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Editorial

Ischemic Stroke: A Consequence of a Diseased Immune System?

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Stroke is a leading cause of long-term disability and the second leading cause of death globally. Approximately 795,000 strokes are reported each year in the US, 87 percent of which are ischemic.[1] The direct medical cost associated with stroke in 2009 was approximately $22.8 billion, with an additional $13.8 billion in indirect costs associated with lost productivity, unemployment, rehabilitation, and follow-up care [1]. The incidence of stroke is expected to increase dramatically over the next decade primarily because of an increase in the elderly population and increased incidence of stroke among the middle-aged population. We must develop and implement improved acute and long-term care options to mitigate the increasing burden of stroke.

This special edition was designed to address innovative areas of stroke research that are expected to have a tremendous impact on current and future clinical care of stroke patients. Historically, ischemic stroke has been studied and treated as a disease of the cerebrovascular system. However, there is a well-characterized and understudied immunological response to ischemic stroke that provides an opportunity for targeting novel therapeutic and rehabilitation strategies. In the opening articles, Doll et al. and Famakin describe the innate and adaptive immune response to stroke and suggests the time window for targeting the immune response to stroke may be longer than previously expected. Studies are needed to follow stroke patients over the course of their injury and recovery to identify time specific patterns of cytokine responses and potential avenues for anti-inflammatory treatment beyond the acute care period. In the third article by Schulze et al, we are reminded of the significant contribution the catecholamine and steroid responses have on guiding the immune response to stroke recovery. Petrone et al. also describes the effects of estrogen on the immune response and how this modulation of the immune response may account for sex differences in ischemic stroke incidence and severity. Continued study in this area will not only be important for stroke, but also other types of brain injuries (traumatic, blast, neurodegenerative) that inevitably alter the hormonal balance and subsequently have immunological consequences.

It is interesting to consider whether a common mechanism of immune dysfunction underlies many complex diseases, such as stroke, heart disease, myocardial infarction, and atherosclerosis. While it is possible that the immune system is similarly altered, gene-environment interactions may control the timing or severity of the dysfunction, and epigenetic modifications may provide a common molecular mechanism to link the immune response among different disease states. In their review, Saban et al. propose a new theoretical model to guide ischemic stroke research to further understand the extent to which epigenetic signatures bridge the psycho-social environment. Our knowledge of the epigenetic control of the immunological consequences of ischemic stroke is limited and requires a new approach to clinical and pre-clinical studies. The question remains whether we can truly model the human condition of ischemic stroke. Given the advances in genomic technology, systems modeling, and biomedical engineering we must re-invent stroke research and begin to utilize human patients as the best model to study the condition. Thus, new collaborations between scientists, clinicians, engineers and patients will be the key to future success.

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We hope this special edition broadens your horizons to the possibility of ischemic stroke being an immunological disease. Before we can begin to make significant improvements in clinical care of stroke, we must strengthen our research collaborations and be creative in our approach. Our patients depend on it.

References

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