Breast cancer genomes have acquired mutations in essential immune defense genes. Although damage to the immune system favors cancer associated infections such as cancer viruses, there are still dense extracellular and intracellular structures that remain to physically block infection. Are these barrier defenses also damaged in invasive breast cancer? Cervical cancers caused by human papilloma viruses (HPVs) were used as a working model for cancer due to viral infection. Functional effects of ~6000 gene mutations in 14 cervical cancers were researched in depth. Functions damaged in this model viral cancer were then compared to functions damaged by ~3000 mutations in 21 complete breast cancer genomes and by ~5000 mutations in 103 breast cancer exomes. DNA sequences were from public databases. Mutations in breast and viral cancers occurred in nearly 1000 identical or closely related genes. Many mutated genes that were shared normally prevent infection, provide physical barriers against infection and prevent the spread of cancer. Loss of some genes in breast cancers can lead to loss or enzymatic digestion of structures surrounding and confining breast duct cells. Mutations in other genes damage cell membranes, allow abnormal cell shapes and interfere with normal cell adhesion, also favoring cancer spread. Additional gene damage opens routes for cancer cells and prevents removal of infection. Mutations affecting cell cycle control add to damage to immune and barrier defenses. Infections may play a larger role in breast cancer than previously believed. Variable damage to barrier defenses may explain variations in ductal carcinoma in situ biopsy results and may be useful in deciding on treatment options.

Biography

Bernard Friedenson is a PhD Research Scientist with nearly 60 publications. He received an NIH Research Career Development Award and an Innocentive Award in competition with nearly 400 other scientists. He received a BA in honors chemistry-mathematics at the University of Minnesota Duluth and a PhD in biochemistry-organic chemistry at the University of Minnesota. During Post-doctoral work at Roswell Park Memorial Institute, he rose to senior cancer research scientist specializing in immunology. At UIC he acquired 13 years additional training in medical sciences, genomics, and molecular biology. He is a member of the American Society of Preventive Oncology, the American Society of Clinical Oncology and an Editorial Board Member of BMC Research News.

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