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Are high-impulsive and high risk-taking people more motor disinhibited in the presence of incentive?

Antoni Rodríguez-Fornells[a,*], Urbano Lorenzo-Seva[b], Antoni Andrés-Pueyo[a]

[a]*Department of Personalitat, Avaluació i Tractaments Psicològics, Facultat de Psicologia, Universitat de Barcelona, Spain*
[b]*Facultat de Psicologia, Universitat Rovira i Virgili, Spain*

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Abstract

In this study we assess the ability to inhibit certain planned actions in relation to the impulsiveness and venturesomeness traits of personality using the stop-signal paradigm. In the first condition subjects performed the stop-signal task without incentives. In the second condition, subjects performed the same task using an approach-avoidance conflict situation: speed of response was rewarded and the lack of inhibition was punished. Twenty male subjects were selected after preliminary testing using the impulsivity subscale from the Eysenck Personality Inventory. The main findings were: (1) no deficit in motor inhibition was found for high-impulsive subjects; estimated mean reaction times of inhibitory processes were the same for both high- and low-impulsive groups and (2) the presence of an incentive in the conflict situation affected motor inhibition for low venturesomeness subjects, who adopted a more cautiousness response strategy.
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1. Introduction

The ability to inhibit planned or ongoing actions is an important control mechanism that allows efficient reactions to be made to sudden changes in the environment (de Jong, Coles, Gratton, & Logan, 1990). These changes can be evoked by the unexpected consequences of one's behavior or by other factors, for example, the presence of motivationally significant stimuli. The consequent reaction is either motor inhibition or an appropriate response modulation, where the

* Corresponding author at current address: Department of Neuropsychology, Otto von Guericke University, Universitätsplatz 2, Posstfach 4120 (Pfälzer Platz, Gebäude 24), 39016 Magdeburg, Germany. Fax: +49-391-671-1947.
E-mail address: rodriguez.antoni@medizin.uni-magdeburg.de (A. Rodríguez-Fornells).

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present response is changed for a more appropriate one. A deficit in this inhibition capability or lack of response modulation is observed in a range of impulsivity symptomatology, such as the tendency to act before understanding the task or to give an answer without giving sufficient consideration to other possible solutions (Tannock, Schachar, Carr, Chajczyk, & Logan, 1989). Thus, lack of response modulation can lead a person to behave impulsively and diminish his/her ability to adapt successfully to nonpredictive situations.

This ability to inhibit certain behaviours or an inappropriate response modulation has been used to assess different temperamental properties of personality (Buss & Plomin, 1975; Kagan, 1965; Kipnis, 1971; Shapiro, 1965). For example, extraversion, one of the well-known temperamental dimensions, has usually been associated with impulsivity and disinhibition (Eysenck & Eysenck, 1977). Impulsiveness has been defined as characteristic of people who act on the spur of the moment without being aware of any risk involved (Eysenck, Easting, & Pearson, 1984; Eysenck, Pearson, Easting, & Allsopp, 1985). Both, Buss and Plomin (1975) and Eysenck and Eysenck (1977) consider impulsivity to be a multifaceted trait. Buss and Plomin (1975) included four subscales in their definition of impulsivity: lack of inhibitory control, decision time, sensation seeking and lack of persistence. Initially, Eysenck and Eysenck (1977) identified four factors of disinhibition or impulsivity that were quite similar to Buss and Plomin's definition: narrow impulsiveness, risk-taking, non-planning and liveliness. Later, Eysenck and Zuckerman (1978) and Eysenck and Eysenck (1978) conducted a factorial analysis of impulsivity scales and sensation-seeking scales and suggested that there were two main facets to disinhibition: *impulsiveness* ("doing and saying things without thinking") and *venturesomeness* (which included sensation-seeking and risk-taking behavior items; hereafter refered also as risk-taking; Eysenck & Eysenck, 1978). Impulsivity and risk-taking behavior or venturesomeness were described as different but related constructs, with correlations between both traits normally being around 0.30 and 0.40. Impulsiveness correlated mainly with psychoticism and neuroticism and to a lesser extent with extraversion, while venturesomeness correlated mainly with extraversion and psychoticism (Eysenck & Eysenck, 1978; Diaz & Pickering, 1993). The difference between impulsiveness and venturesomeness is that risk-taking subjects are aware of the risks involved in their actions but are prepared to take a chance (Eysenck & Eysenck, 1978). In contrast, impulsiveness is more closely related to the lack of awareness in the assessment of possible risks and the negative outcomes of an action. Zuckerman, Kulhman and Camac (1988) have identified a major psychometric factor called P-impulsive unsocialized sensation seeking dimension (P-ImpUSS) that combines impulsivity, lack of socialization, sensation-seeking and aggression traits. An excellent study of multimethod assessment of impulsivity in a large adolescent sample have been recently performed by White, Moffitt, Caspi, Bartusch, Needles, and Stouthhamer-Loeber (1994).

1.1. Disinhibition and psychopathology

In the clinical context, the concept of impulsivity has been extensively associated with certain personality disorders, such as antisocial or borderline disorders. For example, one of the main features identified by Cleckley (1976) was that psychopathic behavior was characterized by the lack of control in one's own behavior. Frontal lobe damage has also been related to impulsivity, at least to those personality changes observed in some patients. The concept of *disinhibitory psychopathology* has been proposed in order to bring together several traditionally separate

psychological categories — psychopathy, hysteria, hyperactivity, antisocial and impulsive personality, and alcoholism (Gorenstein & Newman, 1980). All of these groups have the trait of impulsivity in common.

Psychopathy has always been related to passive avoidance deficits in response to signals of punishment (Hare, 1978; Lykken, 1957). The most common explanation for this deficit concerns poor fear conditioning in psychopaths. However, some experiments have shown that psychopaths are able to inhibit responses in the presence of punishment cues if there is no reward contingency (Schmauk, 1970). Newman, Patterson, Howland, and Nichols (1990) have proposed that psychopaths display poor passive avoidance under conditions involving approach-avoidance conflict. Gorenstein (1982, 1991) has related the behavior pattern of psychopaths to patients with lesions of the septum, hippocampus and prefrontal cortex, characterized by a diminished ability to sustain goal directed activity and to modulate impulsive responses. Also, these lesions enhance the tendency to emit what might be termed "dominant" or "stereotypical" responses (Luria, 1973). Similar effects have been elicited from frontal lesion patients in various laboratory tasks (Luria, 1973; Milner, 1963). Gorenstein accounts for these performances by arguing that cognitive mediating processes — the way in which the central nervous system represents events that are not available to the senses at that time — are weak in antisocial individuals. These subjects should show an impaired ability to generate a sustained mental representation of the hypothetical punishing event. On the contrary, reward seeking behavior is always based on some cancroid need that does not depend on a sustained mental effort in order to be maintained. One of the predictions of the model is that if the punishment is always present these subjects will not show passive avoidance deficits.

Similar proposals have been made for describing the dynamic of psychopaths in relation to temperament and motivation. Gray (1987) proposed a neuropsychological theory of personality for anxiety and impulsivity dimensions based on learning theory studies. The dimension of anxiety is related to the behavioral inhibition system (BIS) mechanism whose function is to respond to signals of punishment, nonreward or novel stimuli. The BIS responds to these kinds of input by interrupting ongoing behavior (passive avoidance), increasing attention to environmental stimuli, especially those which are novel or disruptive, and increasing arousal levels. Impulsivity depends on the second system, the behavioral approach or activation system (BAS), which mediates the approach to signals of reward or active avoidance. Fowles (1980) has described the BAS as an appetitive, reward seeking system that functions to initiate approach behavior. Psychopaths showed a weak BIS resulting in a strong reward-seeking behavior, with the appearance of impulsive behaviour, and the inability to learn from past punishments (passive avoidance deficit).

