ABSTRACT

The study of the mechanisms that control wound healing is an attention-drawing area within the fields of biology and medicine. Wound healing can be usually defined as two basic types. The first type is adult wound healing, which is slow and results in the scar formation. The second type is referred to as embryonic wound healing, which is in contrast fast and scarless. Wound healing is a complicated process that includes many steps, which are regulated by various types of molecules. One of these important molecules is nitric oxide (NO). Its function is usually connected with the regulation of inflammation and angiogenesis during adult wound healing. However, there is currently no information on its role during embryonic wound healing, where the immune and vascular systems are not yet developed. In this work, we explore and describe the role of the NO during the healing of the early embryos.

The highest concentration of the NO post wounding is produced during the first 30 minutes after injury. This applies to all developmental stages, from the blastula stage all the way to the swimming tadpole stage. The main role of the NO during embryonic wound healing is the regulation of the gene expression that is connected with the stress response and the regulation of cellular metabolism. Additionally, we observed that the effects of NO production are also observable even few hours after the post wound closure, in what we describe as a third phase of the embryonic wound healing. The third phase deals with the specific remodelling of the tissue around the closed wound area. Lastly, we also discovered that NO regulates the expression and activity of the matrix metalloproteinases and the migration of naïve immune cells to the wound site during this phase.

This research describes a previously undescribed mechanism for the role of NO during the process of embryonic wound healing and potentially opens new strategies for the treatment of the problematic non-healing wounds.

Keywords: *Xenopus laevis*, nitric oxide, wound healing, gene expression, matrix metalloproteinases, AP-1, leptin