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Book Review

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Book review of trends in breast cancer prevention

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Book review

There is not a single preventive agent that can stop the incidence of breast cancer that is the malignant disease most frequently diagnosed in women of all races and nationalities. Furthermore, its incidence around the globe is increasing in countries that are industrialized, as well as in those that have recently become industrialized. The worldwide incidence of breast cancer has increased 30-40% since the 1970s, reaching an excess of 1,390,000 new cases and a mortality of more than 460,000 cases by 2015. Therefore what is needed is the development of rational strategies for the prevention of his fatal disease. The book *"Trends in Breast cancer prevention"* brings the most recognized experts in preventive strategies in breast cancer providing the accepted as well as the novel ideas that have been introduced for the prevention of this disease.

This book is targeted to all thus that are wishing to be educated on how to prevent this debilitating and devastating disease that affect not only their body but their family and social environment. The book is scholarly written providing novel information to those in public health, academia and researchers in cancer.

In the chapter 1 written by Janssens, it is dicusssed how the disease originates early in life, the impact of epi-genomic imprinting, and the recognition that breast cancer is a family of related but distinct diseases. All these knowledge are connected and are the basis for developing new paradigms in the prevention of breast cancer. The importance of the preclinical interval between generation of susceptibility and appearance of the diseases offers opportunities for primary prevention and presumably has a period when genetic control is modifiable. Early detection becomes now the priority and can be achieved through recognition of risk markers that reliably predict disease. High priority must be given to lifestyle research on affordable reversion of epigenetic alterations.

Lamartiniere and his coworkers provide in the chapter 2 scholar evidence on the use of genistein, a phytochemical component of soy, for the prevention of breast cancer. Prepubertal exposure of rats to genistein via the mothers' milk alters mammary protein expressions whose actions are consistent with regulation of cell turn over and tissue remodeling. In mature rats exposed prepubertally to genistein, but now in the absence of genistein, protein expressions are altered to reflect actions consistent with more differentiated terminal ductal structures, increased apoptosis, and reduced potential for carcinogenesis in the mammary gland. The basic concept is that genistein induces permanent and irreversible modifications that determine how the mammary gland responds later in life, even in the absence of the initial effector. From the blood of adolescent girls with high urine concentrations of genistein, they identify protein biomarkers of exposure and susceptibility. Toxicology studies with genistein in animals and epidemiology reports with soy demonstrate little or no toxicity. The authors of this chapter recommend clinical studies in adolescent girls to determine if soy and genistein can suppress mammary cancer.

In the chapter 3 Manni, Karam El-Bayoumy, Thompson and, Russo with distinguished members of their teams are addressing the role of omega-3 fatty acids in breast cancer prevention**.** Preclinical and epidemiological data suggest that omega-3 fatty acids (n-3FA) protect against breast cancer. Preclinical data from their laboratories indicate that n-3FA potentiates the chemopreventive effect of the antiestrogen Tamoxifen based on the complementarity of their mechanisms of antitumor action suggested by the signaling, genomic, and proteomic studies. Because of their anti-estrogenic and anti-inflammatory properties, n-3FA may be preferentially effective in preventing obesity-related breast cancer. In view of the hyper-estrogenic and pro-inflammatory milieu present systematically and in the mammary glands of obese women, n-3FA may cooperate with weight loss induced by dietary energy restriction in reducing breast cancer risk in these subjects. Evidence-based combinatorial intervention trials targeting appropriately selected populations of women at risk are needed to establish the role of n-3FA in breast cancer prevention.

The Role for Raloxifene and Tamoxifen for the Prevention of Breast Cancer is discussed by Maximov and Jordan in the chapter 4. In this chapter the authors described the history, the current role and deficiencies of Tamoxifen and Raloxifene in the prevention of breast cancer. This chapter clearly illustrates the potential of other SERMs and new approaches to hormone replacement to improve women's health and to reduce the risk of breast cancer.

In the chapter 5 Brodie with her collaborators Chumsri, Yu, Schech and Sabnis described the use of aromatase inhibitors for breast cancer prevention. Aromatase inhibitors offer a new treatment option for breast cancer prevention without increased risks of venous thromboembolism and endometrial cancer. Compared to placebo, both Exemestane and Anastrozole significantly reduced the risk of not only invasive breast cancer but also non-invasive lesions.

