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Closely related sets of genes are mutated in both breast and viral cancers

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Viral infections may play a larger role in breast cancer than previously thought. This is based on an in depth analysis of gene mutations in publicly available breast cancerDNA sequence data. Functions likely affected by ~8000 mutations in 124 breast cancer genomes and exomes were compared to model viral cancer genomes (14 HPV cervical cancers). Like the viral cancers, all the breast cancers had damage to many genes essential for immune responses and barriers to infection. Most breast cancers had mutations in genes for multiple cell attachments, potentially opening easier routes for infectious particles. Normal breast epithelial cells have a basement membrane encircling a layer of breast duct myoepithelial cells. In breast cancers, genes essential for this basement membrane had mutations and specific myoepithelial cell markers under the basement membrane were sometimes absent. Genes that regulate, produce and circulate fluid around individual duct cells were almost always damaged. Other mutations altered genes for the dense, tightly packed cytoplasm and could degrade its architecture, leading to abnormal cell shapes. The nucleus contains additional formidable infection barriers with gene damage. In all, breast cancers paralleled viral cancers with damage to about 1000 identical or closely related genes. Damage to the immune system and barriers to infection may be missing links relating viral infection to cancer causation.

Biography

Bernard Friedenson is a PhD research scientist authoring nearly 60 publications. Friedenson received an NIH research career development award and recently won an Innocentive Award in competition with nearly 400 other scientists. After a BA in honors chemistry-mathematics at the University of Minnesota Duluth and a PhD in biochemistry-organic chemistry at the University of Minnesota, he did post-doctoral work at Roswell Park Memorial Institute, where he rose to senior cancer research scientist, specializing in immunology. As a faculty member at the University of Illinois Chicago, he acquired 13 years further training in medical sciences, molecular medicine and genomics. His recent work found evidence that hereditary breast cancer gene mutations BRCA1 and BRCA2 increase risks in other organs beyond breast and ovary. Nonetheless he showed these mutations do not make breast or any other cancer inevitable. BRCA1 and BRCA2 gene mutations increase susceptibility to carcinogens, especially those capable of causing complex DNA damage. For example, mutation carriers may be unduly sensitive to alcohol and acetaldehyde carcinogens. However hidden alcohol and acetaldehyde are widespread in foods and confound epidemiologic studies trying to demonstrate this increased risk. He showed that differential exposure of organs to carcinogens and some infections play a major role in which organ develops cancer. His background in immunology enabled his recent finding that acquired mutations may cause defects in the ability of breast cancers to respond to microbial infection. This deficit may underlie breast cancer and even help target breast and ovary for BRCA1 and BRCA2 related cancers. He recently chaired sessions at an international cancer meeting and an online meeting. He is a member of the editorial board of BMC Research Notes, a member of the American Society of Preventive Oncology, the American Society of Clinical Oncology, and a reviewer for multiple journals. One of his publications is still advancing among the top 100 most accessed among about 360 BioMed Central journals. He has authored several highly accessed videos.

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