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Type A Behavior Pattern Today: Relevance of the JAS-S Factor to Predict Heart Rate Reactivity

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The authors used 89 undergraduate students' scores in the S-factor of the Jenkins Activity Survey, a measure of speed and impatience, to classify 45 participants as high scorers and 44 as low scorers. They then measured the students' tonic and phasic heart rates during an examination, a genuinely stressful situation. The experiment consisted of three phases: adaptation, task, and recovery. The findings confirmed the authors' hypothesis that the high-S scorers would show higher cardiac reactivity values than the low-S scorers. The authors also observed that the high-S scorers took more time than the low-S scorers to recover their initial heart rate values after being exposed to the stress situation. This finding led the authors to suggest that each group may have different response patterns. They call for further research on individuals with "fast activation–fast recovery" and "fast activation–slow recovery" profiles.

Index Terms: *cardiac reactivity, fast activation–slow recovery profile, Jenkins Activity Scale S-factor*

The generally accepted description of the Type A behavior pattern (TABP) is that postulated by Friedman and Rosenman,[1] who describe TABP as encompassing the following characteristics:

1. Physical components: loud voice, quick speech, psychomotor activity, facial muscle tension
2. Attitudes and emotions: hostility, impatience, anger, aggressiveness
3. Motivational aspects: achievement motivation, competitiveness, guidance toward success and ambition
4. Evident or open behavior: alertness, celerity, hyperactivity, work involvement
5. Cognitive aspects: the necessity of environmental control and characteristic attributional style.

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Conversely, Type B behavior pattern is defined as a relative absence of these qualities.

A number of studies on TABP have shown that this behavioral construct appears to contribute directly to coronary disease.[2–8] However, other studies find no such relationship.[9–11]

Many researchers have reported that Type A individuals experience higher sympathetic activation while working on challenging tasks than do Type B individuals. These characteristics can be seen in an increase in the psychophysiological indexes, namely, heart rate and cardiac response reactivity to the stressful situation.[12–16] Findings regarding cardiac reactivity and the development of coronary heart disease are also contradictory. In fact, some studies seem to point out that Type A individuals do not show greater sympathetic activation and reactivity than individuals who are Type B.[17,18] Moreover, it has also been shown that, after exposure to stressful situations, Type A individuals present a slower recovery of their psychophysiological indexes than Type B

individuals, suggesting that the cumulative effect of the sequences of "fast activation–slow recovery," together with a stressful life, may contribute to cardiovascular disease.[7,8,19]

In the last few years, the reliability of several aspects of the TABP as a predictor of coronary disease has been questioned. One of the main methodological criticisms concerns the nature of the instruments used to measure TABP.[11,20] A range of instruments (eg, the Structured Interview, the Jenkins Activity Survey, the Bortner Scale, the Framingham Scale, and the Thurstone Scale) rely on very different evaluation techniques that could have contributed to the confusing results. Furthermore, the effectiveness of the structured interview can be very subjective because its use depends on the interviewer's personal style in conducting the interview. In addition, small sample sizes in many studies and the primarily male makeup of groups studied may have led to poorly generalizable results.[4,21] Finally, that the Type A construct is defined as a multidimensional pattern also points to differing results.[20,22]

Because the Type A construct includes such different behaviors as hostility, achievement, and impulsive style, intensified behavior in any one of these behavioral manifestations implies that the participant could be categorized as type A, yet it has not been proved that all could carry the same coronary risk. Because of the lack of unanimous results, the scope of research has switched from considering TABP as a multidimensional profile to focusing on the various subcomponents of the pattern as a means of detecting which of these could be considered predictors of cardiovascular disorders.

This line of research seems to point toward the emotional components, mainly anger and hostility, as the "toxic" element.[22–25] Thus, the coronary-prone behavior has substantially changed from being initially described according to the characteristics of the TABP to being defined in relation to the emotional component. The "new" coronary-prone behavior seems to be determined by emotional aspects such as anger, hostility, and aggressiveness (the AHA! syndrome),[26] which may form the link between emotions and cardiovascular disorders.