In chapter 6 Cavalieri and Rogan present a lucid description of the role of specific estrogen metabolites in cancer initiation and how the understanding of their mechanism of action has lead their team to develop preventive strategies against breast and other types of cancer. Estrogens can initiate cancer by acting as chemical carcinogens and reacting with DNA. Specific metabolites of endogenous estrogens, the

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catechol estrogen-3,4-quinones, react with DNA to form depurinating estrogen-DNA adducts. Inhibiting formation of these estrogen-DNA adducts can, therefore, prevent cancer. The finding that high levels of estrogen-DNA adducts precede the presence of breast cancer indicates that formation of these adducts is a critical factor in breast cancer initiation. The discoveries of these two researchers led to the recognition that reducing the levels of estrogen-DNA adducts would prevent the initiation of breast and other types of human cancer.

Pereira, Su and Russo are presenting in chapter 7 the molecular basis of the preventive effect induced by pregnancy. It is a well-accepted knowledge that pregnancy exerts a protective effect in women who delivered their first child before late twenties, when compared to women that never had a full term pregnancy. In addition, multiple pregnancies significantly decrease the risk of developing breast cancer after 50years of age. The authors clearly explain the role of chromatin remodeling mechanisms in the long lasting preventive effect of pregnancy against breast cancer and how to mimic this protective effect using pregnancy-hormones or smaller targeting molecules. These concepts offer a new paradigm in the prevention of this disease.

In chapter 8 Gronich and Rennert discuss the current evidence available for the association between breast cancer and commonly used drugs suggested in the literature as carrying potential preventive activity against breast cancer in vitro, in animal models and in humans. These include vitamin D, bisphosphonates, statins and metformin, all of which are in use for a variety of non-cancer related indications. While all of these compounds have shown a high level of anti-breast cancer activity, in one or more of the different experimental platforms, none have been shown to be preventive in randomized controlled trials (RCTs). A common use of these compounds by the population, if actually have a true preventive effect, would lead to reduction in incidence of breast cancer in the population at large by way of a "natural experiment". The current reduction in breast cancer incidence and mortality seen in many western countries can actually be attributed, at least in part, to an inadvertent effect of these drugs.

In Chapter 9 Shapira has done a comprehensive update on the evidence base that supports the potential for nutritional prevention of breast cancer (BC) . The chapter reviews difficulties in studying direct nutrition-BC correlations, critical periods in the life cycle, and their dietary implications for carcinogenic and patho-metabolic trajectories. Evidence-based risk factors include anthropometric measures – high birthweight, adult tallness, fatness (body mass index), weight-gain; and reproductive events – early menarche, late childbearing without breastfeeding. Gender-based nutrition explains women's specific risk, i.e. with high fatness, estrogen metabolism, and n-6 polyunsaturated fatty acid conversion to pro-inflammatory/carcinogenic mediators. Recent large-scale studies have confirmed effectiveness of evidence-based recommendations for reducing BC risk, emphasizing low dietary energy density, nutritious plant-based diets, physical activity, and body/abdominal fatness management.

In chapter 10 Czerniecki, Nocera, Lowenfeld, Showalter and Koski introduce a fascinating new concept in breast cancer prevention by using vaccination against cancer cells. Vaccines have long been hailed as the most effective medical intervention to prevent a disease. While cancer vaccines have mostly been used therapeutically with little success in established breast cancer, their role in early breast cancer appears more promising, and primary prevention of breast cancer by vaccination is now being contemplated. Although there is no single cause of breast cancer, there are instead, multiple subsets of breast cancers including: Luminal A, Luminal B, HER-2, and subsets of basal-like cancer. Each of these types can be antigenically distinct, and present immune targets that may be phenotype-specific or to some degree overlapping between subsets. Three general categories of such targets are being developed as breast cancer vaccines. These include oncodrivers, breast tissue specific antigens, and cancer specific antigens. It is likely that combinations of these vaccine approaches may be best for treatment and prevention. Carriers of high-risk breast cancer mutations represent a potential target patient population for prevention. However, approximately 85% of breast cancers occur in patients with no identified risk. Recent evidence suggests that a loss of natural immune responses against oncodrivers may identify patients at risk for early breast cancer. Devising tests to identify subjects at risk for breast cancer are needed since these will allow us to focus prevention efforts, including vaccination, on those individuals where such resources are most needed. Preventive breast cancer vaccines may be achievable with our improved understanding of breast cancer biology, and the immune response in breast cancer.

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Conflicts of interest

Author declares there are no conflicts of interest.