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Sommario/riassunto	CD4+ T lymphocytes play an essential role in host defense against bacterial, parasitic and viral infections. During infection, under the influence of intrinsic signals received through peptide-MHC/TCR interactions and extrinsic signals provided by pathogen-conditioned dendritic and other accessory cells, CD4+ T cells proliferate and differentiate into specialized T helper (Th) effectors, which produce distinct sets of cytokines tailored to combat a specific class of microbes. The concept of CD4+ T cell multi-functionality was developed after the seminal discovery of Th1 and Th2 cells nearly 30 years ago. Although the Th1/Th2 paradigm has successfully withstood the test of time, in the past decade additional Th subsets (Th17, Tfh, Th22, Th9) have been identified. Similarly, single cell analyses of cytokines and master transcriptional factors have revealed that, at the population level, CD4+ T cell responses are far more heterogeneous than initially anticipated. While some of the checkpoints in Th cell specification have been identified, recent studies of transcriptional and epigenetic regulation have uncovered a significant flexibility during the course CD4+ T lymphocyte polarization. In addition, Th cells

expressing cytokines with counteracting functions, as a measure of self-regulation, display yet another level of diversity. Understanding the mechanisms that control the balance between stability vs. plasticity of Th effectors both at the time of initiation of immune response and during development of CD4 T cell memory is critical for the rational design of better vaccines and new immunotherapeutic strategies. This research topic will cover current views on Th cell development, with a focus on the mechanisms that govern differentiation, function and regulation of effector Th cells in the context of microbial infections.