to properly assess the efficacy and safety of novel treatments for male and female cancer patients alike. We have now reviewed the information from the PDFs provided. If there is any additional information in the remaining documents that you would like to include in the subsection, please let me know, so we can address that before finalizing the summary. assistant: The article "Sex-Based Dimorphism of Anticancer Immune Response and Molecular Mechanisms of Immune Evasion" by Conforti et al. delves into the sex-based differences in the molecular mechanisms underlying anticancer immune responses and the ways in which non-small cell lung cancers (NSCLCs) evade immune attack in men and women. Specifically, it explores the differences in the tumor microenvironment (TME), the status of T-cell functionality, the expression of inhibitory immune checkpoint molecules, and the abundance of immune-suppressive cells such as cancer-associated fibroblasts, myeloid-derived suppressor cells (MDSCs), and regulatory T cells between sexes. The study reveals that the TME of women is notably enriched with a variety of immune cell types, including specific T-cell subpopulations, which however, display greater dysfunction and higher expression of inhibitory checkpoint molecules compared to men. This suggests a different kind of immune evasion strategy where female tumors may become more adept at inactivating an otherwise more potent immune response. In contrast, tumors in men often present a T-cell–excluded phenotype and potentially impaired neoantigen presentation to the immune system. Importantly, the research shows that these sex-based differences are not related to other potentially confounding variables such as age, stage of disease, tumor histology, or smoking status. The data further correlate with significant sex-based differences in the association between tumor mutational burden (TMB) and the outcome of advanced NSCLC patients treated with anti–PD-1/PD-L1 drugs. Drawing from these insights, the subsection "Impact of sex bias on tumor immunity and immunotherapy" for your paper should highlight the necessity of considering the unique differences in the mechanisms of immune response and immune evasion present in males and females when managing NSCLC. These findings advocate for customized immunotherapeutic strategies in NSCLC to improve prognosis for both women and men. Together with the information from the previous documents, this study complements the overall narrative about the critical need to integrate sex-based immunological differences into cancer research and therapy, particularly concerning the development, evaluation, and administration of immunotherapies. It emphasizes the intrinsic heterogeneity of immune response based on sex and underscores that future clinical trials and therapeutic design must account for these variations to optimize treatment benefits for cancer patients of both sexes.

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