

UVB Induced Skin Cancer Development in Experimental Mouse Model: A Review

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Abstract Skin cancer is a widespread global issue, with ultraviolet (UV) radiation being a significant risk factor. Researchers often use the mouse skin cancer model to develop novel therapeutic chemoprevention strategies. This model involves exposing mice to UVB radiation to induce skin cancer. In most studies, hairless mice were often used for their resemblance to human skin. However, using hairless mice is costly. Therefore, as researchers look into a more cost-effective model as an alternative to be adapted for skin carcinogenesis studies, in this review, we summarised that 69.57% of studies used female SKH-1 hairless mice, 17.39% used BALB/c mice, 8.69% used Swiss albino mice, and 4.35% used HRS/J hairless mice. All studies used mice aged 5-8 weeks. Different models of mice were irradiated with various doses of UVB. SKH-1 hairless mice received UVB radiation twice a week for 10-18 weeks, while Swiss albino mice were exposed to UVB radiation three times a week for 30 weeks. HRS/J hairless mice received UVB radiation five times a week for 15 weeks. BALB/c mice were treated with DMBA and exposed to UVB radiation for 10-16 weeks to induce skin tumors. In conclusion, we can suggest adapting female BALB/c mice, aged 6-8 weeks, treated with DMBA and exposed to 180 mJ/cm² UVB radiation three times a week for 16 weeks for the UVB-induced skin cancer model, as it is more cost-effective than other hairless mouse models.

Keywords: Mice model, carcinogenesis, chemoprevention, skin cancer, UVB.

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Introduction

Skin cancer is a serious health concern that is increasing globally [1]. A multi-step process that ends in the development of malignant skin tumors by a series of distinct phases is shown in Figure 1. Skin cancer begins with initiation, a reversible mechanism brought on by genetic alterations, gene activation, or gene inactivation. Tumor promotion is the next step of carcinogenesis, and it contributes to the growth of benign skin lesions called pre-neoplastic papilloma. Vascular permeability, the initial stage of angiogenesis in skin tumors, occurs concurrently with tumor promotion. Pre-neoplastic lesions grow blood vessels to provide oxygen and other nutrients in a multi-step process. According to Robertson [2] tumor progression is the next stage of carcinogenesis, which occurs when genetic mutations accumulate, resulting in the transformation of pre-malignant skin lesions into cancer, that is most commonly squamous cell carcinomas. Additionally, changes in the expression profiles of particular proteins have been linked to skin cancer [3, 4].