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## BIOMECHANICS OF VISCERAL PAIN: UNDERSTANDING AND MODELING PERITONEAL ADHESIONS.

Madge Martin\*, Thibault Lemaire

CNRS, Univ Paris Est Creteil, Univ Gustave Eiffel, UMR 8208, MSME, F-94010 Créteil, France

\*madge.martin@u-pec.fr

### 1. Introduction

Peritoneal adhesions are pathological fibrotic connections forming between organ surfaces. They typically follow a surgical procedure of the thorax, abdomen or pelvis, or result from inflammatory diseases and can cause small bowel obstruction, chronic pain or infertility. Nowadays, despite a lack of awareness in patients and surgeons, adhesions are now identified as a major economic and health burden. However, the strongest obstacle remains the poor understanding of underlying mechanisms of tissue physiological repair and adhesion formation [1]. Moreover, the role of biomechanics is critical to provide better understanding, prevention and treatments for peritoneal adhesions, especially regarding pain management. Indeed, nociception and mechanics are highly connected [2]. We present a novel and first model of peritoneal adhesions which depicts the main biochemical mechanisms associated with adhesion formation and their mechanical consequences.

### 2. Materials and Methods

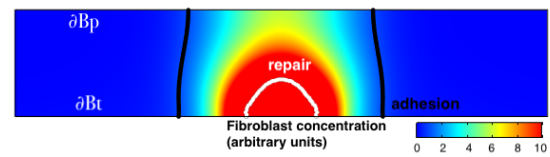
We propose here a 2D model of adhesion formation representing the volume  $\mathcal{B}$  between the peritoneal serosa  $\partial\mathcal{B}_p$  and the tissue boundary  $\partial\mathcal{B}_t$ , where the following mechanisms occur: (i) the formation of the fibrin exudate upon tissue insult, (ii) FB-mediated fibrosis and (iii) typical cyclic loading simulating breathing. We implemented a system of partial differential equations depicting the time- and space- dependent evolution of fibrin ( $F_n$ ), tPA ( $t_{PA}$ ) and FB ( $b$ ) concentrations, accounting for fibrin-mediated chemotaxis of FB (sensitivity  $\chi$ ) in volume  $\mathcal{B}$ :

$$\frac{\partial}{\partial t} \begin{matrix} b \\ F_n \\ t_{PA} \end{matrix} = \nabla \cdot \begin{bmatrix} \mu_{Fb} \nabla b - \chi b \nabla F_n & b \\ D^{F_n} \nabla F_n & -\mathbf{d} \cdot F_n \\ D^{t_{PA}} \nabla t_{PA} & t_{PA} \end{bmatrix}, \quad (1)$$

where  $\mathbf{d} = [a_b, d_{F_n}, d_{t_{PA}}]$  accounts for FB apoptosis  $\mathcal{A}_b$ , fibrin ( $d_{F_n}$ ) and tPA degradation ( $d_{t_{PA}}$ ). The time-derivative of the collagen content  $c$  depends linearly on FB activity and collagen degradation. Boundary conditions and material properties are Outside the injury, the coagulation factor tPA is set to a constant flux  $\phi_{t_{PA}}^0$  to account for the action of MCs and decreased to 1%  $\phi_{t_{PA}}^0$  for a pathological repair.

### 3. Results

Figure 1 depicts FB seeding into the fibrin exudate, producing collagen. The lines mark the bounds of collagen-rich volumes in adhesive and healthy repairs, respectively (threshold  $c \geq 100 \mu\text{g}/\text{mg}$ , for dense adhesions). In the case of a healthy repair, the inhibition of fibrin by tPA-mediated fibrinolysis prevents formation of the dense connective tissue (white), thus preserving the mechanical behaviour of the system.



**Figure 1:** Fibroblast concentration 10 days after tissue trauma, resulting in an adhesive bond (black) or repair (white) depending on fibrinolytic activity.

### 4. Discussion and Conclusions

This is the first attempt to quantify the mechanisms of adhesion formation. We successfully depicted the main events following tissue trauma. Future work will investigate the next remodelling steps, in particular involving innervation and *in vivo* mechanical stimulation.

### 5. References

1. Hassanabad et al., Biomed; 9(8):867 (2021).
2. Feng et al., J Neur Trans; 127(4):415–429 (2008).