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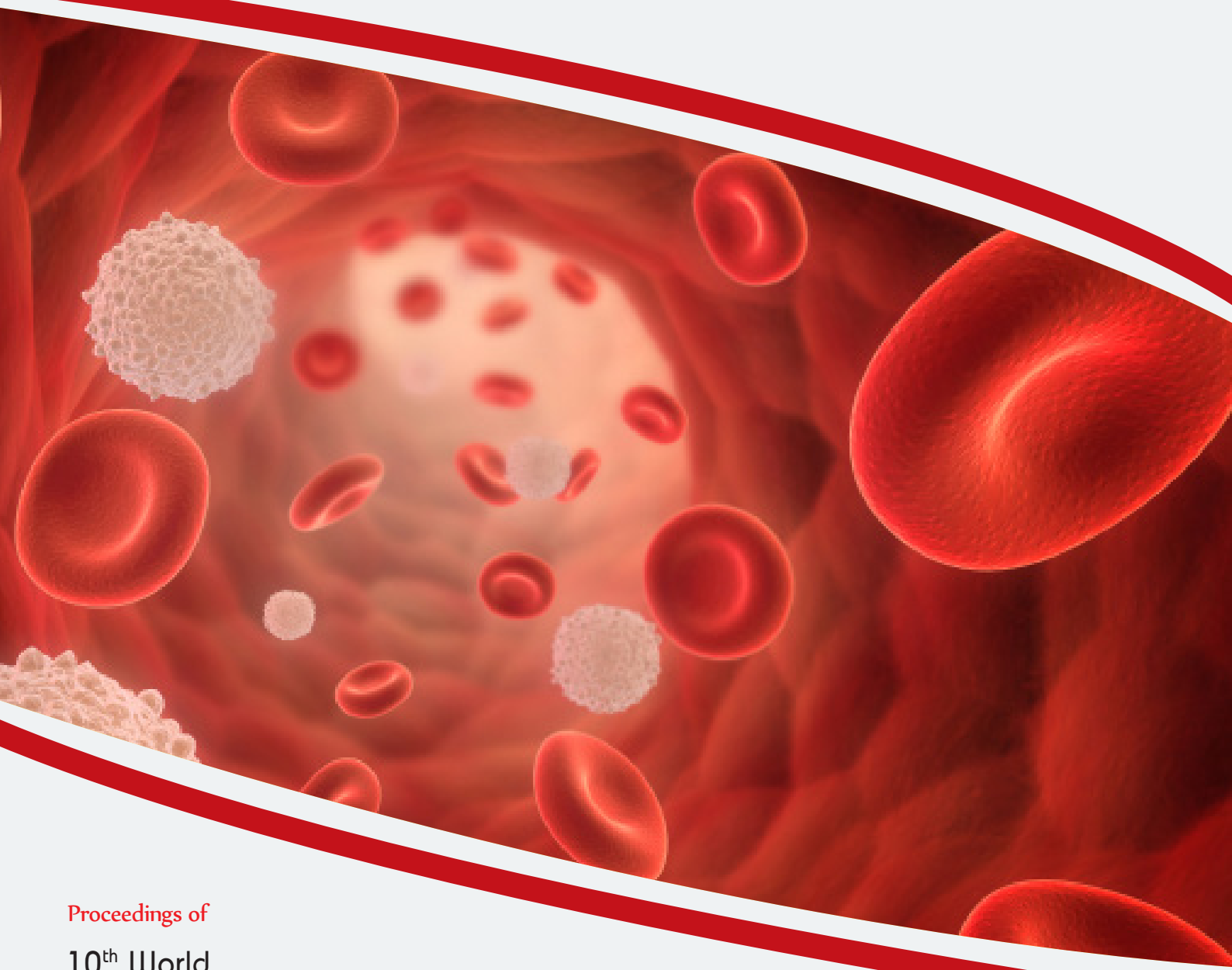

Proceedings of

10th World

Hematology and Oncology Congress

October 22-23, 2018 | Warsaw, Poland

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10th World

Hematology and Oncology Congress

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Keynote Forum

30 YEARS OF EXCELLENCE IN MEDICAL PUBLISHING

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Ojvind Lidegaard
University of Copenhagen, Denmark

Hormonal contraception and the risk of breast cancer

Background: We know little about the risk of breast cancer in users of newer types of hormonal contraception and in users of hormone intrauterine devices.

