Elasto-dynamics of Embryonic Epidermal Wound Closure

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Abstract

This talk is concerned with the elastodynamics of embryonic epidermal wound closing. Underlying the wound recovery process is a proposed mechanism of wound recognition through directed cell-to-cell signaling. The observed actin filament realignment induced by the biological signals leads to a purse-string effect and the resulting (unknown) "active stresses." The circumferential contraction of the epidermis surrounding the wound is then determined by the law of mechanics and propagation properties of the relevant cell-cell signaling that decays with distance. With the wound known to retract for a short period immediately after infliction, the quasi-equilibrium configuration reached during this initial phase serves as the initial condition for the dynamic wound closing phase. A small strain variation of the Sherratt-Murray model of the quasi-equilibrium problem will be formulated for speedy computation of this initial state at the inception of the wound closure phase, with the latter problem being our main interest. For this talk, we limit ourselves to a small strain model also for the wound closing phase for computational expediency. Even then, some theoretical developments are found to be instrumental to an efficient algorithm for the otherwise time consuming task of calculating the effect of the biological signals generated by the presence of a wound. Application of the elasto-dynamic model developed to the case of a circular wound suggests that the signal propagation range must be above a certain minimum fraction of the wound radius in order for a wound to closure. As expected, stress concentration occurs adjacent to the edge of the remaining small wound near the end of the wound closing process. At that point, the present small strain model may no longer be adequate and replacement of our strain expressions by those analogous to the Sherratt - Murray type would be in order.

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