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The Council for Tobacco Research - U.S.A. Grant, "Studies of Nicotine Action (Upon Memory Consolidation."

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PROGRESS REPORT

January 16, 1973 to August 1, 1974.

STUDIES OF NICOTINE ACTION UPON MEMORY CONSOLDIATION

Within the past 17 months, or since the submission of a previous progress report for this research project (January 16, 1973 - Aug. 1, 1974), the research has been specifically concerned with the interaction of putative neurotransmitters and the central effects of nicotine and several of its biologically active metabolites. Changes in brain serotonin (5-hydroxytryptamine) metabolism have, in previous research, been established in conjunction with several ammesic agents and/or events, several of which appear to be temporally contiguous with the ammesic associated inhibition of cerebral protein synthesis. Such effects, particularly as brought about experimentally through the administration of electroconvulsive shock (ECS) have been antagonized or blocked through the administration, prior to administration of the ammesia stimulus of nicotine. These effects have been documented in previous publications from your laboratory. We have also demonstrated that several metabolites of nicotine, namely cotinine and 3-pyridylacetic acid, are capable of antagonizing the amnesic effects brought about by post-training ECS and similarly alter the effects of such ECS upon brain 5-HT metabolites. These effects have also been shown to be time-dependent to the extent that 45 minutes appears to be optimal following nicotine injection, after which the amnesic properties of ECS are antagonized, whereas 15 minutes after treatment, the effects appear to be potentiated. In Figure 1, these behavioral data have been summarized for nicotine and several of its derivatives.