Insufficient Fas ligand (FasL)-mediated apoptosis of T cells might contribute to chronic inflammation

**Figure 6.** Insufficient Fas ligand (FasL)-mediated apoptosis of T cells might contribute to chronic inflammation. (a) Termination of an immune response by the induction of apoptosis in T cells, a process known as 'activation-induced cell death' (AICD) (Ref. 18). (b) Two mechanisms of impairment of T-cell apoptosis, such as might occur in inflammatory conditions (e.g. rheumatoid arthritis) (Ref. 75). Freshly isolated T cells from rheumatoid joints have been shown to express high levels of the anti-apoptotic Bcl-2 homologue, Bcl-xL, which may impair normal FasL-mediated T-cell turnover (Refs 75, 76). Secreted, soluble forms of both FasL (sFasL) and Fas (sFas) have been reported to occur at elevated levels in rheumatoid synovium (Refs 45, 46, 79). These soluble variants might act as receptor antagonists, precluding the interaction of functional cell-surface Fas and FasL, and consequently inhibiting apoptosis (Refs 78, 81) (fig006joc).