Method: We assessed associations between hormonal contraception use and invasive breast cancer risk in a nationwide prospective cohort study following all women in Denmark aged 15-49 years without previous cancer, venous thromboembolism or infertility treatment. Nationwide registers provided individually updated information about use of hormonal contraception, breast cancer diagnoses and information on potential confounders.

Result: Among 1.8 million women followed on average for 10.9 years with a total of 19.6 million person-years, 11,517 breast cancers occurred. Compared to never users, the relative risk of breast cancer among all current and recent users was 1.2 (95% CI 1.1-1.3), increasing from 1.1 (1.0-1.2) with less than one year of use to 1.4 (1.3-1.5) with more than 10 years of use. There was little evidence that the risk varied by type of progestogen in the combined formulations. Current or recent users of the progestin-only intrauterine system also experienced an increased relative risk of breast cancer of 1.2 (1.1-1.3). The overall absolute mean increase in breast cancers among current and recent users of any hormonal contraceptive for all ages was 13 (10-16) per 100,000 person-years, approximately one extra breast cancer for every 7690 women using hormonal contraception for one year, the absolute risk difference increased by age.

Conclusion: Breast cancer risk was increased among current and recent users of contemporary hormonal contraception and increased with longer durations of use however, absolute increases in risk were small and the same products protects against ovarian cancer.

Biography

Ojvind Lidegaard is a Professor of Obstetrics and Gynecology at The Juliane Marie Centre, Rigshospitalet, Copenhagen University Hospital and the University of Copenhagen, Denmark. He is also the Head of the National Quality Database in Early Pregnancy and Abortion. His main research areas include gynecological endocrinology, fetal exposure, postnatal diseases, gynecological cancer, early pregnancy and obstetrics.

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Wancai Yang

Jining Medical University, China

Chronic colitis and colitis-associated colorectal carcinogenesis

The activation of Wnt/beta-catenin signaling pathway and chronic colitis malignant transformation are the two major causes to colorectal cancer. The former is well studies, but the mechanisms of colitis develop and how chronic colitis progress to malignance is largely unknown. Using a unique mouse model, we have demonstrated that the mice with targeted disruption of the intestinal mucin gene *Muc2* spontaneously develop chronic inflammation at colon and rectum at early age, whose histopathology was similar to ulcerative colitis in human. In the aged mice, Muc2-/- mice develop colonic and rectal adenocarcinoma accompanying severe inflammation. To determine the mechanisms of the malignant transformation, we conducted miRNA array on the colonic epithelial cells from Muc2-/- and +/+ mice. MicroRNA profiling showed differential expression of miRNAs (i.e. lower or higher expression enrichments) in Muc2-/- mice. Based on relevance to cytokines and cancer, the miRNAs were validated and were found significantly down-regulated or up-regulated in human colitis and colorectal cancer tissues, respectively. The targets of the miRNAs were further characterized and their functions were investigated. More studies from the Muc2-/- mice showed disorder of gut microbiota. Moreover, a novel tumor suppressor PRSS8 also plays a critical role in colorectal carcinogenesis and progression, for instance, tissue-specific deletion of the PRSS8 gene resulted in intestinal inflammation and tumor formation in mice. Gene set enrichment analysis showed that the colitis and tumorigenesis were linked to the activation Wnt/beta-catenin, PI3K/AKT and EMT (Epithelial-Mesenchymal Transition) signaling pathways. Taken above, the disorder of gut microbiota could result in genetic mutations, epigenetic alterations and activation of oncogenic signaling in colorectal epithelial cells, leading to colitis development, promoting malignant transformation and mediating colorectal cancer metastasis.

Biography

Wancai Yang is the Dean of the Institute of Precision Medicine and School of Basic Medical Sciences, Jining Medical University, China and a Professor of Pathology, University of Illinois at Chicago, USA. He is also an Adjunct Professor of Biological Sciences, University of Texas, El Paso, USA. He has obtained his MD degree and was trained as a Pathologist from China and received his Postdoctoral training on Cancer Biology from Rockefeller University and Albert Einstein Cancer Center and then worked as an Assistant Professor. In 2006, he moved to the Department of Pathology, University of Illinois at Chicago. His research focuses on the determination of mechanisms of gastrointestinal carcinogenesis, identification of biomarkers for cancer detection and patient selection for chemotherapy and implication of precision medicine in cancers. He has published about 90 articles and has brought important impact in clinical significance.

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