

The separate and combined effects of scopolamine and nicotine on human information processing

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Abstract. Previous work in this and another laboratory has shown that nicotine tablets improve the performance of a rapid information processing task and reduce the Stroop effect, whereas scopolamine has the opposite effects. The purpose of this study was to extend these previous findings by determining whether, when administered together, these two drugs have mutually antagonistic effects on task performance. Two experiments are reported, both using within-subjects double-blind Latin Square designs. In the first, six subjects received single and combined doses of scopolamine 1.2 mg and nicotine 1.5 mg, and there was some evidence that the two drugs had mutually antagonistic effects on the rapid information processing task. In the second experiment 12 subjects received the same doses, but rapid information processing testing was carried out over a longer time period and Stroop testing was introduced at the end of the 2.5 h session. Nicotine was found to counteract the depression of performance produced by scopolamine on both the rapid information task and the Stroop test. These results provide further support for the theory that central cholinergic pathways play a major role in human information processing.

Key words: Nicotine – Scopolamine – Rapid information processing task – Stroop test – Cholinergic pathways

In animals, it has been demonstrated that the ascending cholinergic reticular pathways play a major role in the selection of environmental stimuli, while having little involvement in response control (see review in Warburton 1977). The evidence for this has been provided by studies in which compounds such as physostigmine, that enhance cholinergic activity, improved stimulus detection performance in vigilance-like situations (Warburton and Brown 1972), while cholinolytics, such as scopolamine, disrupted stimulus detection performance (Brown and Warburton 1971). Warburton (1975, 1981) has proposed that the behavioural changes produced by cholinergic drugs are the result of the action of these drugs on those cholinergic pathways ascending from the ventral tegmental area to the cortex which control electrocortical arousal.

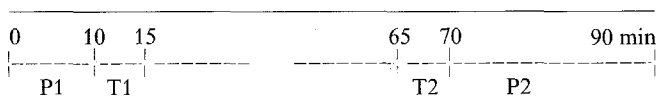
We have tested this proposal in man by studying the effects of the nicotine and scopolamine on tests of mental efficiency. Nicotine is a cholinergic agonist which increases electrocortical arousal by acting at the midbrain reticular

formation (Armitage and Hall 1967; Il'yutchenok and Ostrovskaya 1962; Kawamura and Domino 1969). Scopolamine, on the other hand, is a muscarinic cholinergic antagonist (Domino and Corssen 1967) and decreases electrocortical arousal (Ostfeld and Aruguette 1962). The predictions from Warburton's (1975, 1981) theory that we have tested are that as a result of their opposite effects on central cholinergic activity, nicotine will improve stimulus selection and thus improve performance on tasks requiring sustained concentration, whereas scopolamine will impair stimulus selection, and thus worsen performance on such tasks.

Our findings to date have been consistent with these predictions. In one series of studies cigarette smoking prevented the decline in stimulus sensitivity which occurred over time in nicotine-free conditions during both auditory and visual vigilance tasks (Wesnes and Warburton 1978). Nicotine tablets had a similar effect on stimulus sensitivity in a visual vigilance task (Wesnes et al. 1983), whereas scopolamine lowered stimulus sensitivity in this task (Wesnes and Warburton 1983a). Other workers have found scopolamine to increase the Stroop effect (Ostfeld and Aruguette 1962), which can be considered to be a measure of distractibility, whereas we have found that nicotine tablets decrease this effect (Wesnes and Warburton 1978). In a further series of studies, cigarette smoking increased both the speed and accuracy of the performance of a rapid visual information processing task (Wesnes and Warburton 1983b, 1984a), and there is evidence that this effect is accompanied by an increase in electrocortical arousal (Warburton and Wesnes 1979). Furthermore, in one experiment, 1.5 mg nicotine tablets had similar effects to smoking on the performance of this task, whereas in another experiment scopolamine 1.2 mg produced a marked decrease in accuracy on the task (Wesnes and Warburton 1984b). The experiments reported here are a direct extension of our previous studies, and were designed to determine whether nicotine 1.5 mg and scopolamine 1.2 mg when administered to the same subjects in the same experimental session would have mutually antagonistic effects on the performance of this task as well as the Stroop effect.

