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Organised by Stylianopoulou F., President of the Organizing Committee on behalf of the HSfN and Frackowiak R., President of the EBBS

Abstract:

The effects of scopolamine on visual categorization in macaques

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Learning requires the ability to adapt to new situations and to respond to new categories of stimuli. This function is crucial in children and in adults and malfunction can lead to a debilitating disorder known as Alzheimer's disease or senile dementia. Acetylcholine (ACh) is one of several neuromodulators implicated in the brain's adaptive behavior. It has an important role in several cognitive functions including attention, learning, short-term memory and long-term memory. The Nucleus Basalis of Meynert supplies the cholinergic input to the neocortex and shows marked cell loss in Alzheimer's patients and in other cognitive disorders (Wernicke-Korsakoff syndrome and Creutzfeldt-Jakob disease).

We have examined the effects of scopolamine, an antagonist of muscarinic ACh receptors, on visual object recognition in macaques. Two macaques were taught a categorization task, i.e. to classify stimuli to categories by appropriate behavioral responses. The paradigm involved a task during which an image that belonged to one of the categories was presented. The macaque used levers to categorize the stimulus.

Injections of scopolamine disrupted subsequent performance in this task. When the stimuli presented had not been seen before the experiment, scopolamine significantly impaired categorization accuracy. The monkey was still able to carry out the task with a set of familiar stimuli, i.e. stimuli that it had categorized successfully in previous sessions. Performance deteriorated as the stimulus became less salient by an increase in the level of visual noise. Scopolamine, however, had at best a small effect on performance with familiar stimuli at the different noise levels.

ACh has a variety of effects, peripheral as well as central. In our attempt to localize the effects of ACh we have used an analogue of scopolamine that cannot cross the blood brain barrier. The analogue (butyl scopolamine) mimicked the peripheral actions of scopolamine but caused no cognitive deficit. The cognitive changes, therefore, reflect ACh's effect in the brain. The exact site of the effect has not yet been established but is likely to depend on forebrain mechanisms.