In a previous study,[27] we found an important correlation between two of the main hostility measures–the Buss-Durkee Hostility Inventory (BDHI) and the Cook-Medley Hostility Scale (HO)–with some factors of the Jenkins Activity Survey (JAS). On the one hand, the BDHI "experience of hostility factor" correlated .33 ($p < .0001$) with the JAS-A factor (a general measure of Type A behavior)[28] and .40 ($p < .0001$) with the JAS-S factor (speed and impatience).[28] The BDHI expression of hostility factor correlated .46 ($p < .0001$) with the JAS-A factor and .62 ($p < .0001$) with the JAS-S factor. On the other hand, the HO correlated .43 ($p < .0001$) with the JAS-A factor and .48 ($p < .0001$) with the JAS-S factor.

Because of the appreciable correlation between the hostility measures and the JAS-S factor, we advocate the possible further consideration of the JAS-S factor in future research. That is, it would be necessary to check whether the JAS-S factor predicts heart rate reactivity. This way, we could assess the convenience of continuing to use this scale to predict individuals who are at risk of suffering cardiovascular disorders.

Our present study attempts to determine the effectiveness of the JAS-S factor as an appropriate instrument to detect individuals who are prone to respond with important increments in the heart rate. We measured two of the parameters considered to be indicative of cardiovascular risk, namely, psychophysiological activation and reactivity,[8] in persons who scored high or low on the JAS-S factor. In view of the results of previous studies,[7,11] we suggest that using a real stress situation is particularly appropriate for defining the examined individual's psychophysiological profile.

Our main objectives in conducting this study were (a) to determine cardiac activation in two groups of participants (the high scorers and low scorers on the JAS-S factor) when they face a real-life stressful situation (an academic examination), and (b) to establish group cardiac reactivity to each of the different questions used in the task phase of the test. We hypothesized those high scorers in the JAS-S factor would show greater psychophysiological activation and reactivity than low scorers would. In addition, we predicted that high scorers in the JAS-S factor would show a slower recovery of tonic psychophysiological levels of heart rate than would the low scorers.

METHOD

Participants and Establishing Groups

Our initial sample consisted of 196 men and women in an undergraduate psychology course. They volunteered for this study and completed the JAS form C. Although we administered the entire scale, we considered only speed and impatience (factor S), the factor that shows a higher correlation with hostility measures. Because of the extreme scores the students obtained on the JAS-S factor, we assigned participants to one of the two groups (high-S and low-S scorers). The 89 selected students were aged 19 to 26 years ($M = 22.29$ years, $SD = 1.55$), and all claimed they were in good health. We used the JAS-S scale to classify those students whose standard scores were ≥ 6.5 (percentile 75 or over) as high-S scorers and those whose

standard scores were < –6.5 (percentile 25 or under) as low-S scorers. Thus, we formed two groups: high-S scorers (n = 45; age M = 22.16 years; SD = 1.62) and low-S scorers (n = 44; age M = 22.43 years; SD = 1.47). We detected no significant differences between men and women in the sample in our analyses to discover the eventual existence of gender differences. The participants' safety and privacy were guaranteed because no part of the experiment was physically invasive and the participants' identities were not revealed in the data analyses.

Instruments

We used a four-channel Letica-4000 polygraph with HSC-400 Letica electrocardiograph to detect, amplify, integrate, and register heart rate, and electrodes 5 cm × 2 cm Ag/AgCl Letica and a Letica biogel as the contact medium. We detected the electrocardiogram from standard lead II. The obtained signal was amplified and integrated in a Letica CAR-300 cardiotachometer. Chart speed was 5 mm/sec, and polygraphic records were scored by hand. To display the 10 stimuli in the task phase, we used a Reflecta Diamator AF slide projector and to calculate analyses of variance, we used the Statistical Package for Social Sciences (SPSS /PC+ V4.0.1).[29]

Recording Session

We asked all participants to wash their hands with soap and water before entering the soundproofed experiment room, in which temperature and lighting conditions were kept constant. Once in the room, the participants were seated in comfortable armchairs, electrodes were attached, and they were given taped instructions. Participants were asked to remain quiet (avoiding movement) and relaxed until the recording session ended. If the participant had no further questions, the recording session began. It consisted of (a) a 10-minute period for adaptation to the experimental environment, (b) a 20-minute task period in which the subject was shown the 10 stimuli, and (c) a 10-minute recovery period.

