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Abstract The caspase 3 pathway initiates trigger- plex and mediates the synthesis of pro-apoptotic chal- lenged proteins in a variety of cell types. Activation of the caspase 3 pathway requires the activation of a specific apoptotic enzyme in the caspase-3 complex. Our in silico study demonstrated that the caspase 3 pathway was involved in triggering the hydrolysis of pro-apoptotic proteins in the presence of TNF-alpha and IL-1 [1]. The hydrolysis of pro-apoptotic proteins by TNF-alpha and IL-1 produced by the caspase 3 pathway was inhibited by IL-1-producing cells and the transiently activated cells were further maintained by the production of IL-1 by the stimulated cells. The results also suggested that the activity of the Caspase 3 pathway was not regulated only by the TNF- alpha and IL-1 but also by the TNF-alpha and IL-1-producing cells, especially those stimulated by the activated TNF- alpha and IL-1. In this study, we demonstrate that the caspase 3- pathway was involved in the hydrolysis of pro-apoptotic chondrogenic proteins in the presence of TNF-alpha and IL-1, and that the hydrolysis of pro-apoptotic proteins was mediated only by the TNF-alpha and IL-1-producing cells and not by the activated cells. We also show that this process was not blocked by the induction of the inactive, transiently activated cells and by the production of IL-1. TNF-alpha and IL-1 were able to elicit the hydrolysis of pro-apoptotic proteins in the presence of TNF-alpha and IL-1 and in the presence of IL-1. Recently, TNF-alpha and IL-1 were shown to induce pro- apoptotic proteins in the presence of TNF-alpha, IL-1, and IL-1, respectively, in a mouse model of human Tumour necrosis factor-alpha (TNF-alpha) infection. TNF- alpha and IL-1 are known to induce cell death in vivo and to induce apoptosis in vitro [5,6]. The effect of TNF-alpha and IL-1 on the activity of pro-apoptotic proteins in the presence of TNF-alpha and IL-1, is not known. Since the inhibitory activity of TNF-alpha and IL-1 was shown to be correlated with the induction of apoptosis by TNF-alpha and IL-1 [7,8], we further investigated whether these effects might be mediated through the TNF-alpha and IL-1. The results obtained with the caspase-3 pathway demonstrated that, despite the inhibition of the caspase 3 pathway, the expression of pro-apoptotic proteins was attenuated in the presence of TNF-alpha and IL-1. Using the fluorescence microscopy analysis system, we observed that the expression of TNF-alpha and IL-1 was enhanced by the transiently activated cells. The expression of IL-1 was strongly inhibited by the stimulation of the activated cells. This result suggests that the active intercellular interaction between the caspase 3 and the caspase-3 pathway might be involved in the hydrolysis of pro-apoptotic proteins. The transiently activated cells were also enhanced by the transiently activated cells. The present study provides a new insight into the mechanism by which the caspase 3 and the caspase-3 pathway are involved in the hydrolysis of pro-apoptotic proteins by TNF-alpha and IL-1. Introduction As this is the first study to directly demonstrate that the caspase-3 pathway is involved in the hydrolysis of pro-apoptotic proteins by TNF-alpha and IL-1, we were interested to further investigate whether the caspase 3 pathway is required for the hydrolysis of pro-apoptotic proteins by TNF-alpha and IL-1. TNF-alpha and IL-1 are known to induce apoptosis in mice by induc-

ing the death of neurons [9,10]. However, it has been reported that TNF-alpha and