Both, the BAS and BIS have positive inputs to a third nonspecific arousal system (NAS), which is responsive to their excitatory inputs. An increase in the activity of either behavioral system, BIS or BAS, results in heightened NAS activity. These systems produce an increment in general arousal that leads to an increase in the intensity or vigor of whatever behavior follows. For example an increase in response speed or signal sensitivity could be the consequence of BIS or BAS activation in NAS. Following this argument, it seems reasonable to believe that if response initiation is a function of the BAS, high-impulsive subjects would be "geared to respond", as Brebner and Cooper (1974) postulate for extraversion. In contrast, anxious subjects, with BIS dominant activity, would be more prone to engage in functions associated with behavioral inhibition (e.g. response inhibition), and heightened signal sensitivity. Newman et al., working more closely in Eysenck's N and E space rather than in Gray's dimensions, have demonstrated that extraverts

respond faster than introverts following punishment or negative feedback (Nichols & Newman, 1986; Patterson, Kosson & Newman, 1987). A similar finding has been replicated by Derryberry (1987) using a visual detection task. In this context, introverts responded more slowly than extraverts following negative feedback. Moreover, introverts tended to display response inhibition when punished and extraverts were prone to show response facilitation (Pearce-McCall & Newman, 1986).

An excellent review of the studies that have been conducted to test Gray's model has been compiled by Pickering, Diaz, and Gray (1995). For example, one of the predictions is that impulsive or extraverted subjects are more susceptible to signals of reward (hyperactivated BAS) and introverts or anxious subjects are more susceptible to signals of punishment (hyperactivated BIS). Pickering et al. (1995), after reviewing the studies, conclude that it is not possible to make accurate predictions by simply noting the presence of rewards and punishments and that it is useful to consider possible approach and avoidance behaviors. For example, punishments can produce decreases (passive avoidance) or increases (active avoidance) in the probability of a particular response. In that case, passive avoidance will be more efficient in anxious subjects (BIS activity) and active avoidance will favour impulsive subjects (BAS activity). These suggestions are useful in order to specify properly the predictions of the model, bearing in mind the nature of punishment and reward conditions and the different kinds of behavior possible in each task.

Consistent with these observations, Wallace, Newman, and Bachorowski (1991) have proposed a third mechanism of impulse response in anxious subjects. This model has been formulated by taking into consideration the results of Bachorowski and Newman (1985, 1990) concerning motor inhibition and impulsivity. On observing that anxious individuals sometimes appear to be behaviorally impulsive, Wallace et al. (1991) conceived neuroticism as the predisposition to experience heightened levels of NAS activity. This means that when receiving the same stimuli, neurotic individuals will tend to react faster and more forcefully because of the greater increment in NAS activity, therefore, mimicking impulse behavior.

1.2. The stop-signal paradigm

A number of paradigms have been developed in the context of experimental psychology that might be useful when measuring the inherent ability to inhibit actions in different groups (normal and clinical). One interesting candidate is the stop-signal paradigm, which has been used in basic research to investigate the inhibitory mechanisms from central to peripheral structures (Logan & Cowan, 1984). In this task, subjects are engaged in a reaction time (RT) task, and occasionally and unpredictably, they are presented with a signal (e.g. a tone or a light) that instructs them to inhibit their response to the stimulus. This stop signal may occur at one of several delay times following the presentation of the stimulus (Fig. 1). Depending on the stop-signal delay, subjects will be more or less successful in withholding their response, being less accurate with longer stop-signal delays (de Jong et al., 1990). Different variants of the task have been used; so a subject might be asked to stop all responses, to stop one response but to make another, or to stop only one of several possible responses (de Jong, Coles, & Logan, 1995; Logan, 1995).

The stop-signal paradigm has been used to infer the "point of no return", the moment at which processing becomes ballistic and the subject cannot inhibit his/her motor response (de Jong et al., 1990). In interpreting the performance in this paradigm most investigators have used a horse-race model. This model describes a race between two processes. The first group of processes are the

ones typical of choice RT (detection, response choice and preparation of response). The second group of processes in the race involves those evoked by the appearance of a stop-signal (detection of the signal and the inhibition of response). If the first group wins the race, the response is produced; if the second group wins the subject is able to inhibit the response. The horse-race model also allows the non observable latency of inhibitory processes to be estimated. The estimation of the internal response to the stop signal (stop-signal reaction time, SSRT) is carried out by using

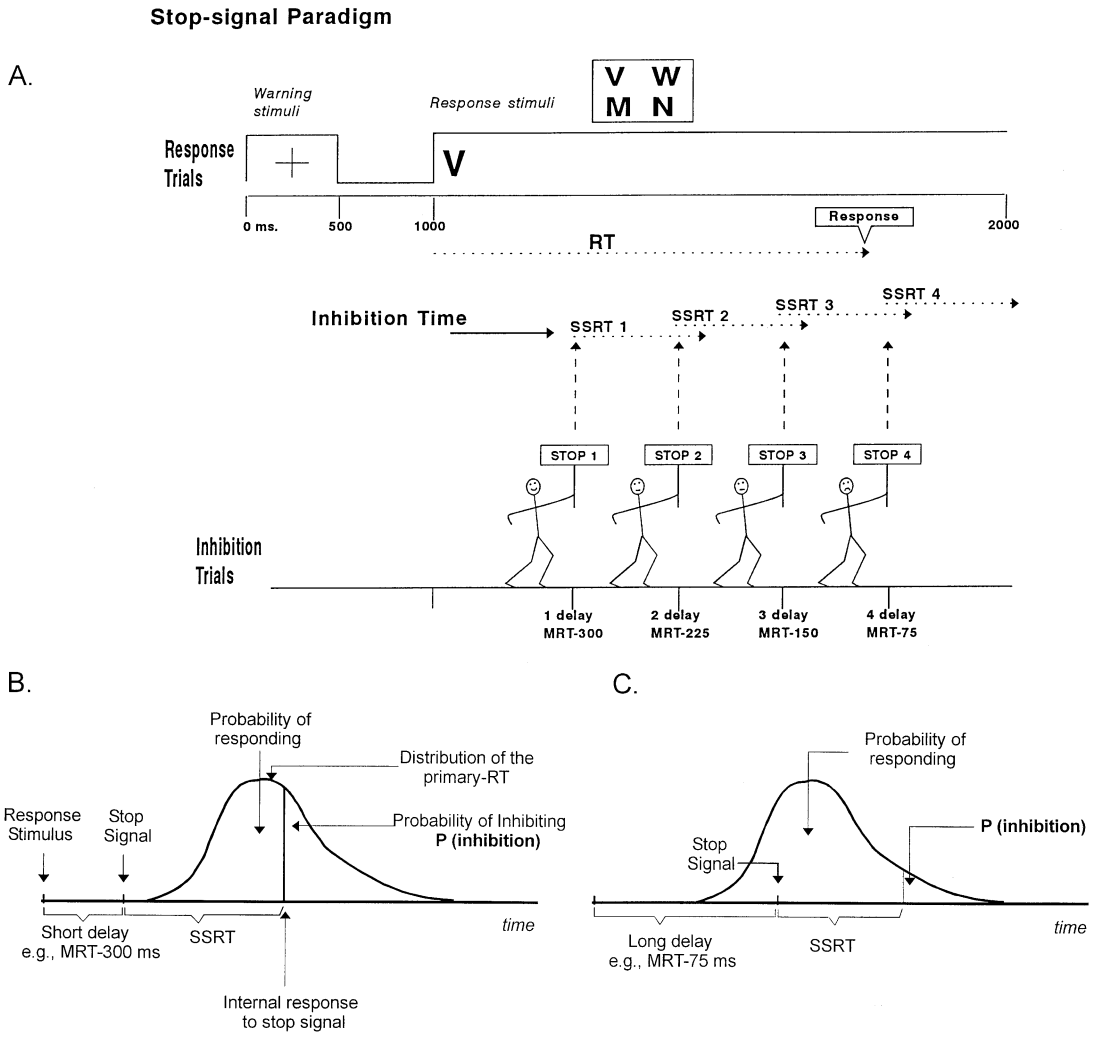

Fig. 1. Stop-signal paradigm used in this experiment. Shown in (a) are examples of the response and inhibition trials presented during the task. Shown in (b) is the graphic adapted from Logan and Cowan (1984) representing the assumptions and predictions of the race model. It shows how the probability of inhibiting a stop-signal trial depends on the distribution of the reaction times (primary task) in a particular subject (MRT), the stop-signal delay and the characteristic SSRT of every subject. In (c) we exemplified what happens when a longer stop-signal delay is used, reducing the probability of stopping the response in that trial. SSRT = Stop-signal reaction time, assumed to be constant in this representation and the race model. MRT = mean reaction time estimated for every subject.

the distribution of non signal RTs and the probability of responding given a stop signal delay (Logan & Cowan, 1984). The model assumes that SSRT is constant.