In the adaptation phase, the aim was to allow the participants to adjust to the experimental environment and measure their basal heart rates; we therefore administered no stimulus.

In the task phase, 10 stimuli in the form of multiple-choice statements were presented on a slide projector, as in the following example: *Which author postulates the "Systems Dissociation Theory" to explain the Activation process? (a) Lindsley; (b) Duffy; (c) Lacey; (d) Malmo.* The participant was to respond orally (ie, say "a," "b," "c," or "d") within 30 seconds, the length of time the stimulus was displayed on the screen. The stimuli were replaced at 2-minute intervals.

We measured both tonic and phasic heart rate dimensions. With the tonic heart rate dimension, the average tonic heart rate was measured throughout the phase. Because the lapsed time of stimulus presentation was quite wide, we also considered the phasic heart rate dimension. We measured the patients' heart rates following stimulus presentation, using a slightly modified version of the Raskin and Hare[30] procedure. We determined cardiac reactivity by averaging heart rate in the 20 seconds immediately after presentation of the stimulus. (To calculate the heart reactivity, Raskin and Hare only consider the average from the 18 seconds immediately following to the presentation of a stimulus.)

Finally, in the recovery phase, we presented no stimulus so that we could observe how each participant's heart rate was recovering its normal level, that is, its tonic dimension. When the recording session ended, we detached the electrodes and thanked the students for their participation.

RESULTS

The data in Table 1 show the mean cardiac activation and standard deviations for both the high and low JAS-factor S scorers across the three experimental phases. We performed a 2 (High, Low) × 3 (Phase of Experiment) analysis of variance, with repeated measures on phase. As readers can see, high-S scorers showed higher heart rate values than low-S scorers in the three phases of the experiment. The analysis of variance showed significant differences for group, $F(1, 87) = 46.57, p < .0001$; phase, $F(2, 174) = 57.39, p < .0001$; and their interaction, $F(2, 174) = 12.67; p < .001$. A t test for independent samples showed significant differences in between-group heart rates in the examination, $t = 4.59, p < .0001$, and recovery phases, $t = 8.01, p < .0001$, but not in the adaptation phase, $t = .99; p < .32$.

Within-groups repeated measures analysis of variance showed significant heart rate differences across the three experimental phases for high-S scorers, $F(2, 88) = 26.82, p < .0001$). as well as low-S scorers, $F(2, 86) = 63.37\ p < .0001$. Figure 1 shows high- and low-S scorers' profiles across the three phases of the experiment.

Our second purpose in conducting this study was to establish heart rate reactivity to each of the 10 stimuli presented in the examination phase. The data in Table 2 show the mean heart rate reactivity and standard deviations for each group.

We performed a 2 (High, Low) × 10 (Stimuli of the Task Phase) analysis of variance with repeated measures on task-phase stimuli. Here again, one can see that high-S scorers had higher heart rate reactivity values than low-S scorers in the 10 stimuli of the task phase. The analysis of variance

TABLE 1
Mean Scores and Standard Deviations of Heart Rate Across the Adaptation, Examination, and Recovery Phases in High-S and Low-S Scorers

	Adaptation	Task	Recovery	
High-S				
M	91.14	108.13	97.32	$F(2, 88) = 26.82$
SD	10.57	13.52	10.95	$p < .0001$
Low-S				
M	89.28	96.93	82.22	$F(2, 86) = 63.37$
SD	6.04	9.49	6.13	$p < .0001$
t	NS	4.59	8.01	
p		< .0001	< .0001	

Note. Analysis of Variance With Repeated Measures on Phase: Group, $F(1, 87) = 46.57$, $p < .0001$; Phase, $F(2, 174) = 57.39$, $p < .0001$; and Interaction, $F(2, 174) = 12.67$, $p < .001$. NS = not significant.

