Neuroimmune-vascular cells and their pathological disorders

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The initiation and persistence of multiple organ pathologies, which may occur during life, are intimately associated with acute and chronic dysregulatory metabolic processes. Multiple organ pathologies may also be functionally associated with initiating traumatic events leading to a chronic state of proinflammatory cellular insults with resultant prolonged debilitation and negative quality of life. Along these lines, a large body of preclinical and clinical literature has established that acute and chronic perturbations of the functional coupling of vascular endothelial, immune, and neural cell types represent major contributory factors to the initiation and persistence of pathological states [1]. Furthermore, metabolic rundown in each of these cell types is intimately linked to primary functional deficits in their endosymbiont mitochondria. Mechanistically, dysregulation of mitochondrial respiration leads to impairment of intermediary energy metabolism, ATP production, and insufficient buffering of harmful reactive oxygen species [2]. Resultant damage to mtDNA has profound effects on mitochondrial rescue and biogenesis upon physiological demand. Similar criteria may relate to the elucidation of key cellular mechanisms underlying normal and premature ageing. In light of the above, common regulatory events and their potential dysregulation in neuroimmune-vascular disorders are listed below.

Endothelia serve as major cellular conduits mediating oxygen and carbon dioxide exchange, nutrient transport, and autonomic tone in the peripheral and CNS vasculature. Notably, functional deficiencies in vascular endothelia has been documented in variety of human metabolic disorders including diabetes and cardiovascular disease [3-19]. Mitochondria-associated oxidative stress has been proposed as a major contributing factor to the severity of endothelial malfunction in these same metabolic disorders [3-19].

The immune system serves to defend the body from external and internal threats and keep multiple organ systems in metabolic homeostasis. Ongoing host-defense mechanisms utilize complementary regulatory processes to perform existential functions ranging from antibody production to immune surveillance and phagocytosis. Functional immunodeficiencies including perturbations in neuroimmune and vascular endothelial cellular coupling has been observed in multiple human pathological disorders [1, 20-34]. It has also become apparent that dysregulation of mitochondrial function mediated by disruption of constitutive nitric oxide signaling mechanisms represents a strong

contributing factor in neuroimmune/vascular endothelial-coupled cellular pathologies [1, 20-34].

The nervous system serves an existential integrative capacity for functional entrainment of multiple organ function. Within the central and peripheral nervous systems, sensory, motor, and intrinsic neurons employ a wide variety of chemical and gaseous messenger molecules. Accordingly, central and peripheral neurons integrate sensory input and coordinated motor responses via immediate and long lasting chemical messengers. As stated above, vital neuroimmune/vascular endothelial-coupled metabolic regulation modulates homeostasis of cellular energy production in multiple organ systems. Its dysregulation is intimately associated with a variety of acute and chronic pathophysiological disorders [35-55].

Multiple organ pathologies are intimately associated with acute and chronic dysregulation of metabolic processes responsible for normal neuroimmune/vascular endothelial coupling events. An in depth analysis of perturbation of common regulatory events underlying normal mitochondrial function will elucidate convergent mechanisms leading to the initiation and persistence of multiple organ pathologies in human metabolic disorders.

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