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Downregulating the activity of kynurenine 3-monooxygenase inhibits the growth of canine mammary tumors

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Kynurenine 3-monooxygenase (KMO) is one of the enzymes involving in tryptophan metabolic pathway. The high level expression of KMO in canine mammary tumor and human hepatocellular carcinoma was found to relate to tumor malignancy and short survival time. KMO was previously found its downstream metabolites were excitotoxic to central nervous system (CNS) and may contribute to neurodegenerative diseases. Recently, KMO overexpression is found in some cancer patients; however, the role of KMO in tumor malignancy needs to be further dissected. Our study intends to investigate whether KMO is significant in the growth and deterioration of canine mammary tumors. Our results showed the proliferation and migration of canine mammary tumor (CMT) cells was reduced by KMO inhibitor. By inhibiting KMO activity, ERK1/2 and STAT3 signalings were turned down and suppressed cell growth of CMT cells. Also, AKT and FAK signal pathways were inactivated when treating cells with KMO inhibitor and resulting in fading migration ability. KMO may serve as a novel prognostic marker for cancers and targeting KMO may provide a promising strategy for tumor treatments. Our findings further reveal the significance of KMO in tumorigenesis.

Biography

Chen-Si Lin has completed her PhD from National Chiao-Tung University and Post-doctoral studies from National Taiwan University School of Veterinary Medicine. She is the Associate Professor of National Taiwan University School of Veterinary. She has published more than 20 papers in reputed journals and has been serving as a reviewer for many of them.

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