Experiment 1

The purpose of this experiment was to determine the effects of both single and combined oral administration of scopolamine 1.2 mg and nicotine 1.5 mg on the efficiency of performance of a rapid visual information processing task

Table 1. Design and dose regimen of Experiment 1

Where:

P1 represents first 10 min baseline performance period

P2 represents 20 min post-drug performance period

T1 represents taking 1.2 mg scopolamine tablet or placebo

T2 represents taking 1.5 mg nicotine tablet or placebo

Four dose conditions:

T1	T2
1. placebo	placebo
2. placebo	nicotine 1.5 mg
3. scopolamine 1.2 mg	placebo
4. scopolamine 1.2 mg	nicotine 1.5 mg

which has previously been used in this laboratory to study the effects of cigarette smoking (Wesnes and Warburton 1983a, 1984a), nicotine tablets and scopolamine (Wesnes and Warburton 1983a, 1984b).

Method

Subjects. Six male and six female undergraduate non-smokers aged between 18 and 21 took part in this study as paid volunteers.

Scopolamine and nicotine tablets. The tablets were prepared by adding measured amounts of the two drugs to quarter pieces of dextrose tablets using a microlitre syringe. To make the nicotine and scopolamine tablets indistinguishable from each other in terms of taste and appearance, a drop of tabasco sauce was added to each. The placebos were prepared by simply adding tabasco to the pieces of dextrose tablets. We have found this technique sufficient to prevent subjects from identifying the verum on the basis of either taste or appearance (Wesnes and Warburton 1978, 1984; Wesnes et al. 1983).

Test. The task was identical to that used in previous experiments (Wesnes and Warburton 1983a, 1984a, b). A series of digits was presented on a visual display unit at the rate of 100 digits per min, and subjects were instructed to press a response button as quickly as possible when they detected sequences of three consecutive odd or three consecutive even digits. On average, 80 of these sequences were presented every 10 min. Any two sequences were separated by a minimum of 5 and a maximum of 30 digits. After the onset of the 3rd digit of an experimental target, 1,500 ms was allowed for a correct response to be made; responses occurring at any other time being counted as errors. Three measures of performance were made during each 10 min of the test: the probability of correctly detecting an experimental target (probability of a hit = total number of correctly detected targets/number of targets presented), the average time taken to respond to an experimental target, and the number of responses made in error.

Apparatus. The task was controlled by a PDP-12 mini-computer which generated the digits and recorded all responses and response times. Three visual display units were housed

in a darkened room and were separated by screens which prevented subjects being in visual contact. The response buttons were silent and were interfaced together with the screens to the computer. While performing the task, subjects wore headphones through which low intensity white noise was played to mask extraneous sounds.

Design and procedure. Four different dose regimens were used which are presented in Table 1 together with the design of each session. A double-blind repeated measures design was employed so that each subject received each of the four experimental dose regimen, the order of administration over sessions being counter-balanced using a 4×4 Latin Square Design. Prior to the study each subject visited the laboratory for an introductory session, was informed about the general details of the experiment and given 20 min training on the task. The subjects came to the laboratory on a subsequent day for a second 20 min training session. Then they attended four experimental sessions which all began at either 9.00 or 10.30 a.m. and lasted for 90 min. They were requested not to drink alcohol, tea or coffee from 11.00 p.m. onwards on the evenings preceding the experimental days.

On arrival at the laboratory, the subjects were asked if they had complied with the experimental instructions and, if they had, they proceeded to the experimental room where they performed the task for a 10 min period (P1 in Table 1). Immediately after, they were given either placebo or scopolamine 1.2 mg, according to the condition, and were asked to hold the tablet in their mouths for a 5 min period before swallowing (T1 in Table 1). They were then allowed to go to a common room for 50 min and to rest or read material of their own choice. Then they returned to the laboratory and were given either nicotine 1.5 mg or placebo, and again held the tablets in their mouths for 5 min before swallowing (T2 in Table 1). After swallowing the remains of the second tablet, they left their wrist watches with the experimenter, entered the experimental room and performed the task for a continuous 20 min period (P2 in Table 1).

Results

A fault on one of the response buttons was not discovered until the experiment had been completed, (it transpired that the switching mechanism intermittently failed to make contact), with the result that a proportion of the responses were not recorded. It was not possible, retrospectively, to determine at what stage of the experiment the fault had developed, and thus to avoid the complications involved in the unsatisfactory procedure of estimating missing data points, only the data from the six subjects who did not use the faulty response button were analysed.

