Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Type I interferon

In this issue of Cytokine, we present a Special Issue on type I interferon. We highlight recent work relevant to human disease pathogenesis, and cover a diverse range of topics and conditions. Type I interferon (IFN) is a particularly relevant topic this year, as the COVID-19 crisis highlights the importance of anti-viral immunity. In addition to being a critical mediator of the anti-viral response, it is also clear that the type I IFN pathway is over-active in many autoimmune diseases [1]. This year we also saw the first successful phase clinical III trial of specific type I IFN antagonist - an antibody that blocks signaling via the type I IFN receptor was successful in treating lupus patients [2]. While the type I IFN family of cytokines is not new, it has been an exciting and eventful year for the type I IFNs.

In this Special Issue, Hile et al comprehensively review the role of type I IFNs in the skin in various cell types, and how these impact a variety of autoimmune and inflammatory conditions [3]. Also on the topic of skin inflammation, Skaug et al review type I IFNs in the autoimmune disease scleroderma, in which skin fibrosis is a cardinal feature [4]. Three papers address genetic variants in the type I IFN pathway in human populations. Previous studies support the concept that activation of the type I IFN pathway varies between individuals based upon genetic variation [5], and this likely impacts autoimmune disease susceptibility and antiviral responses. Matta et al review the role of the interferon regulatory factor 5 (IRF5) transcription factor and type I IFNs in various immune cell types as they relate to lupus [6]. IRF5 has been genetically associated with risk of lupus [7], and the authors review both the genetic and non-genetic aspects of IRF5 in lupus pathogenesis. Nezos et al study the impact of genetic variations in TREX1 upon lymphoma risk in the autoimmune disease Sjogren's syndrome [8]. Sjogren's syndrome is characterized by elevated type I IFN levels in blood, and the TREX1 gene has been linked to monogenic interferonopathies [9]. Ghodke-Puranik et al perform a replication and fine-mapping study to identify genetic polymorphisms that are associated with higher levels of type I IFN in lupus patients, and identify a number of novel polymorphisms that contribute to IFN levels in this condition [10]. Gao et al study the impact of type I IFN on bone marrow mesenchymal stem cells [11]. They studied human bone marrow aspirates from lupus patients, and found a discrete subset of bone marrow mesenchymal stem cells characterized by upregulation of type I IFN-induced genes which correlated with type I IFN in circulation [11]. And Choubey et al review the type I IFN-inducible Absent in Melanoma 2 (AIM2) proteins as regulators of the type I IFN pathway, and how these proteins may impact lupus kidney disease [12].

The papers brought together in this Special Issue illustrate some of the ways in which type I IFN can impact human disease. This of course cannot be comprehensive, as the type I IFN pathway is involved in a wide array of diseases, including defense against infection, cancer immunity, and autoimmunity which is strongly represented in the papers in this issue. As noted above, it is an exciting time for the type I IFN field, and these papers in autoimmune disease will likely inform ongoing work in other areas of medicine and biology as well. We expect that rapid progress will continue in type I IFN research, and we are pleased to present the articles in this Special Issue as a contribution to this effort.

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