#### UNIVERSITI TEKNOLOGI MARA

# CISPLATIN TREATMENT AGAINST BREAST CANCER CELL LINES MCF-7, MDA-MB-231 AND MDA-MB-468

Thesis submitted in fulfilment of the requirements for the degree of Bachelor of Pharmacy (Hons.)

### **MOHAMMAD FARIZUL BIN SHAMSUDIN**

Faculty of Pharmacy, UiTM Puncak Alam Selangor

**July 2017** 

#### **ABSTRACT**

Cisplatin is a chemotherapy drug that is widely used as anticancer drug. Cisplatin increases cytotoxicity by interfering with DNA replication and transcription mechanism. Platinum compounds damage tumours via induction of apoptosis, which is mediated by the activation of various signal transduction pathways such as first inducing reactive oxygen species (ROS), interrupting DNA damage and disrupting calcium signalling in and out of cell. Breast cancer cell lines MCF-7, MDA-MB-231 and MDA-MB-468 were selected and cultured then treated with cisplatin to investigate whether different types of cancer cells give different effect. MTT assay is currently the most extensively used method for IC50 measurements. Here, we used MTT to determine the percentage of cell viability after treated with anticancer agent. The value of IC<sub>50</sub> of cisplatin for MCF-7 cell lines after 24 hours is 232.60μg/ml. MDA-MB-231 have the highest IC<sub>50</sub> value which 237.58μg/ml compared to both cell lines. The results obtained highlight the differences between the IC50 for each type of cells. We can conclude that cisplatin reduces cell viability and proliferation in breast cancer cells MCF-7, MDA-MB-231 and MDA-MB-468 cultured for 24 hours.

#### **ACKNOWLEDGEMENT**

I would like to thank to all those who gave me the opportunity to complete this report.

Special thanks to the final year project coordinator of Faculty of Pharmacy, Dr. Gurmeet

Kaur Singh who helps me to coordinate my project.

Special thanks went to my friend as well, Mohammad Azam Bin Rasidi and Muhamad Syafif Safwan Bin Sarudin who help me throughout the experiment.

My biggest thanks also goes to the head of the project and my supervisor, Dr. Normala Abd Latip, who has always find time for us to discuss about the project and guide the team in order to complete the project.

My appreciation also goes to my co supervisors as well, Dr Salfarina Ramli for providing the extract and also help us by giving us lot of tips for presentation.

Lastly, I would also like to acknowledge the staff of Pharmaco-toxicology Laboratory, who gave the permission to use all required equipment and the material in order to complete the research.

## TABLE OF CONTENT

ABSTR	ACT		.II
ACKNO	WLE	DGEMENT	Ш
TABLE OF CONTENTIV			
LIST OI	FIG	URES	VI
LIST OI	ABB	REVIATIONSV	/ <b>II</b>
CHAPT	ER 1:	INTRODUCTION	1
1.1		PROBLEM STATEMENT	2
1.2		OBJECTIVES	2
1.3		HYPOTHESIS	2
CHAPT	ER 2:	LITERATURE REVIEW	3
2.1		HALLMARK OF CANCER	3
2.2		BREAST CANCER	7
2.3		TREATMENT ON CANCER	9
CHAPT	ER 3:	METHODOLOGY	10
3.1		MATERIAL	10
	3.1.1	CELL CULTURES AND CULTURING MEDIUM	10
3.2		CELL CULTURES AND TREATMENT	10
	3.2.1	PROCEDURES	10
		3.2.1.1 CELL CULTURES	10
		3.2.1.2 TREATMENT WITH CISPLATIN	11
3.3		DETERMINATION OF IC50 BY USING MTT ASSAY	11
3.4		EXPERIMENTAL DESIGN	12
3.5		GANTT CHART	13
CHAPTER 4: RESULT15			
4.1		IN VITRO ANTICANCER ACTIVITY	15

#### **CHAPTER 1: INTRODUCTION**

Cancer cells are made up of trillions of cells and they can develop anywhere in our body. Human cells normally grow and divide to replace dead cells. For example, when cells are damaged, they will undergo apoptosis process, and will be replaced by new cells. But once cancer developed, these cells will survive when they should die, and new cells form when they are not needed. These extra cells can divide and grow until tumours are formed (What is cancer, 2015)<sup>1</sup>. There are six hallmarks of cancer that we will discuss further on this paper which are self-sufficiency in growth signal, antigrowth signal, evasion of apoptosis, continuous replication, sustained angiogenesis and metastasis. Additionally excessive adipose tissue expansion in obesity will cause dysfunction of adipose that can stimulate tumour growth (Antonia et al., 2016).

Cisplatin is a chemotherapy drug that is widely used as anticancer drug. Cisplatin increases cytotoxicity by interfering with DNA replication and transcription mechanism. Platinum compounds damage tumours via induction of apoptosis, which is mediated by the activation of various signal transduction pathways such as first inducing reactive oxygen species (ROS), interrupting DNA damage and disrupting calcium signalling in and out of cell (Florea & Büsselberg, 2011).

<sup>&</sup>lt;sup>1</sup> https://www.cancer.gov/about-cancer/understanding/what-is-cancer