As in the previous experiments, the first 10 min of performance prior to drug administration was taken as a baseline and subtracted from the performance scores in the two successive 10 min periods of the post-drug testing period to give difference from baseline measures.

The difference from baseline hit probabilities are presented in Table 2. Analyses of variance were performed for each 10 min period. In the first 10 min the between condition differences were not significant [$F(3,15) = 2.96, P < 0.1$], but in the second 10 min they were [$F(3,15) = 3.53, P < 0.05$]. Newman-Keuls testing revealed that there was a significant difference between the condition where they received

Table 2

The difference from baseline hit probability, reaction times and errors during the two successive 10 min periods on the rapid visual information processing task in Experiment 1

Difference from baseline	Placebo	Nicotine 1.5 mg	Scopolamine 1.2 mg	Nicotine 1.5 mg and scopolamine 1.2 mg
Hit probability:				
0–10 min	– 0.011	0.065	–0.072	– 0.045
10–20 min	– 0.070	0.063	–0.193	– 0.098
Reaction time (ms):				
0–10 min	–19.5	–16.7	–5.2	–19.0
10–20 min	1.2	–15.3	9.8	1.0
Errors:				
0–10 min	2.0	– 1.5	1.8	1.2
10–20 min	4.2	2	1.7	2.0

nicotine 1.5 mg but no scopolamine and that where they received scopolamine 1.2 mg but no nicotine.

The changes over time in each condition were analysed, but only the decrement with scopolamine reached significance [$F(2,10) = 5.53, P < 0.05$]. There were very few errors made and much variability in both errors and reaction times. There were no significant between condition differences in either of these measures.

Experiment 2

Despite the equipment failure in the previous study, there was some indication that nicotine and scopolamine have mutually antagonistic effects, which warranted further investigation. In addition to the rapid information processing test the Stroop test was administered as a test of selective information processing under conditions of distraction. In a review of the effects of scopolamine in humans, Safer and Allen (1971) concluded that the central effects of scopolamine occur between 90 and 150 min after administration. In order to determine whether this was true for the effects of scopolamine on rapid information processing, a second testing period was introduced in this experiment 140 min after the administration of scopolamine.

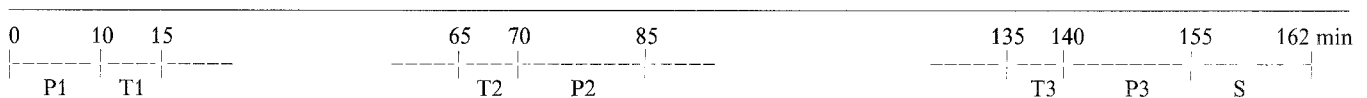
Method

Subjects. Six male and six female undergraduate non-smokers aged between 18 and 21 took part in this study as paid volunteers.

Scopolamine and nicotine tablets. The same strengths of nicotine and scopolamine tablets were prepared in an identical fashion to those in Experiment 1.

Rapid visual information processing test. The same task was used as in Experiment 1. The task was controlled by a PDP-12 mini-computer which generated the digits and recorded all responses and response times. Four visual display units were housed in separate windowless rooms which all had extractor fans to mask any extraneous noises. The response buttons were silent and were interfaced together with the screens to the computer.

The Stroop test. The Stroop test was the same version as that used previously to study the effects of nicotine tablets (Wesnes and Warburton 1978). The stimuli were presented on 50 × 60 cm cards, each card having 20 rows of ten stimuli each side. On one side the stimuli were oval patches of the colours brown, red, green, black and blue. The order of the patches was randomised with the constraints that the same

Table 3. Design and dose regimen of Experiment 2

Where:

P1 represents first 10 min baseline performance period

P2 represents first 15 min post-drug performance period

P3 represents second 15 min post-drug performance period of the rapid information processing task

T1 represents administration of 1.2 mg scopolamine tablet or placebo

T2 represents administration of 1.5 mg nicotine tablet or placebo

T3 represents administration of 1.5 mg nicotine tablet or placebo

S represents Stroop testing

Four dose conditions:

