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Description

Viral infection causes major endothelial dysfunction disorders that involve the immune system, the in ammatory response and the apoptosis-angiogenesis interdependency which is a major process in tissue repair and regeneration [1]. Restoration of the unbalanced angiogenesis-apoptosis pathway in viral infection, as well as in wound healing, depends on endothelial functions which are regulated by shear stress [2].

As a reminder, Endothelial Shear Stress (ESS) controls and maintains endothelial functions [3], as well as vasculogenesis, cardiogenesis, embryogenesis, organogenesis through the angiogenesis-apoptosis interdependency process, from the 8th day of gestation until death. Also, ESS stimulates chemical facilitators like monocyte chemo attractant protein-1 (MCP-1); TNF- ; bFGF and MMPs and promote potent vasodilators like Nitric Oxide (NO) [4,5].

Despite the ability of endothelial cells to adapt to various pathological conditions [6], the outcome of post-viral infection depends on the host biological responses, which are governed by the diversity of patients' conditions, viruses and target organs [7-10]. is is clearly demonstrated by the controversial results of post-viral remodeling and metabolic processes of vital organs such as liver and brain [11,12], which become even worse with dynamic vital organs like the cardiopulmonary system. As restoration and maintenance of endothelial functions, the key vector to complete recovery of the hosts metabolic processes, depends on ESS-inducing circulatory driving forces, causing the therapeutic dilemma of cardiopulmonary viruses [13].

Taking the example of the Covid-19 virus which invades host cells via Angiotensin Converting Enzyme Receptor 2 (ACE2) [14], we can recognize that most of the patients succumb to multiple organ failure and/or Sudden Cardiac Arrest (SCA) as a result of aggravated endothelial dysfunction disorders, whether in the form of comorbid conditions e.g., arterial hypertension, mediated by the virus, e.g., in ammatory response and/or iatrogenic, e.g., thromboembolic syndrome [15]. Likewise, the outcome of viral myocarditis like almost all types of Dilated Cardiomyopathy (DCM) remains a potentially life-threatening condition [16,17]. e restoration of the unbalanced angiogenesis-apoptosis pathway in post-viral DCM that requires neovascularization with a full-thickness myocardial reconstruction, is principally regulated by vasculogenesis, angiogenesis and cardiogenesis endothelial function processes [18].

As is known, the early vasculogenesis process begins at the embryonic endometrial implantation around the 6th day of gestation, to create the rst blood vessels from blood islands, to be followed by sprouting-splitting angiogenesis to construct the whole cardiovascular system: heart, vessels and blood components [19,20]. Similarly, early cardiogenesis process which employs the constitution of endocardium, myocardium (atrioventricular myocytes and Purkinje bers) and epicardium from the original cardiac mesoderm precursor cells (cardiogenic mesoderm) [21], depends on both Endocardial Endothelium (EE) and Myocardial Capillary Endothelium (MyoCapE),

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a vicious circuit of energy losses and endothelial dysfunction raised by opposing hydraulic circuits [13,34].

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