

## MEDICAL PHILATELY

## Julius Axelrod &amp; Neurochemistry

Jayant Pai-Dhungat



**Neuron cells and chemical formula of dopamine. World neurochemistry conference Venezuela, 1987**

**J**ulius Axelrod (1912-2004), an American biochemist and pharmacologist, was born in New York City in 1912. Graduate of the College of New York City (1933), he worked briefly as a laboratory technician at New York University. In 1935 he got a job with New York City Department of Health for testing vitamin supplements added to food (1935-45). He lost his left eye in a lab accident, and wore an eye patch for the rest of his life. While working at the department he attended night school, and received his master's degree.

Axelrod joined research division at Goldwater Memorial Hospital (1946). He began his research on metabolism of analgesic medications, and began his studies on sympathetic amines. But, Julius came to realize that his research contributions would not be fully recognized unless he had a doctorate. It was at this stage that he took leave of absence from NIH (1954) to study in the Department of Pharmacology of George Washington University for his PhD, which he thought was vital for his further recognition. At the Washington University he was allowed to submit some of his earlier research towards



**Julius Axelrod Nobel Prize, 1970. Nerve endings with release and reuptake of neurotransmitters. Stamp-Sweden, 1984**

his degree. Hence he graduated within a year in 1955. He returned to NIH and began some key research on nervous system and its main neurotransmitters-adrenalin and noradrenalin. Axelrod initially worked on mechanism and effect of caffeine. Working with Bernard Brodie, their research focused on non-aspirin analgesics. This analgesic caused methemoglobinemia in some individuals. The duo discovered acetanilide as the main culprit in the formulation and further, that one of the metabolite acetaminophen (paracetamol) was more effective and safer analgesic. During this time he also conducted research on codeine, morphine, methyl-amphetamine, ephedrine and performed first experiments on LSD-25.

Axelrod's Nobel Prize winning research grew out of work done by Euler, especially Euler's discovery of noradrenalin that transmits nerve impulses. Axelrod in turn discovered neuro-transmitter's reuptake and storage by pre-synaptic nerve endings-research carried out by Euler and Hillarp earlier, unknown to Axelrod. His Key discovery for winning Nobel Prize was that noradrenalin could

be neutralized when not needed, by an enzyme- catechol-O- methyl transferase, which he isolated and named in 1958. The enzyme proved critical in the understanding of the entire nervous system. It was shown to be useful in dealing with the effect of certain psychotropic drugs and in research on hypertension and schizophrenia.

Re-uptake and storage of neurotransmitters laid the ground work for later selective serotonin reuptake inhibitors (SSRIs), such as fluoxetine, which blocks the reuptake of serotonin. Axelrod also worked on dopamine, monoamine oxidase (MAO) inhibitors in 1957, which are used as antidepressants. Axelrod and Solomon Snyder elucidated and concluded that antidepressants work in a variety of ways. ((1964).

Some of Axelrod's later research focused on the pineal gland. He along with colleagues obtained hormone melatonin which is generated from tryptophan. Rate of synthesis and release follow body's circadian rhythm driven by suprachiasmatic nucleus within hypothalamus. They went on to show that melatonin has wide ranging effects throughout CNS, allowing the pineal gland to function as a biological clock.

Axelrod was awarded the Nobel Prize in Physiology or Medicine in 1970, together with Von Euler and Bernard Katz "for their discoveries concerning humoral transmitters in the nerve terminals and mechanism for their storage, release and inactivation)"

Better  
performers  
have

# Volibo<sup>®</sup>

(Meprobamate, 250 mg/5ml syrup)



for **α GI** therapy

# Volibo<sup>M</sup>

(Meprobamate, 250 mg/5ml syrup)

Does  
not  
affect  
GI  
tract



Preserves  
β-cell  
function

