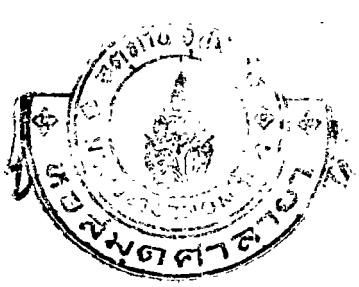

ORAL ACUTE AND SUBACUTE TOXICITY OF CYTOCHALASIN E IN MICE

BY

MISS BUSABAN OUNJANAM, B.Sc. (Pharm.)

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF

THE REQUIREMENTS OF THE DEGREE OF

MASTER OF SCIENCE

(TOXICOLOGY)

IN THE

FACULTY OF GRADUATE STUDIES

OF

MAHIDOL UNIVERSITY

1985

ORAL ACUTE AND SUBACUTE TOXICITY OF CYTOCHALASIN E IN MICE

BUSABAN OUNJANAM, B.Sc. (Pharm.)

Toxicology Program, Department of Physiology, Faculty of Science,
Mahidol University, Rama VI Road, Bangkok 10400

ABSTRACT

Oral acute and subacute toxicity of cytochalasin E was determined in Swiss Albino mice. With various routes of administration to one-day-old mice, the oral LD_{50} value was 3.30 mg/kg BW, and the LD_{50} values for i.p. and s.c. administrations were 2.21 and 2.00 mg/kg BW respectively. One-day-old mice given oral route of cytochalasin E developed gas bubbles in stomach and small intestine within 1 h after administration. Lethal oral dose of cytochalasin E killed one-day-old mice within 3 h after administration, gas bubbles were massively produced and the stomach was distended. Cyanosis and convulsions generally proceeded death.

One-day-old mice given multiple sublethal doses of cytochalasin E for 15 consecutive days developed gas bubbles in the stomach and intestine. The number of animals with the presence of gas bubbles as well as the number of dead animals were in dose related manner. Neonatal mice given multiple sublethal doses of cytochalasin E for 45 days gained less body weight than control. Percentage of liver weight to body weight was increased whereas percentage of testis weight was depressed. Histopathological changes included severe tubular nephrosis with vacuolar degeneration, congestion and fatty infiltration in liver, hydropic degeneration in gastric and intestinal epithelium and necrosis of cardiac muscle. These lesions were seen in early dead pups whereas enlargement of the liver cell nuclei was observed in liver of the survivors. Effect of

multiple sublethal doses of cytochalasin E on intestinal glucose absorption was studied in vitro by using intestinal segment from the survivors. The inhibitory effect was seen in high dose treated groups.