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PROGRAMME BOOK
ASSESSMENT OF IBUPROFEN, NON-SELECTIVE COX INHIBITOR, AS A NEUROPROTECTIVE AGENT IN ALZHEIMER’S MODEL OF RATS.
Exhibitor: Anil Kumar Saxena, Basic Medical Sciences, Kulliyyah of Medicine

ASSESSMENT OF CELECOXIB, A SELECTIVE COX-2 INHIBITOR, AS A NEUROPROTECTIVE AGENT IN ALZHEIMER’S MODEL OF RATS.
Exhibitor: Anil Kumar Saxena, Basic Medical Sciences, Kulliyyah of Medicine

KNOWLEDGE, ATTITUDE AND PRACTICE (KAP) OF ASTHMA FOOD TABOOS AMONG ASTHMATIC PATIENT
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CROSS-SECTIONAL DESCRIPTIVE STUDY OF MANAGEMENT MODALITIES, CHALLENGING IN DECISION MAKING AND (QUALITY OF LIFE) OF SURGICAL PATIENTS IN PALLIATIVE CARE UNIT
Exhibitor: Tan Natong Soe, Surgery, Kulliyyah of Medicine

STUDIES IN WORLD RELIGIONS: AN INTRODUCTION
Exhibitor: Ahmad Yousif, ISTAC, International Institute of Islamic Thought and Civilization
ASSESSMENT OF CELECOXIB, A SELECTIVE COX-2 INHIBITOR, AS A NEUROPROTECTIVE AGENT IN ALZHEIMER’S MODEL OF RATS.

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Reduced cerebral blood flow (CBF) has been associated with neurodegenerative disorders including Alzheimer’s disease. Experimentally, a condition of chronic cerebral hypoperfusion due to reduced CBF can be induced by permanent bilateral occlusion of common carotid arteries (2-vessel occlusion, 2VO) in rats. Neuroinflammation has been suggested to play a crucial role in the development and progression of many neurodegenerative diseases. The neuroinflammatory response induces activation of microglia and release of inflammatory mediators such as prostaglandins, leukotrienes, and cytokines. The sustained release of inflammatory mediators works to perpetuate the inflammatory cycle and leads to apoptosis and neuronal cell death. Since neuroinflammation, leading to neuronal apoptosis and death, is one of the mechanisms which is thought to play a significant role in chronic degenerative neurological disorders like Alzheimer’s disease, the present study was planned to assess the neuroprotective role of celecoxib, a selective COX-2 inhibitor, in Alzheimer’s model of rats (2VO). After one week of acclimatization, fifteen Sprague Dawley rats weighing 200-250 g were equally divided into three groups. Group A served as – sham control, Group B – 2VO, and Group C – 2VO-C (treated daily with celecoxib 50 mg/kg, orally following 2VO). On 8th week, all the rats were euthanized and the hippocampi were isolated. Viable neuronal cells in the hippocampal CA-1 region were counted and hippocampal COX-2 mRNA expression and prostaglandin E2 (PGE-2) levels were estimated. There was a significant difference in neuronal cell death, increase in COX-2 mRNA expression and PGE-2 levels in 2VO group as compared to sham control group. In celecoxib-treated 2VO (2VO-C) rats, the viable neuronal cell count of the hippocampal CA-1 region was significantly higher as compared to the untreated 2VO group. The hippocampal COX-2 mRNA expression and hippocampal PGE-2 levels were found to be significantly lower in the celecoxib-treated 2VO rats as compared to untreated 2VO rats. The results clearly point out that celecoxib is an effective neuroprotective agent in Alzheimer’s model of rats and can be successfully used in the management of Alzheimer’s disease.

Keywords: Celecoxib, neuroinflammation, neurodegeneration, Alzheimer’s disease, 2VO rats.