Potential Role for Spleen Tyrosine Kinase (SYK) in Breast Cancer

Nur Izzyan Bungsu¹, Prof Dr Abdalla Mohamed Jama², Dr Mas Rina Wati Hj Abd Hamid¹

¹ PAPRSB Institute of Health Sciences, Universiti Brunei Darussalam, Tungku Link Road, Bandar Seri Begawan, Brunei Darussalam BE1410
² Faculty of Science, Universiti Brunei Darussalam, Tungku Link Road, Bandar Seri Begawan, Brunei Darussalam BE1410

E-mail: rina.hamid@ubd.edu.bn

Keywords: breast cancer, drug induction, SYK, apoptosis, caspases

INTRODUCTION

Spleen Tyrosine Kinase (SYK) is a non-receptor tyrosine kinase produced mainly by hematopoietic cells reflecting their important role in B-cell progenitor development. While the roles of many tyrosine kinases in cancer cell development are well-documented, the ability of others like SYK to modulate the growth ability of cancer cells is less well-understood. SYK has been described to act as a tumour promoter or suppressor of cancer development depending on the stimulating environment. We were interested to study if SYK was involved in apoptosis in breast cancer cells.

METHODS

Stable transfection of the SYK gene into breast cancer cells was performed using FuGENE®. Stable cell line was induced to undergo cell death using staurosporine. LC50 of staurosporine was determined using Vybrant MTT cell proliferation assay. Apoptosis was investigated by qPCR and Western blotting to show gene and protein expressions of the cell cycle genes, caspases and inhibitors and promoters of apoptosis respectively.

RESULTS

Our results showed a substantial cell death of breast cancer cells carrying the SYK gene when induced with staurosporine. LC50 was determined to be 10µm. Cell cycle genes CDK6 and cyclin D mRNAs were reduced during cell growth and apoptosis was indicated and characterised by the expression of mRNAs for caspases 8 and 9 and the proteins Bax and Bcl-2 respectively.

CONCLUSIONS

SYK gene was stably-expressed in breast cancer cells. Upon staurosporine-induction, SYK appeared to cause a downregulation of the cell cycle genes and upregulation of apoptosis genes indicating a tumour suppression role in breast cancer development.