The intriguing subset of effector CD4+ T cells termed T\textsubscript{H}17 cells are now widely appreciated for their role in coordinating immune and inflammatory responses. The dynamic nature of the T\textsubscript{H}17 cell subset allows for the adoption of inflammatory or regulatory functions as needed, in a microenvironment-dependent fashion. The ontogeny, tissue residence, migratory properties, and biological functions of these cells are areas of intense research focus given the broad spectrum of human disorders associated with aberrant T\textsubscript{H}17-type responses.

While T\textsubscript{H}17 cells are so named for their characteristic Interleukin 17 (IL-17) production, \textit{bona fide} T\textsubscript{H}17 cells of human or murine origin produce, at times, a cacophony of inflammatory mediators, which can include IL-17, IL-21, IL-22, and IL-26. As such, dissecting the biological consequences of robust T\textsubscript{H}17 responses, properly or improperly controlled, has presented a number of challenges. Further confounding the study of T\textsubscript{H}17 cells and individual cytokines are the many observations documenting non- T\textsubscript{H}17 cell sources of these same cytokines. Given the complexity of T\textsubscript{H}17 biology, we welcome the reports found within this issue, highlighting current findings and observations and illuminating several components of T\textsubscript{H}17 cells and known associated cytokines.

N. Qu et al. provide an interesting overview of the roles T\textsubscript{H}17 cells and their associated cytokines play in various inflammatory diseases. N. Y. A. Hemdan et al. build on this premise, addressing the T\textsubscript{H}17 cell contributions to autoimmunity, in particular that which arises following exposure to xenobiotic substances.

In the absence of overt chronic inflammation, T\textsubscript{H}17 cells predominantly reside at mucosal surfaces. H.-C. Tsai et al. provide an elegant update on the functions of T\textsubscript{H}17 differentiation and on the functional consequences of IL-17 signaling in pulmonary inflammation. Y. Morishima et al. highlight the role of T\textsubscript{H}17-associated cytokines in asthma, especially in steroid-resistant disease. Further interesting findings from the Hizawa laboratory suggest an important role for IL-17F in particular.

T\textsubscript{H}17 cells, which are known to be induced in response to a variety of bacterial and fungal infections, may also be selectively depleted, as in the early stages of an HIV infection. S. L. Bixler and J. J. Mattapallil elegantly discuss potential mechanisms by which T\textsubscript{H}17 cells are depleted or improperly regulated during HIV infection.

Further, T\textsubscript{H}17 cells and T\textsubscript{H}17-produced cytokines have also been associated with tumor immunity and conversely with promoting the initiation/progression of tumorigenesis. In this issue, D. Alizadeh et al. discuss how T\textsubscript{H}17 cells and T\textsubscript{H}17-associated cytokines may act directly or indirectly toward shifting local microenvironments to favor tumor promotion or tumor suppression. Focusing on AML, T. Tian et al. examined T\textsubscript{H}17 cell frequencies in acute myeloid leukemia patients and discuss their observed stage-dependent variation.

It is our hope that you will find the articles within insightful; we have enjoyed reading all of these articles immensely.

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