Modulation of B10R mouse macrophage signalling pathways using non-thermal atmospheric pressure plasma treatments

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In recent years, there has been growing interest in developing non-thermal atmospheric pressure plasma (NAPP) devices for biomedical applications. Studies investigating interactions between cells and NAPPs have uncovered many potential applications, including sterilization of living tissues, apoptosis of cancer cells, blood coagulation and wound healing[1]. The latter was proposed given the capacity of NAPP devices to produce nitric oxide (NO). In the process of wound healing, NO is abundantly produced by inflammatory cells, especially macrophages, during inflammation[2]. In this stage, the main role of NO is as a cytotoxic agent as part of the non-specific immune response against pathogens such as bacteria, virus, parasites and fungi. Furthermore, NO also acts as a signalling molecule which mediates important events during wound healing, including cell proliferation, collagen formation and gene expression[3]. In order to further understand the effects of NAPP during wound healing, the interactions between cells present in the wound system and NO-producing NAPP must be explored.

In this study, the effect of a NAPP device on B10R mouse macrophage cells is investigated. The NAPP device produces a miniature atmospheric pressure plasma jet, with helium as the main plasma gas, driven by a pulsed radio-frequency power supply. Reactive species generation in the plasma is characterized using optical emission spectroscopy, demonstrating effective production of NO, hydroxyl radicals, atomic oxygen and other excited species. Effective transport of NO from the jet to the cells is demonstrated, and the post-treatment viability of cells following treatment is assessed. The treatment is shown to induce endogenous NO generation by the cells. Signalling-protein activation by phosphorylation following the plasma treatment is also shown to occur. Proteins tested for activation include protein kinase C isoforms, tyrosine kinases and p38 mitogen-activated protein kinases.

References

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