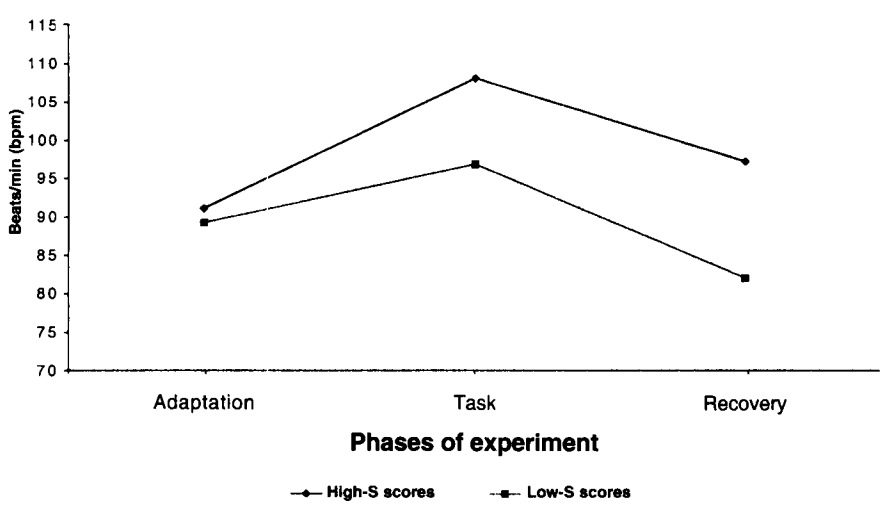

Figure 1. Evolution of averaged cardiac activation in high and low scorers on the Jenkins Activity Scale-S, a measure of speed and impatience, across the three experimental phases.

showed significant differences for group, $F(1, 87) = 105.63$, $p < .0001$; stimuli, $F(9, 783) = 26.83$, $p < .0001$; and their interaction, $F(9, 783) = 12.01$, $p < .0001$. A *t* test for independent samples showed significant differences in between-groups heart rate reactivity in stimuli 5, $t = 8.44$, $p < .0001$; 6, $t = 6.73$, $p < .0001$; 7, $t = 9.25$, $p < .0001$; 8, $t = 4.36$, $p < .0001$; 9, $t = 5.44$, $p < .0001$; and 10, $t = 3.63$, $p < .0001$), but not in stimuli 1, $t = .65$, $p < .51$; 2, $t = .98$, $p < .32$; 3, $t = 1.41$, $p < .16$; and 4, $t = 1.89$, $p < .06$. Likewise, within-group repeated-measures analysis of variance showed significant differences in heart rate reactivity across the 10 stimuli of the examination phase for high-S scorers, $F(9, 396) = 12.77$, $p < .0001$, as well as low-S scorers, $F(9, 387) = 28.03$, $p < .0001$. For the high- and low-S scorers' profiles across the 10 stimuli of the task phase and for standard deviations for both groups, see Figure 2.

TABLE 2
Mean Scores and Standard Deviations of Cardiac Reactivity Throughout the 10 Stimuli of the Examination Phase in High-S and Low-S Scorers

	Reactivity, by stimulus										
Scorers	1	2	3	4	5	6	7	8	9	10	
High S											
M	107.35	99.80	100.19	103.42	107.59	105.90	104.10	99.98	95.50	96.26	$F(9, 396) = 12.77$
SD	12.72	7.74	6.93	7.20	9.67	10.07	6.49	5.51	6.04	8.07	$p < .0001$
Low S											
M	105.92	98.30	98.27	99.93	92.18	93.27	90.80	93.82	88.13	90.40	$F(9, 387) = 28.03$
SD	7.21	6.59	5.78	10.06	7.37	7.42	7.06	7.66	6.74	7.12	$p < .0001$
t	NS	NS	NS	NS	8.44	6.73	9.25	4.36	5.44	3.63	
p					.0001	.0001	.0001	.0001	.0001	.0001	

Note. Analysis of variance with repeated measures on stimuli: group ($F[1, 87] = 105.63$; $p < .0001$), stimuli ($F[9, 783] = 26.83$; $p < .0001$) interaction ($F[9, 783] = 12.01$; $p < .0001$).