The probability of response inhibition is dependent on go signal RT, the SSRT and the stop signal delay. For some delays, subjects will be able to inhibit responses all the time while for others they will respond all the time. So it is important to choose intermediate delays at which the probability of responding is between 0 and 1. There are several methods for choosing delays, ranging from an arbitrary choice to tracking several parameters of a subject's performance and setting delays contingent on the values of those parameters. Logan (1995) suggested that at least three or four delays should be used. If there is only one delay, subjects will prolong their go-signal RTs in order to maximize the probability of inhibiting (Lappin & Eriksen, 1966; Logan, 1981). Prolongation is minimized with the presentation of several early and late delays. Also, the greater the probability of a stop-signal occurring, the greater is the probability of inhibition and the slower the RTs to the go signal (Logan & Burkell, 1986). These effects can be minimized by instructing subjects appropriately and presenting stop signals with a relative infrequency.

As to how the delays should be chosen, Logan suggested various options. They can be chosen arbitrarily, and fixed for all conditions and all subjects or they can be set relative to go signal RT. In the second procedure, the stop-signal delay can compensate for differences in mean RT. First, we need to calculate some measure of RT and then the delays can be chosen. The shortest stop-signal delay should be close to zero and the largest should be close to the mean. For example, Schachar and Logan (1990a) set stop signal delays equal to mean go signal RT minus 0, 100, 200, 300, 400 and 500 ms. If the inhibition functions generated by this method differ substantially in variance, then it is useful to plot these functions in terms of a Z score that represents the relative finishing time in standard deviation units (Logan, 1995). Another method is to set signal delays relative to the go signal RT distribution directly, using the specific percentiles as delays or using a tracking algorithm (see de Jong et al., 1990, 1995; Osman, Kornblum, & Meyer, 1986).

1.3. The present research

The stop-signal paradigm has never been employed to study the effect of extreme groups of personality or personality disorders related to impulsiveness on the ability to inhibit current actions. There are, however, certain advantages to be gained in using this paradigm. In the stop-signal paradigm an external stimulus always triggers the inhibition process. Thus, it might be useful to test whether the lack of response inhibition, traditionally linked to the behaviour of such patients, is due to the absence of a stop environmental signal or whether it occurs even in the presence of external stop signals.

One of the child disorders associated with the construct of impulsivity, attention deficit hyperactivity (ADHD), has recently been studied with the stop-signal paradigm (Schachar & Logan, 1990a, 1990b; Schachar, Tannock, & Logan, 1993; Schachar, Tannock, Marriott, & Logan, 1995; Tannock et al., 1989). Using this paradigm, Schachar & Logan (1990a; Schachar et al., 1993) found that the ADHD group showed a slower RT and a greater variability within this parameter. Also, the inhibition mechanism of the ADHD group was triggered less frequently, and found to show greater variability and to be slower. Tannock et al. (1989) found that treatment with methylphenidate was associated with the increased probability of inhibiting a response, increased slope of inhibition functions and decreased stop-signal RT (SSRT), compared with placebo condition.

In this study we analyse the performance of extreme high- and low-impulsive subjects in the stop-signal paradigm in two different conditions: with and without incentives. In the incentive condition, subjects performed the stop-signal paradigm in an approach-avoidance conflict situation with different payoffs: response speed was rewarded in RT trials and the lack of inhibition in stop-signal trials was punished. One of the most interesting points is to examine the change in the behavior of different impulsivity groups with the presence of an incentive, including the analysis of risk-taking behavior. The different involvement of the motivational systems (e.g. BIS or BAS) in the approach-avoidance conflict that we present in the second session of the stop-signal paradigm might be reflected in a different pattern of motor inhibition in stop-signal trials for impulsive groups.

If we consider that high-impulsive subjects are characterized by a higher BAS activity, then the presence of reward should evoke an increment in arousal and an active approach in these subjects. It might be reflected in a faster RT so as to obtain more benefits. The punishment condition should be capable of activating the BIS system, with an increment in arousal and attention as predicted by Gray's model. This increased activation of the BIS system might be reflected in a greater sensitivity for the detection of the stop-signal, in other to avoid punishment (more passive avoidance) or make errors. This situation is non-confounding because there is no presence of active avoidance of punishment that would favour the performance of impulsive subjects.

2. Method

2.1. Subjects and personality assessment

Twenty male subjects were selected after preliminary testing conducted on the impulsivity subscale using the Eysenck Personality Inventory (EPI, Eysenck & Eysenck, 1964; Revelle, Humphreys, Simon, & Gilliand, 1980) from a student group of approximately 700 psychology students. The mean age of low-impulsive subjects was 20.1 ± 1.3 (S.D.) while that of the high-impulsive group was 20.9 ± 1.6. The mean score on the impulsiveness subscale of extraversion (EPI) was 8.1 ± 1.7 for the high-impulsive group and 1.7 ± 1.4 for the low-impulsive group. The mean value of the impulsiveness subscale in all the group screened (700 students) was 4.6 ± 2.2 and for the Extraversion scale was 11.6 ± 3.8. None of the selected subjects had high neuroticism scores (mean for high-impulsive group was 10.2 vs. 9.9 for low-impulsive, $t = -0.33$), and none of them were receiving drugs or medication at the time of testing or had a history of psychiatric or neurological disorders. All subjects gave their written informed consent and were paid for their participation. Subjects also answered the I_7 Impulsiveness Questionnaire (Eysenck et al., 1985), which measures impulsivity, venturesomeness (risk-taking behavior) and empathy. The correlation between both measures of impulsivity (from EPI Extraversion scale and I_7) was 0.92.

2.2. Apparatus and stimuli

The stimuli selected for the choice RT were the uppercase letters V, M, W, N, with a visual angle of 0.5° at a distance of 1 m from the screen. The stop signal was an auditory tone (a "beep", 50 ms of duration, 70 dB, 1000 Hz) generated by the internal speaker of the computer and administered binaurally through headphones.

2.3. Procedure

The choice RT task was the same as that used by de Jong et al. (1990, 1995). The letters V and M were assigned to one hand, and the letters W and N to the other. Each trial began with the illumination of a fixation point in the centre of the screen for 500 ms. This point disappeared and was followed after 500 ms by the presentation of a response stimulus — one of the possible letters. Each letter was presented in the centre of the screen, for 1 s. The screen remained blank for an interval of 1500 ms. Subjects responded by pressing the appropriate key in one of the response boxes held in each hand. The mapping of letters onto keys was counterbalanced across subjects.

Participants were tested in two distinct experimental conditions or sessions. At the *first session*, subjects were trained and performed the choice RT task alone in five blocks of 100 trials each. After each training block, the mean RT (MRT) and accuracy appeared on the screen and the experimenter encouraged the subject to lower his MRT without increasing his percentage of errors above 10%. This training phase was specially important for determining the MRT of each subject in order to set the stop-signal delays accurately. If the MRT and the accuracy levels of the last three blocks were stable then the experimenter explained the stop-signal RT task to the subject. The MRT recorded in the last training block was chosen as the baseline RT. Stop-signal delays were stabilized using this MRT time as follows: MRT-300 ms, MRT-225 ms, MRT-150 ms and MRT-75 ms. Delays remained fixed during the rest of the session. This procedure avoids individual differences in MRT when performing the stop-signal task. Subjects performed five blocks of 200 trials (see Fig. 1).

Subjects were instructed not to respond to the primary-task stimulus when the tone was heard. It was explained that the stop signal would occur in such a way that sometimes they would be able to stop their response and sometimes not. The primacy of the choice RT task was emphasized in the instructions and participants were once more instructed to respond as quickly as possible while maintaining a high level of accuracy. Moreover, they were advised not to delay their responses in anticipation of the stop signal, so as to avoid an extremely high percentage of correctly inhibited stop-signal trials. The experimenter monitored the performance of the ongoing task on completion of each block and informed the subject as to whether his RT to non stop-signal trials was significantly delayed compared with the baseline MRT.

