Chapter 1

Introduction

1.1 Background

Indonesia is located within the equatorial region, resulting in intense and consistent ultraviolet (UV) radiation exposure throughout the year. As the largest organ of the human body, the skin functions as the primary physical barrier against environmental stressors, including pathogens, chemicals, and solar radiation (Lee & Kim, 2022). Chronic exposure to UV radiation is a major driver of skin photoaging and cutaneous damage, with ultraviolet B (UVB) radiation playing a particularly critical role in epidermal injury (Chen et al., 2021; Hidayati et al., 2023).

UVB radiation (280–320 nm) penetrates the epidermis and directly induces DNA damage, oxidative stress, and inflammatory signaling in keratinocytes. These events impair cellular proliferation and migration, two key processes required for epidermal repair and wound re-epithelialization, ultimately contributing to delayed wound healing and premature skin aging (Tang et al., 2024). As a result, identifying interventions capable of enhancing post-UVB cellular recovery has become an important focus in dermatological and skin recovery research.

Current dermatological approaches to address photoaging and UV-induced skin damage include topical treatments, device-based procedures, and clinic-administered injections e.g., fillers, injectable growth factors, and collagen stimulators (Anggraini & Damayanti, 2024). Although these approaches can be effective, they are often costly, require repeated clinical administration, and are unsuitable for daily use, particularly in populations exposed to high UV levels on a daily basis. Consequently, there is an increasing need for safe, affordable, and accessible interventions that support skin recovery following routine UV exposure, particularly in tropical regions.

Natural plant-derived compounds have gained attention as potential alternatives for low cost, topical cosmeceutical agents. Essential oils (EOs) are complex mixtures of bioactive terpenoids and phenolic compounds that have demonstrated antioxidant, anti-inflammatory, antimicrobial, I'd wound-healing properties in both in vitro and in vivo models (Pomi et al., 2023; Hulankova, 2024). Among these, Frankincense essential oil (FEO), derived from Boswellia species, contains monoterpenes hydrocarbons and oxygenated compounds reported to modulate inflammatory pathways and enhance wound healing (Kotb et al., 2023). Similarly, Labdanum cistus essential oil (LCEO) has

demonstrated antioxidant and biological activities relevant to skin protection and tissue repair (Frazão et al., 2022). Based on the biological activities exhibited in past studies, these EOs may serve as potential solutions for UVB-damaged keratinocytes.

Despite their promising biological activities, most studies investigating EOs focus primarily on their protective or preventive roles prior to UV exposure. Their potential to enhance cellular recovery after UVB-induced damage remains insufficiently explored. This represents an important research gap, particularly in the context of developing accessible and natural interventions for skin recovery under chronic UV exposure.

This study aims to establish an in vitro UVB damage model using human keratinocytes (HaCaT) keratinocytes and investigate the recovery effects of FEO and LCEO in HaCaT following UVB-induced damage. By examining post-UVB cellular metabolic activity, viable cell number, and wound closure, this research seeks to provide experimental evidence supporting the use of EOs as affordable and accessible agents for promoting skin recovery under conditions of chronic UVB exposure.

1.2 Objective

The general objective of this research is to evaluate the recovery effects of FEO and LCEO in UVB-damaged HaCaT keratinocytes. The specific objectives include: (1) To determine non-toxic treatment concentrations of FEO and LCEO; (2) To establish and optimize a UVB-induced keratinocyte damage model; (3) To evaluate post-UVB cellular metabolic activity following EO treatment using MTS assay; (4) To evaluate viable cell number following treatment through manual cell counting; and (5) To assess scratch closure capacity using scratch assay.

1.3 Hypothesis

This study is based on the hypothesis that treatment with FEO and LCEO will improve cellular recovery of UVB-damaged HaCaT keratinocytes, reflected by enhanced cellular metabolic activity, viable cell number and/or scratch closure.