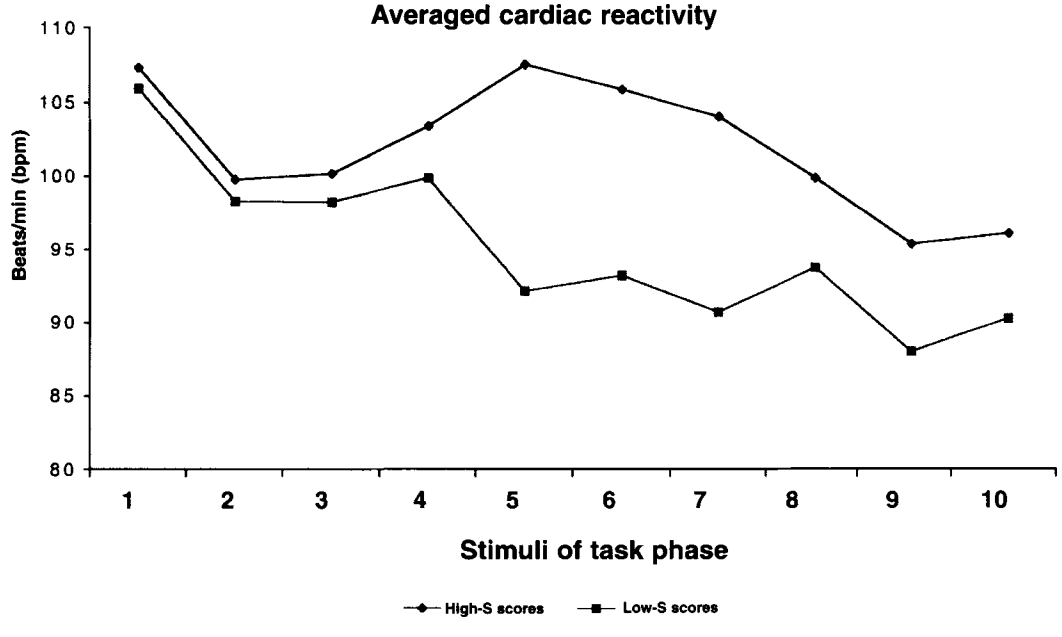

Figure 2. Evolution of averaged cardiac reactivity across the 10 stimuli of the examination phase in high and low scorers on the Jenkins Activity Scale-S, a measure of speed and impatience.

COMMENT

Our first purpose in conducting this study was to determine heart rate in its tonic dimension. The results we present here appear to confirm our hypotheses, that is, high-S scorers exhibit higher activation in the three experimental phases (adaptation, examination, and recovery) than low-S scorers. Indeed, these results seem to be consistent with previously reported studies.[12–16]

In the adaptation phase, the lack of significant differences is consistent with previous studies showing that situations before participants' exposure to stress had a similar effect on both groups of participants.[31,32]

Regarding the examination phase, the finding that high-S scorers obtained significantly higher scores than low-S scorers agrees with a great number of studies[18,33–35] but not with others.[36–43] However, it is worth noting that group heart rates increased during the examination phase in both groups.

The significant between-groups differences in the recovery phase are consistent with some previous research findings[8,19,44] but disagree with other studies[37–41] whose design did not include the recovery phase as a decisive aspect in identifying participants prone to suffer cardiovascular disorders.

As for the change in the mean heart rate throughout the three experimental phases, two aspects are worth considering: (a) the heart rate increase during the examination phase in both groups, and (b) the considerable heart rate decrease during the recovery phase in low-S scorers.

The increase in heart rate during the examination phase was somewhat expected because mental activity per se produces a great cardioaccelerator effect.[45,46] The differences we found during the recovery phase supported our hypothesis and agreed with findings in previous studies.[8,19,44] That is, compared with the high-S scorers, the low-S scorers seemed to recover their previous basal values more quickly. Indeed, we claim that the characteristics of the recovery phase (rather than the participant's initial physiological reactivity) could be relevant in detecting those persons prone to experiencing cardiovascular disorders.

Cardiovascular recovery following stress exposure has not been included in many investigations. However, we believe that the study of this parameter provides much information about the cardiovascular functioning of participants and is useful in predicting the eventual appearance of cardiovascular dysfunction.[44] We encourage further research on the fast activation–fast recovery and fast activation–slow recovery profiles, which are defined as the response that statistically and functionally does not sustain a disturbance in the organism's homeostatic and adaptive processes.

The fast activation–fast recovery profile is the characteristic response of individuals who are not prone to cardiovascular disease.

Conversely, the fast activation–slow recovery profile corresponds to the psychophysiological response characteristic of individuals prone to cardiovascular disorders. Concretely, the longer time these people take in recovering their basal values implies a greater exposure of the organism to the effects of the catecholamines and cortisol, implying that the probability of dysfunction increases.

