VARIABLE RISK OF HEART DISEASE AND INBORN IMMUNITY

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Abstract: The immune system, when functioning optimally, serves to protect the body against pathogens and other harmful entities. However, in the presence of hyperlipidemia, the immune response can become maladaptive, contributing to the pathogenesis of diseases. This maladaptive response is often characterized by chronic inflammation, which is a common underlying factor in the development of atherosclerosis, a leading cause of cardiovascular events.Recent insights suggest that the interplay between lipids and the immune system is complex and bidirectional. For instance, certain lipoproteins have been found to modulate immune cell function, influencing the development of atherosclerotic plaques. Conversely, immune cells can influence lipid metabolism, affecting the levels and types of lipoproteins in circulation. This dynamic relationship means that changes in lipid levels can alter immune responses, and immune responses can, in turn, affect lipid levels, creating a feedback loop that can either protect against or promote disease progression. The innate immune system, in particular, plays a crucial role in this interaction. Modulating the immune response to hyperlipidemia has the potential to become a novel approach in treating related diseases. For example, therapies aimed at reducing lipid levels could also have beneficial effects on immune function, while immunomodulatory therapies could potentially influence lipid metabolism and reduce the risk of disease progression. Furthermore, lifestyle interventions such as exercise have been shown to have both lipid-lowering and anti-inflammatory effects, suggesting that they could be an effective strategy for simultaneously addressing hyperlipidemia and maladaptive immune responses. Such interventions could be particularly valuable as they address multiple modifiable risk factors and have the potential to improve overall health outcomes. Macrophages, white blood cells that are part of the innate immune system, are found in large numbers in all healthy organs, where they closely interact with their surroundings. While these studies clarify that leukocyte production and phenotype are shaped by exposure to stress, diet choice and sleep hygiene, to date the uncovered mechanisms have relied on cellular and molecular components well known from work in neuroscience, metabolism, hematology and immunology. Connecting these fields in interdisciplinary teams reveals those cross-cutting pathways. Lifestyle choices play a pivotal role in the health of multiple organ systems, particularly influencing the risk of cardiovascular events. Research indicates that nearly half of all premature deaths may be attributed to modifiable lifestyle factors such as physical inactivity, poor diet, and smoking, which are known to exacerbate conditions like hypertension, diabetes, and dyslipidemia, ultimately leading to heart attacks and strokes. The central nervous system is affected by stress and sleep patterns, while the endocrine system is influenced by diet and physical activity, impacting metabolic functions. Hematopoietic and immune system health can be compromised by behaviors that affect inflammation and clotting, which are critical in the development of atherosclerosis. The culmination of these effects on the cardiovascular system can be mitigated by adopting healthier behaviors, underscored by guidelines from leading health organizations which recommend regular exercise, a balanced diet rich in fruits, vegetables, and whole grains, and the avoidance of tobacco and excessive alcohol consumption. These changes, while challenging to implement, have been shown to significantly reduce the risk of cardiovascular disease and improve overall health outcomes. However, the currently known mechanisms are unlikely to be the only important players; rather, they establish a proof-of-principle. The experimental platforms described herein, including sleep disruption, chronic mild stress and voluntary exercise are robust, have been used in their respective fields for multiple years and are straightforward to implement in any laboratory. We posit that these tools

can serve as discovery platforms in the search for currently unknown pathways and targets that build resilience against cardiovascular inflammation