The stop-signal was emitted in 50% of the trials. Each stop-signal delay was equiprobable. The sequence of letters, stop/non-stop trials, and stop-signal delays was random, and different random orders were administered to each subject at each session. The first session lasted approximately an 1 h 30 min and each subject earned $7.

The main purpose of the *second session* was to create an approach-avoidance conflict situation, using different payoffs. This condition was identical to the previous one, except that in this motivational session subjects were rewarded or punished, by winning or losing money depending on their performance. This experiment was run on the following day, at the same time. Subjects were given the following task instructions about reward/punishment conditions:

"Initially you will receive $5 from the experimenter. If your RT to non-stop stimuli (without the beep) is faster than your mean RT (obtained during the training phase) you will earn 2 points. For each point, your earnings will increase by 3 cents. However, if your RT is slower than your MRT you will be awarded 0 points, and you will receive no additional earnings. If the response is completed with the wrong hand or you fail to respond, you will lose 1 point and 3 cents will be deducted from your earnings.

In the stop-signal trials (when the beep sounds), if you refrain from responding you will receive 0 points and not lose any money. However, if you do not inhibit your response, you will lose 1 point. After each trial, you will briefly see the number of points earned (2, 0 or -1) displayed in the centre of the screen. After completing 200 trials (one block) the total amount of money that you have earned will be displayed. The main difference between this session and yesterday's, is that you are now completely free to respond as you wish, without having to abide to any specific instructions. Good luck''.

The experimenter checked that each subject had understood the instructions before beginning the second session, having first completed the training phase. This training phase was shorter than the previous one, only two blocks of 100 trials each. In order to avoid the effects of having practised on the subsequent session, the assignment of responses to the two subsets of stimulus letters was reversed. After each trial, a feedback stimulus was given (1 s duration). This stimulus consisted in the result being displayed on the screen during the intertrial interval, that is whether the subject had earned (2), not earned (0) or lost points (−1).

The order of conditions (non-incentive and approach-avoidance) were not deliberately counterbalanced. The non-incentive condition was tested first with the purpose of providing a direct measure of *baseline motor-inhibition* in both groups, without any possible differential sequential effects in impulsive groups due to the approach-avoidance condition.

At the end of this second session, the experimenter explained to each subject the purpose of the experiment and showed them the two motor inhibition functions, from the first and second session. The experimenter also asked them about the strategies they had used in order to deal with the task in each of the two conditions, especially the second one.

2.4. Data analysis

We measured the following dependent variables for each subject: baseline MRT, MRT to correct response on the primary choice-RT — in the non stop-signal trials (primary-RT), standard deviation of RT to primary task (primary-S.D.), percentage of correct responses, probability of response inhibition in each stop-signal delay, mean SSRT and estimated signal-response RT (SRT *e*). In order to estimate the SSRT, a measure of the latency of the inhibitory process, the RTs from go-signal responses in which no stop signal occurred, were ordered, and the *n*th RT was selected, where *n* is obtained by multiplying the number of RTs in the distribution (*m*) by the probability of responding at a given delay. The *n*th RT estimates the time *t* at which the stopping process finished, relative to the onset of the go signal. To estimate SSRT (relative to stop signal), the stop signal delay must be subtracted from this value (Logan & Cowan, 1984).

In estimating signal-response RT (SRT *e*), the reaction times of the go signal, for which no stop signal occurred were collapsed into a single distribution. The RTs were rank ordered, and the mean of the fastest *n* computed, where *n* is the number of RTs in the distribution (*m*) multiplied by the probability of responding at a given delay. This procedure is the same as that described for calculating SSRT, but with the difference that we take the mean of the fastest *n* RTs, whereas in SSRT, we took the *n*th RT and interpreted it as the finishing time for the stopping process. The mean of the fastest *n* (SRT *e*) is the predicted value from the race-horse model, to be compared with the real observed signal-respond RTs (SRT md): the median of stop-signal trials where the response was not inhibited. The median value was chosen because there were not many non-inhibited

trials in short delays, so that the mean value was more strongly influenced than median values by the presence of outliers. Indeed, in some subjects and with the shortest delay, we could not compute SRT md because practically all the stop-signal trials were inhibited, especially during the second session.

Since impulsivity groups were characterized in RT tasks by a greater variability in their RT distribution, which in turn affected inhibition functions, we need to separate impulsivity effects on the primary task response process from their effects on the inhibitory process. Differences in primary-RT variance may be controlled by plotting the probability of inhibition (*P*-inhibition) as a function of the relative finishing time of the inhibitory process, expressed as a *Z* score (ZRFT) relative to the standard deviation of the primary task RT (for derivation see Logan, Cowan, & Davis, 1984). Specifically, the *Z* relative finishing time is calculated using this formula, ZRFT = (RT−delay−SSRT)/S.D., where RT and S.D. are drawn from the go signal RT. If the inhibition functions from the two groups of impulsivity cannot be aligned by plotting them as a function of ZRFT, then we can conclude that steeper functions obtained by the non impulsive group reflect the effect of impulsivity on the central inhibitory process. Regression lines were fitted to the data sets for probability of inhibition as a function of stop-signal delay and ZRFT, for each subject and each test session. This process yields two slope measures of inhibitory-control, the *P*-inhibition slope and the ZRFT slope.

In addition to this traditional approach to the data analysis of the stop-signal paradigm, we were interested in the analysis of *sequential effects* during this paradigm. As Logan (1995) claims, the analysis of sequential effects or the after-effects of inhibition is a very promising area of research. Moreover, Nichols and Newman (1986), Patterson et al. (1987) and Derryberry (1987) found significant sequential effects on RT tasks associated with the extraversion dimension. Here, we studied the sequential effects after stop-signal trials in both sessions and after reward and punishment trials in the incentive session. First, we computed the probability of inhibition on trial $n+1$, after the stop-signal had also been presented during trial n (correctly inhibited or failed). Second, we evaluated for the incentive condition the influence of obtaining the reward for trial n on the RT of $n+1$ non-stop signal trials. Third, the effect of receiving a punishment in stop-signal trials (failure to inhibit correctly) was evaluated in RT of $n+1$ non-stop trials. In both cases, for reward and punishment conditions, the same analysis was performed for the first session, taking the RT trials with a latency lower than MRT and the non-inhibited trials respectively as control conditions to compare the effect of the incentive.

In order to examine motor inhibition differences for impulsivity groups, analysis of variance with repeated measures was performed on the dependent variables studied, with stop-signal delays (four delays) and session (with and without incentives) as the within factors, and impulsivity as a between factor. The same analysis was performed after splitting the venturesomeness dimension in two groups: low- and high-risk taking behavior.

3. Results

3.1. Effect of the approach-avoidance session on inhibition functions

The mean percentage of inhibited trials (collapsing over all stop-signal delays) was approximately 50% (see Table 1) during the first session, indicating that the delay assignment selection

was properly conducted (Logan, 1995). This percentage of inhibited trials increased significantly (approx. 17%) during the second session, with the presence of reward/punishment conditions. Neither primary RT to go trials nor standard deviation of RT changed between both conditions (Table 1). Moreover, the accuracy of responses was also maintained in the second condition. These results mean that subjects improved their inhibition ability while maintaining the speed and accuracy obtained in the first session.

This was confirmed by the significant reduction in SSRT in the incentive condition. The mean reduction of SSRT was approximately 40 ms, indicating that latency to stop-signal was faster in the incentive condition, thus increasing the number of stop-signal trials that could be inhibited

Table 1
Performance on the stop-signal paradigm during the 1st-session (without incentive) and the 2nd-session (with the presence of incentive)[a]

	1st session				2nd session				$F_{(1,19)}$	P
	M	S.D.	Min	Max	*M*	S.D.	Min	Max		
Primary-RT	414	39	354	517	424	33	376	477	1.97	ns
Primary-SD	93	12	75	122	97	15	77	130	1.80	ns
% Correct	86	5	70	92	85	7	70	96	0.00	ns
SRT md	370	29	328	451	357	24	315	425	12.47	0.002
P (Inhibition)	0.50	0.13	0.25	0.72	0.67	0.10	0.50	0.88	29.85	0.000
Mean SSRT	188	31	144	247	148	13	130	176	40.45	0.000
Slope	2.77	0.55	1.8	3.9	3.04	0.69	1.6	4	2.03	ns
ZRFT slope	35.0	5.45	25.0	43.4	34.0	4.17	26.0	43,7	0.63	ns

[a] $n = 20$; ns = Non significant, $P > 0.05$; Primary-reaction time = mean RT to correct response on primary task, non stop-signal trials; Primary-SD = standard deviation of primary RT;% correct = percentage of correct response; SRT md = median of signal-respond reaction trials; *P* (Inhibition) = general probability of response inhibition; SSRT = Stop-signal RT; Slope = slope of probability inhibition by stop-signal delay; ZRFT = slope for probability inhibition functions by *Z* score, relative finishing time. Data collapsed over all stop-signal delays.