	T1	T2	T3
1.	placebo	placebo	placebo
2.	placebo	nicotine 1.5 mg	nicotine 1.5 mg
3.	scopolamine 1.2 mg	placebo	placebo
4.	scopolamine 1.2 mg	nicotine 1.5 mg	nicotine 1.5 mg

colour never appeared consecutively and each colour appeared 40 times in total. The stimulation the other side were the words "brown", "red", "green", "black" and "blue", printed in the same five colours of ink. The orders of the words and the ink colour were randomised with the constraints that the ink colour and the word were never matched, no two consecutive stimuli used the same colour or word, and each word and colour appeared 40 times. The subjects were instructed to name the colours in which the stimuli were printed, starting with the words, as quickly and as accurately as possible, and this was recorded for subsequent analysis. The Stroop effect was derived by subtracting the time taken to name the colour patches from the time taken to name the colour of the ink of the colour names to give a measure of the distraction by the semantics of the words. The same calculation was made for the number of errors.

Design and procedure. Four different dose regimens were used and these are presented in Table 3 together with the design of each session. A double-blind, repeated measures design was employed so that each subject received each of the four experimental dose regimens, the order of administration over sessions being counter-balanced using a 4 × 4 Latin Square Design.

Prior to the study each subject visited the laboratory for an introductory session in which they were informed about the general details of the experiment and given 20 min training on the rapid visual information processing task followed by training on the Stroop test. The training of the Stroop test consisted of naming the colours of the inks of the words on one side of a card and then the colours of the patches on one side of another card. The subjects came to the laboratory on a subsequent day and underwent identical training on the two tests. Then they attended four experimental sessions which all began between 2.00 and 3.00 p.m. and lasted for approximately 165 min. They were requested not to drink

alcohol, tea or coffee prior to attending on the experimental days.

On arrival at the laboratory, the subjects were asked if they had complied with the experimental instructions and, if they had, they performed the rapid information processing task for a 10 min period (P1 in Table 3). Immediately after, they were given either placebo or scopolamine 1.2 mg, according to the condition, and were asked to hold the tablet in their mouths for a 5 min period before swallowing (T1 in Table 3). They were then allowed to go to the common room for 50 min and to rest or read material of their own choice. After their return to the laboratory they were given either nicotine 1.5 mg or placebo, and again held the tablets in their mouths for 5 min before swallowing (T2 in Table 3). After swallowing the remains of the second tablet, they performed the task for a continuous 15 min period (T2 in Table 3) before going back to the common room for a further 50 min. They then returned to the laboratory for a third time and were given either nicotine 1.5 mg or placebo, again holding the tablet in their mouths for 5 min (T3 in Table 3) before performing the rapid information processing task for a second continuous 15 min period (P3 in Table 3). Immediately after they performed the Stroop test (S in Table 3).

Results

Rapid information processing test. The same three measures of performance were calculated for each time period as in Experiment 1: the probability of a hit, average reaction time and number of errors. The first 10 min of performance prior to drug administration was taken as a baseline and subtracted from the performance scores in the two successive 10 min periods of the post-drug testing period to give difference from baseline measures.

Table 4

The difference from baseline hit probability, reaction times and errors in the rapid visual information processing task in Experiment 2

Difference from baseline	Placebo	Nicotine 1.5 mg	Scopolamine 1.2 mg	Nicotine 1.5 mg and scopolamine 1.2 mg
Hit probability				
70–85 min	– 0.009	– 0.019	– 0.109	0.007
140–155 min	0.031	0.013	– 0.105	– 0.021
Reaction time (ms)				
70–85 min	– 7.2	– 1.94	29.15	– 8.98
140–155 min	– 10.13	– 23.28	29.7	– 3.45
Errors				
70–85 min	0.42	1.04	0.42	0.75
140–155 min	0.09	0.90	0.22	0.47

Table 5. Times (seconds) and number of errors made under the two conditions of Stroop testing in Experiment 2

	Placebo		Nicotine 1.5 mg		Nicotine 1.5 mg and scopolamine 1.2 mg		Scopolamine 1.2 mg	
	Time	Errors	Time	Errors	Time	Errors	Time	Errors
Patches	134.7	4.3	133.6	4.9	130.6	5.1	134.6	5.5
Words	172.2	4.1	176.8	4.4	171.4	4.8	185.8	5.0

