Actinic keratoses (AKs) are common skin lesions heralding an increased risk of developing squamous cell carcinoma (SCC) and other skin malignancies, arising principally due to excessive UV exposure. They are predominantly found in fair-skinned individuals, and are becoming an increasing problem in the immunosuppressed.

AKs may regress spontaneously, remain stable or transform to invasive SCC. The risk of SCC increases for those with more than 5 AKs, and the majority of SCCs arise from AKs.

The main mechanisms of AK formation are inflammation, oxidative stress, immunosuppression, impaired apoptosis, mutagenesis, dysregulation of cell growth and proliferation, and tissue remodelling. Human papilloma virus has also been implicated in the formation of some AKs.

Understanding these mechanisms guides the rationale behind the current available treatments for AKs.

One of the main principles underpinning the management of AK is that of field-cancerisation. Wide areas of skin are exposed to increasing amounts of ultraviolet (UV) light and other environmental insults as we age. This is especially true for the head, neck and forearms. These insults do not target only the skin where individual lesions develop but large areas where crops of AKs may appear. The skin between lesions is exposed to the same insults and is likely to contain as-yet undetectable preclinical lesions or areas of dysplastic cells. The whole affected area is known as the ‘field’.

Management is therefore divided into lesion-directed and field-directed therapies.

Current therapies include lesion-directed cryotherapy and/or excision, and topical field-directed creams: 5-fluorouracil, imiquimod, diclofenac, photodynamic therapy and ingenol mebutate. Combining lesion and field directed therapies have yielded good results and several novel therapies are under investigation. Treatment is variable and tailored to the individual making a gold standard management algorithm difficult to design.