At the end of an immune response, apoptosis drastically reduces the numbers of activated T cells. It has been a matter of intense research how this form of apoptosis is regulated and initiated, and a number of proteins have been identified that contribute to this process. The present, widely accepted model assumes that the interplay of pro- and anti-apoptotic Bcl-2 family members determines the onset of activated T cell death, with the BH3-only protein Bim activating pro-apoptotic Bax/Bak. In the search for up-stream signals, factors from other immune cells have been shown to play a role, and the NFκB family member Bcl-3 has been implicated as a signalling-intermediate in T cells. Recent work has tested the interrelation of these factors and has suggested that Bcl-3 acts as a regulator of Bim activation, that the induction of apoptosis through Bim can be complemented by its relative Puma, and that the presence of certain cytokines during T cell activation delays the activation of Bim and Puma. Here we discuss these recent insights and provide a view on how the regulation of activated T cell death is achieved and how extrinsic signals may translate into the activation of the apoptotic pathway.