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Opinion Article

Field Cancerization and the Development of Cutaneous Squamous Cell Carcinoma

Jonas Stattin*

Department of Surgery, Umeå University, Umea, Sweden

DESCRIPTION

The most prevalent type of cancer in the US are cutaneous malignancies, which include non-melanoma skin tumours. Squamous cell carcinomas and basal cell carcinomas make up the majority of them (cSCC). The incidence of cSCC is rising, which has a significant morbidity impact and leads to rising treatment costs—which are already exceeding $1 billion annually. Here, users discuss research describing the molecular foundation and development of cSCC that intends to offer fresh perspectives on pathogenesis and fuel the creation of cutting-edge, affordable, and morbidity-reducing treatments.

According to estimates, there are over 1.1 million new occurrences of Cutaneous Squamous Cell Carcinoma (cSCC) per year in the US, making it the second most frequent cancer among Caucasians. While the majority of patients have positive results and the majority of cSCCs may be completely removed to cure them, an estimated 3-7% of patients get metastases, which increase their risk of substantial morbidity and death. Hence, to reduce morbidity and save healthcare expenses, it is essential to detect and treat cSCCs and their premalignant precursors early. The majority of cSCCs develop over time from precursor lesions that are premalignant or noninvasive. Actinic keratosis is the earliest clinically discernible precursor lesion (AK). AKs can be recognised from neighbouring keratinocytes histologically by basal keratinocyte dysplasia and overlaying parakeratosis, as well as clinically by the presence of hyperplasia and hyperkeratosis. Acanthosis, which is connected to human papillomavirus infection or arsenic poisoning, and other, less frequent lesions that may also be precursors to keratinocytic malignancy share certain histologic features with AKs. Nonetheless, the AK, which may endure as premalignant lesions or even regress spontaneously, is the most extensively researched and well acknowledged precursor lesion in the field of cancerization hypothesis. A small percentage of AKs, incidentally, develop additional genetic and epigenetic alterations and progress to cutaneous Squamous Cell Carcinoma *In Situ* (SCCIS) and eventually cSCC, both of which are clinically larger lesions that share the same histological characteristics of parakeratosis with more pronounced dysplasia in which the full thickness of the epidermis is replaced by malignant keratinocytes that are either bounded by the basement membrane in the case of Smaller subsets of cSCC may develop new genetic and epigenetic characteristics that lead to a metastatic illness. Research is currently focused on the development of malignant characteristics that underlie this progression, and it includes the idea of "field cancerization," which asserts that precancerous lesions like AKs and SCCIS develop from mutated, subclinical clones of keratinocytes within a clinically unremarkable epidermis. UV exposure typically starts the mutagenic process in skin, causing mutations in specific keratinocytes that, if they have a chance of surviving, are chosen over time. Due to this selection, clinically normal-appearing skin contains a number of mutant keratinocyte subclones. Further genetic and epigenetic alterations in these clones may encourage neoplastic selection, leading in AKs, SCCIS, and finally polyclonal cSCCs made up of many, antagonistic keratinocyte clones. Molecularly, cSCC occurs from the accumulation of genetic and epigenetic changes in keratinocytes over time that allows growth of an invasive tumour.

When inherent defences are weakened, the accumulation of genetic changes and the emergence of malignant and premalignant lesions are sped up. Examples include people who are receiving long-term immunosuppression or those who have a hereditary propensity for cancer, such as individuals who have Xeroderma Pigmentosum or Bloom syndrome. Defects in DNA replication, repair, or recombination pathways can lead to DNA mutations that affect the way genes are expressed in a qualitative way. Endogenous mutagens cause DNA to spontaneously change, such as depurination, deamination, or free radical damage brought on by reactive oxygen species. Sunlight (UVB and UVA), smoking, and dietary components are examples of exogenous mutagens. Inappropriate transcriptional activation and silencing of genes can also be facilitated by epigenetic modifications that result in quantitative changes in gene expression. The total mutation rate, cell proliferation, and cell death can all be influenced by genetic and epigenetic changes.

Correspondence to: Jonas Stattin, Department of Surgery, Umeå University, Umea, Sweden, E-mail: jonas.stattin@urologi.umu.se

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