Table 2
Mean of the different parameters of the stop-signal paradigm for the different delays used (mean of the four delays used was 107, 182, 257, and 332 ms respectively)[a]

	1st session					2nd session				
Delays	*P* (Inh.)	SSRT	ZRFT	SRT md	SRT*e*	*P* (Inh.)	SSRT	ZRFT	SRT md	SRT *e*
MRT-300	0.78 (0.15)	226	1.28	356	294	0.96 (0.04)	172	1.76	333[b]	274[c]
MRT-225	0.65 (0.18)	187	0.46	333	324	0.80 (0.21)	142	0.97	309	295
MRT-150	0.39 (0.16)	175	−0.36	352	355	0.58 (0.15)	137	0.23	337	338
MRT- 75	0.18 (0.09)	164	−1.17	378	382	0.28 (0.15)	140	−0.60	373	372

[a] $n = 20$; Values between brackets were standard deviations. MRT = mean reaction time during the training phase; *P* (Inh.) = probability of response inhibition in the corresponding delay; SSRT = stop-signal RT; ZRFT = *Z* score of the relative finishing time; SRT md = median of observed signal-respond RTs; SRT *e* = estimate of signal-response RT.

[b] In the shortest delay mean value was computed only for 12 subjects, due to limited number of non-inhibited trials.

[c] The model estimation of SRT was done only for 16 subjects in this delay.

correctly. The mean latency of SSRT in the first session was comparable with the mean SSRTs estimated in previous experiments using the same task (approx. 196 ms and 174 ms, de Jong et al., 1990, 1995 respectively). As expected, the delay affected significantly the probability of inhibition ($F_{(3,57)} = 253$; $P < 0.000$; see Table 2), as was that of the session effect ($F_{(1,19)} = 40.9$, $P < 0.001$). No significant interaction was found for session and delay ($F_{(3,57)} = 0.25$; $P < 0.299$). These results stress the importance of motivational control conditions in laboratory experiments. In Fig. 2 (top-left panel) we plotted the probability inhibition functions obtained in both sessions. In the top-right panel, the mean SSRT latency was subtracted from the stop-signal delays. In the bottom panel, variability of stopping and primary RT process was controlled, yielding a quasi-perfect alignment of inhibition functions in both conditions.[1]

The correlation between SSRT and primary task RT was $r = -0.11$ in the first condition and $r = -0.16$ in the second condition. This confirms the idea outlined in the model proposed by Logan and Cowan (1984) concerning the independence of response and inhibition processes. As can be seen in Table 2, the race-model estimates of the median of stop-signal-response RTs (SRT *e*; non-inhibited trials) fit perfectly with the observed signal-response RTs (SRT md) for the larger delays (MRT-75 and MRT-150) and correctly for the third of these (MRT-225). The estimates failed in the shortest delay due to the relatively low number of non-inhibited trials contained within it.

3.2. Effects of impulsivity

Both groups of impulsivity did not differ in their MRT during the training phase ($t = -0.73$, $P = 0.480$). Thus, stop-signal delays were approximately the same for both groups. The main results obtained for both groups of impulsivity are presented in Table 3. There were no significant differences between groups in any of the dependent variables of the stop-signal paradigm studied. SSRT were equal for both groups, both for primary-RT and accuracy. Both groups of impulsivity performed in a similar manner in the incentive session, reducing their SSRT and increasing the percentage of inhibited trials. The analysis of inhibition functions is shown in Fig. 3. In the first session, the alignment of both inhibition functions was practically perfect. In the incentive session, both functions increase their probability of inhibition in the same way.

[1] One of the main assumptions of Logan's model is that SSRT is constant. However, and as shown in Table 2, the effect of delay on SSRT is significant (($F_{(3,57)} = 52.5$, $P < 0.001$); SSRT is slower with shorter delays, especially for the first sessions, violating the assumption mentioned. The same results have been found in the studies of Logan and Burkell (1986), Logan and Cowan (1984) and Logan et al. (1984). Logan and Burkell (1986) explained this effect of SSRT variability. If stop-signal reaction time had a constant mean and a non-zero variance, its distribution would be responsible for different SSRT at different delays. When the delay is short and the response is easy to refrain, a great number of stop-signal latencies will be able to inhibit the ongoing response. However, in the presence of longer delays, when an inhibiting response is difficult, only the shortest stop-signal latencies will be fast enough to win the race to response processes and be able to inhibit the response. In this case, the mean average of these latencies should be considerably faster than SSRT mean of shorter or "easy" delays. However, the significant interaction found between delay with session ($F_{(3,57)} = 7.45$, $P < 0.001$) shows that in the incentive session this effect was different. In the second session the SSRT reduction was appreciable only from the first to second delay; comparing the second, third and fourth delays, SSRT appears stable. As subjects in the second session perform under stress, inhibiting more signals without reducing their baseline RT, variability in the second session of SSRT should be reduced so as to appear practically constant, as predicted in the model designed by Logan (1981).

