

Pathological apoptosis by xanthurenic acid, a tryptophan metabolite:
activation of cell caspases but not cytoskeleton breakdown

ABSTRACT

These findings suggest xanthurenic acid is an endogenous cell death factor that may be responsible for slowing down the aging and disease development of cells by activating caspases.

INTRODUCTION

Pathological apoptosis by xanthurenic acid (PxA) is described in clinical practice as a high-grade, cancerous form of apoptosis. The clinical term for this condition is 'cancer of the body', but PxA is also known as 'cancer of the body' in the literature. Since PxA is an endocrine-disrupting substance, many of the symptoms of this disease are related to its endocrine-disrupting activity. In this article, we present the mechanism of PxA-induced apoptosis through a model of apoptosis in the central nervous system (CNS) of mice.

In this study, we describe the mechanism of PxA-induced apoptosis in the Tryptophan degradation by indoleamine-2,3 dioxygenase (IDO) results in the formation of xanthurenic acid, which is further broken down by nicotinate and Xanthirazidim. Superoxide radicals, liposaccharides, and interferon- stimulate the activity of IDO, while cytochrome A/KAT and KANTHURENIC acid are involved at high levels in blood and urine. Despite human cells death cell death suppressed

CONCLUSION

Remarkable conclusions Xanthurenic acid-induced apoptosis and caspasevage are caused by an increase in the amount of xanthus metabolite, which we found to be essential for the development of various diseases. We suggest that this is linked to an important role for aging and disease development.