Resolution of inflammation is now considered an active process, which is accompanied by a switch in the profile of cytokines, lipid mediators, and other signaling molecules. In addition to the alteration in the profile of the involved mediators, resolution of inflammation is also linked to cellular plasticity, which allows the polarization of immune cells from a pro-inflammatory to an anti-inflammatory cell type. A well-established example is the polarization of monocytes to pro-inflammatory M1-like macrophages and anti-inflammatory M2-like cells. These changes in macrophage polarization are accompanied by a switch in the formed lipid mediators, partly due to the slight downregulation of 5-lipoxygenase (5-LO) and 5-LO activating protein (FLAP) and the prominent upregulation of 12/15-LO. A current paradigm is that changes in macrophage polarization is associated with a change in the released lipid mediator profile from pro-inflammatory prostaglandins and leukotrienes to so-called specialized pro-resolving lipid mediators (SPMs) such as lipoxins and resolvins. A hallmark of lipoxins and resolvins is that their biosynthesis from arachidonic acid (AA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA) requires the consecutive action of 5-LO and 12/15-LO.

Schebb et al. critically review the formation, signaling, and analytics of SPMs. Since 5-LO is mainly expressed in leukocytes, these cells are considered the main source of SPMs. The review article summarizes the biosynthetic pathways of SPMs in polymorphonuclear leukocytes (PMNL) as well as in M1 and M2 macrophages. It was concluded that human leukocytes only have a very limited biosynthetic capacity for trihydroxylated SPMs (lipoxins, RvE1, RvD1, RvD2, RvD3, RvD4). Therefore, the formation of most of
the role of T cells in the resolution of inflammation is reviewed by Hartel et al. with a special focus on sphingolipids. The authors summarize how sphingolipids can interfere with the differentiation and activation process of T cells. The authors highlight that a particular sphingolipid membrane composition seems to be required in the different subsets of T cells for individual signaling. The authors conclude that sphingolipids can modulate the T cell activities in diverse chronic inflammatory diseases that are driven by Th1 or Th2 cell responses or by an imbalance between Th17 and Tregs and that they are important for the resolution of inflammation.

Dalbeni et al. review the role of platelets in non-alcoholic steatohepatitis (NASH). The authors emphasize that the accumulation of platelets in the liver, platelet adhesion, and activation can prime immunoinflammatory reactions in the liver and the activation of stellate cells. Recent data suggest that antiplatelet drugs may interrupt this cascade, prevent/improve NASH, and improve some metabolic alterations. The pathophysiology of inflammatory liver disease and the role of platelets are discussed in this review.

Tang et al. summarize the therapeutic efficacy of resveratrol for acute lung injury. The authors conducted a meta-analysis based on 17 studies published from 2005 to 2017. It was concluded that resveratrol treatment seems to be effective in reducing the severity of acute lung injury but that more animal studies and clinical trials are required to elucidate the therapeutic benefit fully.

Author contributions

PP, BB, and DS wrote the editorial.

Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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