We also sought to assess heart rate in its phasic dimension by determining cardiac reactivity for each of the 10 stimuli we presented to the participants. Our results support the stated hypothesis because high-S scorers showed higher cardiac reactivity values than low-S scorers did after each stimulus was shown. That cardiac reactivity does not show significant differences among groups in the first 4 stimuli of the task phase reproduces the profile we observed in analyzing the tonic dimension of the heart rate, namely, an important increment in the two groups' heart rates when they pass from the adaptation to the task phase. However, when analyzing the heart reactivity in both groups along the 6 remaining stimuli, we found significant differences. Again, this made us consider the existence of different response patterns for each group.

Indeed, when we consider the changes in cardiac reactivity during the presentation of the 10 stimuli in each group, interesting profiles appear. The change of cardiac reactivity for high-S and low-S participants during the 10 stimuli showed significant differences, but the differences are considerably bigger in the low-S scorers. Although a habituation process is observed in both groups of participants, this process is faster in the low-S scorers than in the high-S scorers. Low-S scorers seem to habituate progressively to the examination phase, whereas high-S scorers appear to undergo a much slower habituation process.

Thus, whereas high-S scorers maintain high response levels, low-S scorers progressively diminish their own responses, and these differences are greater as the examination phase progresses. In short, on the basis of the data we compared, one could think that low-S scorers progressively adjust to the examination or a task situation, whereas each new stimulus seems to elicit a relevant response from high-S scorers.

Perhaps the existence of higher autonomic lability among the high-S scorers measured by the heart rate index could explain why these participants incur cardiovascular disorders in the medium and long term. Particularly, the existence of a differential physiological effect between high- and low-S scorers could be observed with regard to real life stress situations, such as the test taken by the participants in the task phase. This consideration may allow us to establish completely different patterns for high- and low-S scorers.

To sum up, a few considerations are worth noting. First, although a clear association among hostility and CHD is not detected in some studies,[47] this relationship is uneven in others.[48-50] Therefore, because hostility measures could predict CHD incidence and because hostility measures, in turn, positively correlate with the JAS-S Factor,[27] we proposed that it would be pertinent and interesting to verify the relevance of the speed and impatience factor (JAS-S) in detecting individuals who are prone to coronary disease. Such a study could contribute information on the conflicting findings on this point.

Second, the profile obtained by each group in our experiment reveals a peculiar change pattern in each case because high-S scorers need more time than low-S scorers do to recover their basal psychophysiological levels.

Third, we can state that high-risk individuals (or high-S scorers) are identified by their slow recovery, rather than by their initial psychophysiological reactivity.[8,11,19] In fact, one clear differential response between the two groups is found in the recovery phase. In particular, we propose to consider the fast activation–slow recovery profile as an important variable in detecting individuals prone to coronary disorders.

Most studies conducted so far have focused on detecting differences in cardiac activation and reactivity between high-S and low-S scorers. However, in the light of our findings, we propose that clinicians also consider the recovery phase when studying people who are prone to coronary disease.

Fourth, psychophysiological activation, reactivity, and recovery seem to be the appropriate parameters for detecting individuals prone to coronary disorders from a biopsychosocial perspective. However. because activation, reactivity, and recovery are probably not the only factors involved in the process, we should also consider psychological (internal demands), social (social support), cognitive (appraisal processes), situational (external demands and challenges), and personal (abilities and skills) factors.

Last, if we assume that hostility could be considered a risk factor for cardiovascular disorders,[51–53] and if we also assume the existence of an important correlation between measures of hostility and the JAS-S factor (speed and impatience), we suggest the relevance of continuing to investigate this scale of the JAS.

Some authors[54] point out that those patients who had additional infarctions or died during the 1st year reported increased irritability and frequent anger more often than patients who survived without any complications.

In this study, analyses of the standard subcomponents of the JAS show that only the speed-impatience factor predicted poor prognosis. Results indicate that global Type A scores were not associated with the prognosis of myocardial infarction. We would like to suggest that before completely rejecting TABP as a multidimensional profile related to coronary heart disease, clinicians should conduct further research to determine which of the TABP components might contribute individually to cardiovascular risks.

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NOTE

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