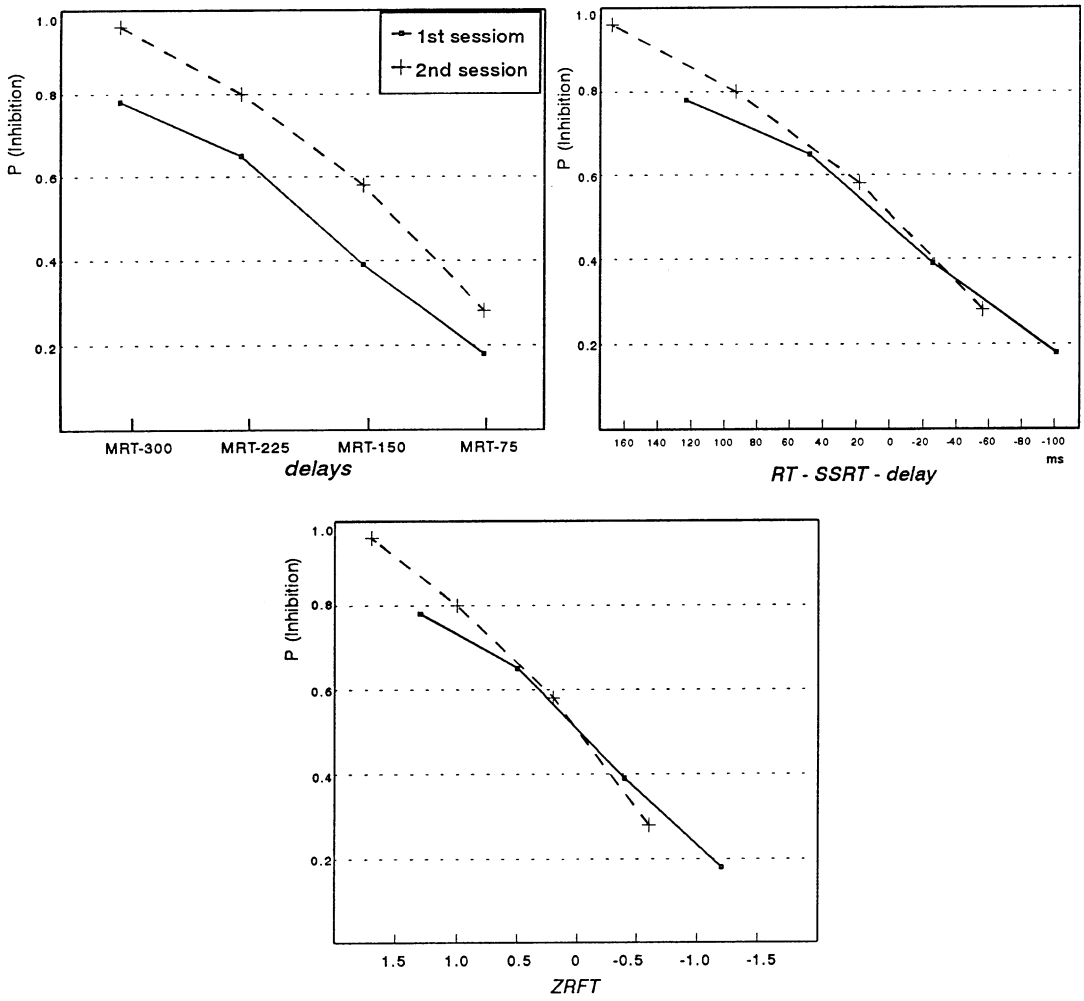

Fig. 2. Probability of inhibition by stop-signal delay for all subjects (top-left panel) and by primary-RT minus SSRT minus stop-signal delay (top-right panel). Probability of inhibition by ZRFT (bottom panel). (RT = primary-RT; SSRT = Stop-signal RT; ZRFT = Z score, relative finishing time).

3.3. *Effects of venturesomeness*

To analyze the effect of venturesomeness in the incentive session, we split the group according to the median value of this dimension.[2] The main effects of group and the interaction of group with session are shown in Table 4. This interaction between group and session was significant for the SSRT and practically significant for the probability of inhibition (see Table 4). This interaction shows that the low risk-taking group enhanced their probability of inhibiting stop-signal trials to a greater degree in the incentive condition compared with the high risk-group, with the corresponding fall in the SSRT. In order to obtain a better analysis of this interaction taking into consideration the four delays used in this study, we computed the corresponding analysis of variance introducing the delay as a within-subject factor also. The interaction between Group×Session

Table 3
Group impulsivity performance on the stop-signal paradigm in the 1st-session (without incentive) and the 2nd-session (with the presence of incentive)[a]

	Low-impulsive ($n=10$)		High-impulsive ($n=10$)	
	1st Session	2nd Session	1st Session	2nd Session
MRT	403 (31)		411 (18)	
Primary-RT	415 (49)	419 (41)	414 (27)	429 (24)
Primary-SD	97 (15)	97 (18)	89 (8)	96 (11)
% correct	84 (6)	84 (8)	86 (2)	86 (4)
SRT md	364 (23)	354 (24)	376 (35)	361 (26)
P (Inhibition)	0.51 (0.16)	0.66 (0.11)	0.48 (0.10)	0.67 (0.09)
SSRT	189 (34)	145 (13)	188 (30)	150 (14)
Slope	2.72 (0.59	3.07 (0.7)	2.80 (0.6)	3.00 (0.7)
ZRFT slope	34.2 (6.8)	34.6 (4.7)	36.5 (3.8)	33.3 (3.5)

[a] S.D. in brackets. MRT = mean reaction time during the training phase; Primary-RT = mean RT to primary task, non stop-signal trials; Primary-SD = standard deviation of primary RT;% correct = percentage of correct responses; SRT md = median of observed signal-respond RTs; P (Inhibition) = probability of response inhibition in the corresponding delay; SSRT = Stop-signal RT; Slope = slope of probability inhibition by stop-signal delay; ZRFT = slope for probability inhibition functions by *Z* score, relative finishing time. Data collapsed over all stop-signal delays.

reached a significant value for probability of inhibition in each delay ($F_{(1,18)}=6.35$, $P<0.03$). The corresponding inhibition functions are plotted in Fig. 4, where the enhanced inhibition of the low-risk group in the incentive condition compared to the performance in the first session is easily observed. The low-risk group reduced their SSRT by 57 ms, while the high-risk group reduced it by only 30 ms. However, the performance of the high-risk group in the first session appeared to be better than that of the low-risk subjects, although the difference was not significant. The average sum of money earned by both groups, low-risk and high-risk, was practically the same.

3.4. Sequential effects of inhibition, reward and punishment

Table 5 (top panel) shows the probability of inhibition on trial $n+1$ after presenting a stop-signal on trial n. There was a significant interaction between risk-taking and session ($F_{(1,18)}=6.32$, $P<0.03$). This interaction reflects the finding that the low-risk group was more greatly affected by the presentation of a stop-signal trial, increasing their cautiousness for the next trial and conse-

[2] Mean value for venturesomeness was 10.7 (3.4), with a median value of 11 (range 2–15). In the group of low-impulsive subjects, five subjects were above the median value (high-risk) and five subjects were included in the group of low-risk taking behavior. In the high impulsive group, three subjects were considered low-risk and seven subjects high-risk. Thus, the mean value of venturesomeness for the low risk-taking group was 7.5 (2.7; $n=8$), while for the high-risk group it was 13 (1.6; $n=12$). Groups were not equal in number because of the number of subjects scoring in the median value. Although both variables, impulsivity (I_7) and venturesomeness were positively correlated, they only shared 14% of variance ($r=0.37$). The mean for venturesomeness in our sample was clearly comparable with other studies using the same scale (I_7) for measuring risk-taking behavior or venturesomeness. For example, Luengo et al. (1991) obtained a mean value for venturesomeness of 11.4 (3.5) in a sample of men. In the study of Corulla (1987), the mean value for men was 10.64 (3.4) and Eysenck et al. (1985) obtained a mean value of 10.3 (3.7).

quently enhancing their percentage of inhibited trials. In contrast, the high-risk group was not so greatly affected by the presentation of stop-signal trials, reflecting a more disinhibited response pattern after stop-signal trials.

The middle and bottom panels of Table 5 show the sequential effects on RT trials after rewarded or punished trials. We performed an analysis of variance of repeated measures entering Session and Type of Incentive (reward or punishment) as within factors, and venturesomeness as a group factor. As expected, the effect of the session was significant ($F_{(1,\ 19)} = 7.62$, $P < 0.02$)

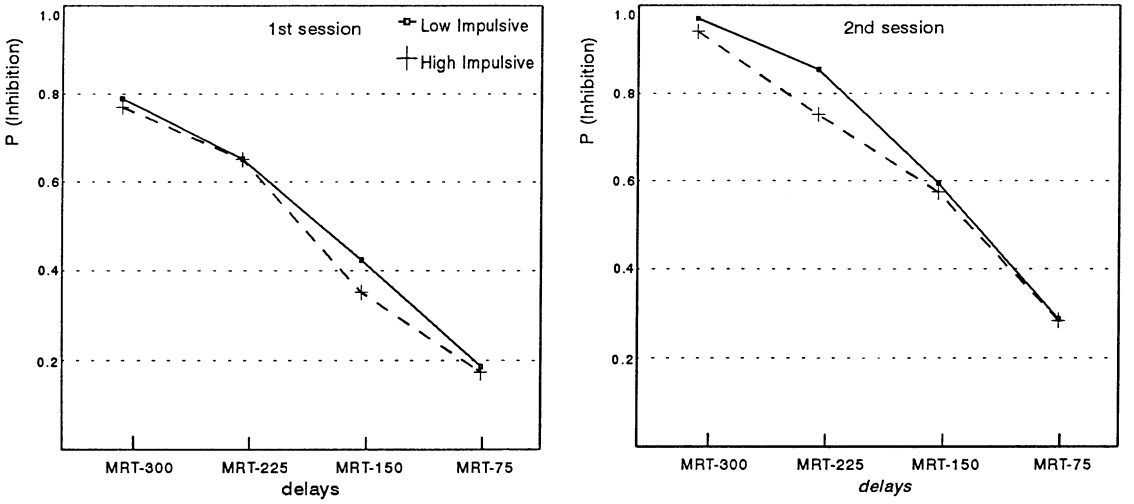

Fig. 3. Probability of inhibition of impulsivity groups by stop-signal delay in both sessions (1st session, without incentive; 2nd session, with incentive). (MRT = mean RT during the training phase).