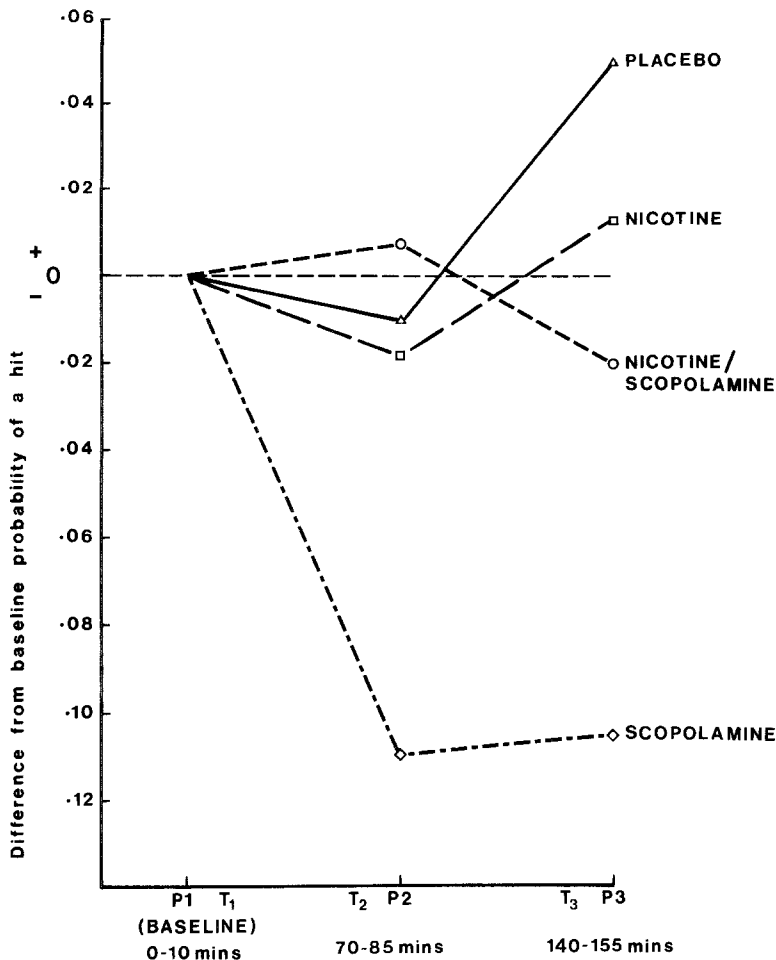


Fig. 1. The difference from baseline hit probabilities on the rapid information processing task under the four conditions of Experiment 2. See Table 3 for description of conditions

The difference from baseline hit probabilities are presented in Fig. 1. A decrease in the probability of a hit relative to the baseline pre-drug performance period is represented by a negative value. Analyses of variance of the difference from baseline scores revealed significant drug effects in both the first [$F(3,24) = 4.48, P = 0.012$] and the second test periods [$F(3,24) = 6.34, P = 0.0025$]. Newman-Keuls testing revealed that the scores in the scopolamine condition were significantly different to those in the placebo, nicotine and nicotine plus scopolamine conditions in both the first ($P < 0.05$) and the second time period ($P < 0.01, 0.01$ and 0.05 respectively). No other comparisons reached significance. The mean baseline hit probability was 0.607, and thus following scopolamine 1.2 mg, performance fell to 82% and 82.7% of the baseline value in the two post-drug testing periods, which compares well with the previous study (Wesnes and Warburton 1984b) in which hit probability fell to 82% of the baseline value following scopolamine 1.2 mg.

Analysis of the changes in hit probability over the three performance periods indicated that the decline following scopolamine was significant [$F(2,22) = 8.95, P < 0.01$], whereas the changes over time in the other three conditions did not approach significance.

The differences from baseline reaction times are presented in Table 4 and they follow a similar pattern to that

for hits. However, as there was more variability in the data, there was only a trend towards a significant treatment effect in the second time period [$F(3,24) = 2.38, P = 0.095$]. As has been found previously (Wesnes and Warburton 1983b, 1984a, b), error responding was very low, averaging less than one per subject per time period, and there were no between condition differences.