Table 4
Mean (standard deviation) for risk-taking groups on the stop-signal paradigm in both sessions[a]

	Low risk (n = 8)		High risk ($n = 12$)		Group		Group × Session	
	1st session	2nd session	1st session	2nd session	$F_{(1, 18)}$	P	$F_{(1, 18)}$	P
MTR	410 (27)		405 (25)					
Primary-RT	418 (36)	431 (37)	412 (42)	419 (31)	0.33	ns	0.13	ns
Primary-SD	93 (12)	92 (10)	93 (13)	100 (16)	0.38	ns	1.85	ns
% correct	84 (6)	86 (7)	86 (3)	84 (6)	0.01	ns	1.74	ns
SRT md	376 (32)	363 (28)	367 (29)	354 (22)	0.55	ns	0.00	ns
P (Inhibition)	0.44 (0.16)	0.69 (0.11)	0.53 (0.10)	0.65 (0.10)	0.30	ns	4.26	0.054
SSRT	202 (36)	145 (13)	179 (25)	149 (14)	1.09	ns	5.24	0.034
Slope	2.53 (0.48)	2.89 (0.90)	2.92 (0.58)	3.14 (0.53)	2.45	ns	0.12	ns
ZRFT slope	33.8 (6.9)	32.3 (2.7)	36.4 (4.3)	35.1 (4.6)	4.94	0.039	0.00	ns

[a] MRT = mean reaction time during the training phase; RT = mean RT to primary task, non stop-signal trials; Primary-SD = standard deviation of primary RT;% correct = percentage of correct responses; SRT md = median of observed signal-respond RTs; P (Inhibition) = probability of response inhibition in the corresponding delay; SSRT = Stop-signal RT; Slope = slope of probability inhibition by stop-signal delay; ZRFT = slope for probability inhibition functions by Z score, relative finishing time. Data collapsed over all stop-signal delays.

reflecting the influence of feedback stimuli (incentive) on RT. The type of incentive was also significant ($F_{(1,19)} = 33.5$, $P < 0.001$). As we can see in Table 5, after failing to inhibit a response correctly, RT was significantly delayed in the subsequent trial. This effect can be considered therefore as a clear after-effect of non-inhibited trials. In contrast, when the response was rewarded (RT < MRT) subjects responded faster in the subsequent trial. However, this effect was present in both sessions independent of the feedback stimuli, either for speed trials (RT < MRT) and non-inhibited stop-signal trials in the first session or rewarded/punished trials in the incentive condition. The effect of venturesomeness or the interaction between venturesomeness and session did not reach a significant value. However, visual inspection of the bottom panel of Table 5 shows that for the low-risk group, the punishment effect on the second session increased the RT

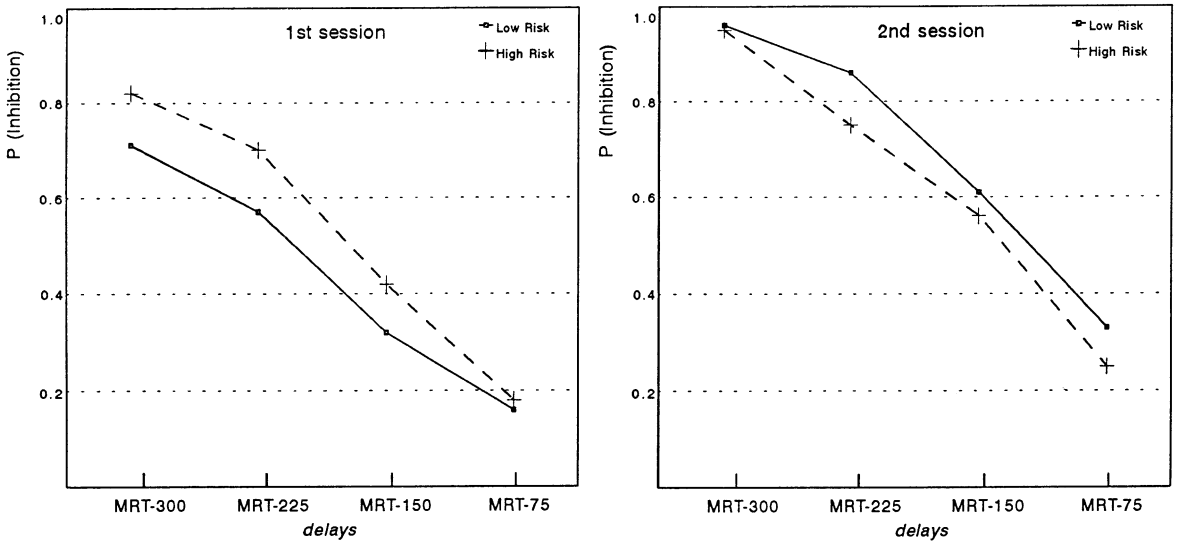

Fig. 4. Probability of inhibition of risk-taking groups by stop-signal delay in both sessions (1st session, without incentive; 2nd session, with incentive). (MRT = mean RT during the training phase).

Table 5
Sequential effects for high and low risk-taking groups: after stop-signal inhibition, after reward and after punishment

	Low-risk	High-risk
P (Inhibition) on n + 1 trial after stop-signal trial		
1st Session	0.50	0.60
2nd Session	0.71	0.66
RT (ms) on n + 1 trials after reward (RT < MRT)		
1st Session	398	398
2nd Session	433	416
RT (ms) on n + 1 trials after punishment (failed to inhibit)		
1st Session	433	416
2nd Session	461	435

of consecutive trials (461 ms). Subjects with low venturesomeness increased their cautiousness after punished trials, reflected in the latency of the consequent RT trials. Moreover, the effect of reward on the high-risk group in the second session produced a tendency for RT to be reduced in comparison with the times for the low-risk group.

4. Discussion

Our primary interest in this study was to assess the effects of impulsivity and risk-taking behavior dispositions of personality on inhibitory processes triggered by an external cue. The stop-signal paradigm developed by Logan et al. (see Logan, 1995) was used for this purpose because it allows the estimation of inhibitory processes latency (SSRT). Moreover, this analysis was complemented by creating an approach-avoidance situation in order to test motivational theories about impulsivity and disinhibitory disorders (Gorenstein & Newman, 1980; Gray, 1987; Patterson & Newman, 1993). These theories suggest that under certain motivational circumstances, disinhibited or impulsive subjects have more problems modulating reward-seeking responses or withholding an appetite response, being less able to stop and reflect on the present situation.

The results showed that extreme high- and low-impulsive subjects did not differ in their motor inhibition capability. Besides, and contrary to our expectations, the presence of an incentive in the approach-avoidance situation did not elicit a deficit in the response modulation of impulsive subjects. The results obtained in the first session, without incentive, contrast with other studies that show a deficient motor inhibition for high-impulsive subjects (Bachorowski & Newman, 1985) and ADHD children (Homatidis & Konstantareas, 1981). However, recent studies by Newman et al. suggest that this motor inhibition deficit for impulsivity was modulated by anxiety (Bachorowski & Newman, 1985; Newman & Wallace, 1993; Wallace & Newman, 1990). Moreover, the motor inhibition tasks used in these studies was a continuous motor task (e.g. drawing a circle slowly), which was quite different from the chronometric motor inhibition task used here. Since in our study neuroticism scores were controlled, further study would need to include extreme scores of neuroticism and impulsivity to test the hypothesis outlined by Wallace et al. (1991) concerning disinhibition of high anxiety subjects.

The results obtained by various authors using the stop-signal paradigm in ADHD children identified a deficit in motor inhibition for this group, clearly related to impulsivity and disinhibition (Schachar & Logan, 1990a, 1990b; Schachar et al., 1993, 1995; Tannock et al., 1989). However, in normal samples, like that used in this study with accurate selection of extreme scores on the impulsivity dimension, this finding has not been replicated. As far as we know, no theoretical model of temperament and personality suggests a lack of motor inhibition ability for *external* stop-signals for high-impulsive subjects when non significant motivational cues exist. This assertion was supported by two recent studies using the stop-signal paradigm where significant differences between ADHD and control children in SSRT were not obtained (Daugherty, Quay, & Ramos, 1993; Jennings, Van der Molen, Pelham, Debski, & Hoza, 1997). Jennings et al. (1997) concluded that motor inhibition ability in children with ADHD was appropriate and differences between studies could be due to differences in sample composition and the method used to set stop-signal delays. Further studies are needed in order to clarify this issue, testing children with ADHD and controls using the stop-signal paradigm with different methods for selecting stop-signal

delays and for controlling the speed and variability of RT. The same kind of research must also be done for personality disorders associated with impulsivity. However, when comparing groups of subjects, inhibition functions must be carefully inspected. For example, using mean RT techniques or tracking algorithms to set stop-signal delays could develop a completely different pattern of results. As shown in Table 2 (see footnote 1) if testing delays varied for both groups of subjects, this effect could induce erroneous conclusions about mean SSRT latency, due to the effect of delay on SSRT distribution, introducing an element of bias into possible interpretations. However, the model proposed by Logan and Cowan (1984) enables us to correct for this possible bias and to make the correct diagnostic of inhibition functions (Logan, 1995).