Stroop testing. The group mean times for both the patches and the words together with the number of errors are presented in Table 5. There were no between condition differences for the time taken to name the colour patches [$F(3,24) < 1$], but there was a significant between condition difference in the time taken to identify the colours in which the words were printed [$F(2,24) = 5.97, P < 0.01$]. Newman-Keuls testing revealed that the subjects were significantly slower in the scopolamine condition than in the placebo ($P < 0.01$), nicotine ($P < 0.05$) or nicotine plus scopolamine ($P < 0.01$) conditions. The slight increase in errors in the scopolamine condition was not significant.

Discussion

In Experiment 2 scopolamine 1.2 mg produced a decrement in correct detections on the rapid visual information process-

ing task, the magnitude of which was directly comparable to that found in a previous study (Wesnes and Warburton 1984b). Scopolamine also disrupted performance on the Stroop test, confirming a previous finding (Ostfeld and Aruguette 1962). When nicotine 1.5 mg was administered prior to testing, the effects of scopolamine were markedly reduced, both on the rapid information processing task and the Stroop test. These findings indicate that nicotine is able to counteract the deleterious effects of scopolamine on tasks requiring sustained attention.

While in Experiment 1 there was some suggestion that nicotine alone may improve performance, this was not evident in Experiment 2, and therefore it is not possible to conclude from these data that scopolamine 1.2 mg antagonises the effects of nicotine 1.5 mg. The absence of an effect of nicotine alone in this study deserves some consideration, as nicotine tablets have previously been found to produce improvements on both the rapid visual information processing task (Wesnes and Warburton 1984b) and the Stroop test (Wesnes and Warburton 1978). There were, however, some methodological differences between this and the earlier studies. Firstly, in previous studies of the effects of cigarette smoking and nicotine tablets on the performance of the rapid information processing task (Wesnes and Warburton 1983a, 1984a, b), there was a gap of only 10 min between the baseline testing period and the post-smoking/drug testing periods. However, in the previous experiment on the effects of scopolamine (Wesnes and Warburton 1984b), as in the present study, it was necessary to have a gap of 60 min between the baseline testing and the post-drug testing due to the slower absorption of scopolamine. In the previous smoking and nicotine studies, in the control conditions there was a decrement in efficiency compared to baseline after 10 min rest period, indicating that this period of rest was not sufficient to restore performance to its original level. However, in the previous study with scopolamine and in Experiment 2, the 60 min rest period following baseline was sufficient to restore performance to the baseline level of efficiency in the placebo conditions. Therefore, it is possible that, with oral doses of nicotine 1.5 mg, performance will only be affected if it is depressed by other factors such as fatigue. This is certainly consistent with a previous finding that nicotine tablets reduced the decrement in efficiency which occurred over time in a visual vigilance task (Wesnes et al. 1983). Similarly, in the previous study using the Stroop procedure, the subjects performed the test twice following nicotine administration (Wesnes and Warburton 1978). When the performance on the two successive Stroop tests was analysed separately (Wesnes 1979) it was found that nicotine only reduced the Stroop effect on the second test. This again suggests that oral doses of nicotine in this range only facilitate performance under conditions of task-specific fatigue. These explanations are entirely consistent with the findings of Experiment 2 that nicotine can counteract the decrements in performance produced by scopolamine on both the rapid information processing task and the Stroop task.

It was evident that the decrease in efficiency on the rapid information processing task produced by scopolamine 1.2 mg was no greater during the second post-drug testing period than during the first. This would suggest that the central effects of orally administered scopolamine occur earlier than the estimate of Safer and Allen (1971) of 90 and 150 min following administration. Thus in this and previous

experiments (Wesnes and Warburton 1983a, 1984b), central effects of scopolamine can be detected within 70 and 80 min following administration, and from Experiment 2 it would appear that they are no greater at 140–155 min following administration.

The findings of this study provide further support for the theory that cholinergic pathways are involved in the early stages of information processing. In both experiments, the scopolamine-induced cholinergic blockade disrupted performance, confirming previous findings (Wesnes and Warburton 1983a, 1984b). Furthermore, in Experiment 2, this disruption of performance was counteracted by nicotine 1.5 mg, showing that a drug which has the opposite effect on cholinergic function can counteract the resulting decrements in information processing. It was not possible to conclude that scopolamine antagonised the actions of nicotine from this study, and further experimentation is necessary to investigate this possibility.

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