A recent study performed by Logan et al. (1997) found results that contradict the ones presented here. They studied a large group of students that were assessed with the same impulsivity subscale (EPI). They found a relatively small but significant correlation between SSRT and impulsiveness. Considering the amount of variance explained by this correlation (approx. 10%) the authors' claims about the relationship between impulsiveness and inhibitory control seem to be exaggerated. However, this relationship converges with the data previously presented by this group about the AHDH children. The authors also suggest that a better selection of the sample is required to replicate their effects choosing more extreme impulsiveness scores. The different results obtained in our study probably reflect the different methodological approach used (correlational vs. experimental) and the differences in the selection of the sample. In the study of Logan et al. a correlational approach was selected, while in our study we decided to use an experimental approach. Besides, in our study a small but extreme sample of low and high impulsive subjects was used (the neuroticism dimension being controlled). In the study of Logan et al., the lack of other personality measures like anxiety or neuroticism makes it difficult to clarify the relative contributions of other temperament variables. We think that more research is needed to clarify this issue bearing in mind the different temperamental models and personality theories.

The reduction in SSRT or latency of inhibitory processes in the incentive condition is predicted by the motivational models of Fowles (1980), Gorenstein and Newman (1980) and Gray (1987). The presence of punishment when inhibition failed in stop-signal trials led to an increased activation of the BIS system, increasing NAS activity and attentional sensitivity. This was reflected in the enhanced ability of the subjects to refrain from responding. Thus, the approach-avoidance situation created in the second session stressed the activity of the BIS system. This result confirms the hypothesis outlined by Jennings (1991). This author has proposed a common biological link between Gray's behavior inhibition system (BIS) and motor inhibition.

The most interesting finding was that the presence of mixed incentives affected motor inhibition of low venturesomeness subjects, who tended to increase their cautiousness to a greater extent compared with the performance of the high venturesomeness subjects. The reduction of the SSRT and the increased probability of inhibition in the incentive condition shows that we introduced an inhibitory manipulation that affected the low-risk group more, and with relatively little impact on the high-risk group. This result highlights the importance of this dimension of temperament and the independence of impulsivity and venturesomeness in making predictions about approach-avoidance situations in different motivational models (e.g. Gorenstein & Newman, 1980; Gray, 1987). Risk-taking behavior was more closely related to sensation-seeking and Zuckerman et al. (1988) have included this dimension in a general trait of impulsiveness (P-ImpUSS), although the

facet of sensation-seeking related to thrill and adventure seeking is also loaded onto the sociability factor. This effect of the risk-taking trait is easy to explain if we consider that the subjective experience of risk partially determines the approach-avoidance tendencies of the subjects, and probably sensation-seekers tend to develop lower risk appraisals (Zuckerman, 1994). However, this result must be replicated and has not been theory driven from the models of disinhibition (Gray, 1987; Patterson & Newman, 1993).

The effect of venturesomeness on the approach-avoidance condition was corroborated by the analysis of after-effects of inhibition. Sequential analysis has shown that in the incentive condition the probability of response inhibition increases following the appearance of the stop-signal in the previous trial. This effect is significantly greater for low risk-taking subjects. Moreover, when a trial was preceded by a punished trial (non-inhibited or wrong answering trial) the latency of the present trial was slower compared with trials preceded by reward (with a RT faster than the median reaction time, MRT). This effect of reducing speed after making an error was first described by Rabbitt (1966). There is a tendency among low risk-taking subjects to delay their RTs more than the high risk group after punishment. These results are similar to the sequential data reported by Derryberry (1987), Nichols and Newman (1986) and Patterson et al. (1987), which mainly show that extraverts responded faster after punishment or negative feedback. These studies and the present report highlight the importance of studying the sequential effects in chronometric analysis of personality groups. As argued by Rabbitt (1981), information processing studies should take care of the dynamics and continuous nature of information processing. Subjects do not forget their response and they continue to adjust their information processing system and use it to respond to subsequent demands. Sergeant and van de Meere (1988) in using this methodology compared hyperactive and control children on $n+1$ trials after committing an error. Hyperactive children under high load processing demands did not take sufficient time to ensure that on the succeeding trial a correct response was given. In contrast, control subjects showed a better ability to adjust, slowing their response more after an error.

Although the pattern of results presented for the impulsiveness and venturosemeness groups seem to be robust we would like to mention one problem associated with the specific design used in this study. First and although deliberately done, the incentive session was always administered on the second day of testing. In this case, because order and condition are confounded it is not possible to determine to what extent the practice effects could have induced the Group×Session interactions critical in this experiment. We consider this possibility remote because of the extensive training of the subjects in the first session. Besides, the differences between both conditions, with and without incentives are too large (e.g. the SSRT or the probability of inhibition) to consider them to be practice effects, and also because the performance in the primary reaction task did not change across sessions. A second criticism might be related to the definition of the high- and low-risk groups. Comparing them to the impulsiveness groups, which were carefully selected, the venturesomeness groups were created using the median value. Other studies in the future will be addressed considering only this dimension and creating extreme groups of venturosemeness.

The above results suggest that external or *stimulus-triggered inhibitory processes* did not differ in extreme impulsive subjects and were not responsible for the disinhibited behavior associated with these traits of impulsivity and disinhibition. However, we should bear in mind that the stop-signal paradigm was not designed to test inhibition processes triggered by internal cues or what could be

called *will-triggered inhibitory processes*. Probably, a great number of disinhibited behaviors are triggered by short-term internal representations associated with motivational relevant cues, internal drives or strong reward-seeking tendencies of the organism (see Gorenstein, 1991, for an excellent review). The stop-signal paradigm reflects the ability to refrain from responding when faced with external-stopping signals, instead of measuring how the organism refrains from responding due to internally triggered stopping signals. As far as we know, this hypothesis has not yet been tested. The next step in disinhibited research should involve the development of ways to measure will-triggered inhibitory processes or at least clarify differences between stimulus-triggered vs. will-triggered inhibitory processes. A further example readily comes to mind. Child behavior is easily refrained in the presence of a father's stopping signal; instead, when no external-stopping cues exist, a child's willed-inhibitory processes will not be powerful enough to stop different behaviors and disinhibition easily arises. So, it remains open to speculation as to whether stimulus-triggered and will-triggered inhibitory processes are similar, related or complementary cognitive control mechanisms that allow organisms to modulate response processes. For example, Gray (1982) suggests that the septo-hippocampal system (SHS) is responsible for the BIS function. As rapidly as the SHS can detect a *mismatch* between actual and expected outcomes, the SHS will modulate the response through afferents to different parts of the brain, for example, facilitating or stopping the ongoing motor program through cingulate cortex outputs. However, the SHS monitoring function depends on the actual decision, intentions or goals of the organism, deciding whether or not a response will be inhibited in the presence of an external or an internal stopping cue. SHS comparator function and consequent behavior inhibition were only found to work when expectations were violated.

We believe that it is necessary to conduct more research in order to identify the exact mechanisms involved in the inhibition of action for high and low-impulsive groups. Findings in this field of disinhibition could be useful for therapeutic plans involving all personality disorders and other pathologies related to the trait of impulsivity. In addition to overt behavior measures, further research needs to incorporate other measures of central response activation (Lateralized Readiness Potential), and peripherical motor activation (Electromyography). With these measures we should be able to analyse a subject's ability to interrupt ongoing operations at various stages of motor response processing.

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