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Lateralized Response to Diazepam Predicts Temperamental Style in Rhesus Monkeys

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Based on previous findings in humans and rhesus monkeys suggesting that diazepam has asymmetrical effects on frontal lobe activity and other literature supporting a role for the benzodiazepine system in the mediation of individual differences in anxiety and fearfulness, the relation between asymmetrical changes in scalp-recorded regional brain activity in response to diazepam and the temperamental dimension of behavioral inhibition indexed by freezing time in 9 rhesus monkeys was examined. Animals showed greater relative left-sided frontal activation in response to diazepam compared with the preceding baseline. The magnitude of this shift was strongly correlated with an aggregate measure of freezing time ($r = .82$). The implications of these findings for understanding the role of regional differences in the benzodiazepine system in mediating individual differences in fearfulness are discussed.

A striking feature of behavior in many mammalian species is the pronounced variability among individuals in emotional reactivity and temperament. A temperamental characteristic that has received considerable attention in the recent literature has been termed behavioral inhibition and refers to an individual's propensity to display wariness in response to novelty (for review see Kagan, Reznick & Snidman, 1988). In addition to the considerable corpus of human research on this temperamental dimension, a number of investigators have also described similar temperamental qualities in rhesus monkeys and other species (e.g., Suomi, 1987; Adamec & Stark-Adamec, 1989). In humans as well as other species, this characteristic has been found to be stable over many years (Kagan et al., 1988), at least in part heritable (e.g., Plomin & Stocker, 1989), and associated with a specific cluster of biological concomitants.

Most of the research on biological concomitants of behavioral inhibition has focused on measures of peripheral autonomic function. For example, Kagan and his colleagues (Kagan et al., 1988) have reported that inhibited children, compared with their uninhibited counterparts, show higher heart rates, less heart rate variability, and elevated salivary cortisol levels in response to stressful challenges. Davidson and his coworkers (Straus, Davidson, & Finman, 1991) have recently found that inhibited children also show decreased forearm skin temperature, reflecting increased sympathetic activity when compared with uninhibited children. Moreover, among inhibited children, skin temperature is inversely correlated with salivary cortisol, suggesting that increased sympathetic function (reflected in decreased skin temperature) is associated with higher levels of activity in the hypothalamic–pituitary–adrenal (HPA) axis. In uninhibited children, these variables were uncorrelated, and the difference between the correlation for inhibited compared with uninhibited children was significant.

Recently, Davidson and his colleagues have embarked upon an investigation of possible central nervous system differences between inhibited and uninhibited children (for review see Davidson, 1992a). This study was based on an extensive corpus of literature suggesting that the left and right frontal lobes are differentially specialized for certain positive and negative emotions, respectively, and that tonic differences among individuals in asymmetrical frontal activation are associated with individual differences in affective reactivity and dispositional mood (for review see Davidson, 1992b). In a study of patterns of regional brain electrical activity in a carefully screened cohort of extreme inhibited and uninhibited children, as well a group of children in the middle range, all of whom were approximately 3 years of age at the time of initial electrophysiological testing, we found that inhibited children showed a pattern of relative right-sided frontal activation compared with their uninhibited counterparts. The middle children fell predictably in between (Davidson, Finman, Rickman, Straus, & Kagan, 1993).

In adults, Davidson and his colleagues have reported on numerous associations between baseline frontal activation asymmetry and other psychological and biological characteristics related to emotional responding and psychopathology. In particular, we have found that depressed subjects exhibit less relative left-sided frontal activation compared with nondepressed subjects (Henriques & Davidson, 1991). Subjects with greater relative right-sided frontal activation report more dispositionally negative and less positive mood compared with their left frontally activated counterparts (Tomarken, Davidson, Wheeler, & Doss, 1992). Those with greater relative right-sided frontal activation also report more negative and less positive affect in response to film clips designed to elicit

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these emotions (Tomarken, Davidson, & Henriques, 1990; Wheeler, Davidson, & Tomarken, 1993). Finally, we have recently reported that subjects with greater relative right-sided frontal activation display significantly less natural killer cell activity than their left-activated counterparts (Kang et al., 1991).

In a recent study with rhesus monkeys using brain electrical activity measures similar to those used in human studies, we found that a single acute dose of diazepam increased relative left-sided frontal activation compared with vehicle injection (Davidson, Kalin, & Shelton, 1992). This finding is consistent with human data of Mathew and his colleagues, who demonstrated that a single acute dose of diazepam increased relative left-sided cerebral blood flow in the frontal region compared with placebo (Mathew, Wilson, & Daniel, 1985). Other workers have reported that pharmacological agents (e.g., barbiturates and alcohol) that share certain similarities with benzodiazepines in potentiating GABAergic effects also produce asymmetrical effects (see Frumkin & Grim, 1981).

The purpose of the present study was to examine the relation between a measure of behavioral inhibition in rhesus monkeys and measures of asymmetrical activation of the frontal and parietal brain regions derived from quantitative assessment of brain electrical activity during a baseline condition and in response to an acute dose of diazepam. The differential effects of diazepam compared with vehicle injections on measures of brain electrical activity are reported in Davidson et al. (1992). The central question posed in this study concerned the association between an animal's asymmetrical response to diazepam and the animal's propensity to freeze in response to a standardized challenge. We have previously reported that diazepam has a larger effect on left compared with right frontal brain activity. Kalin and Shelton (1989) have also recently found that diazepam significantly reduced the duration of time monkeys spent freezing in response to the same fear elicitor used in the present study. In the present experiment, we recorded brain electrical activity from the left and right parietal regions to compare with the frontal effects so that we could ascertain whether any association between temperament and asymmetrical response to diazepam was specific to the frontal region. Finally, in addition to examining the relation between freezing and the asymmetrical response to diazepam, we also examined relations between baseline frontal and parietal asymmetry and freezing.

Method

Subjects

There were 9 subjects, 4 male and 5 female infant rhesus monkeys (*Macaca mulatta*). They ranged in age from 1.08 to 1.24 years and weighed between 1.95 and 3.02 kg. The care and treatment of all animals was in accordance with institutional guidelines. The animals were housed with their mothers in a cage (71.5 × 71.5 × 68 cm). Animals were maintained on a 12:12-hr light–dark cycle with lights on at 6 a.m. Infants were given water ad libitum and fed monkey biscuits as well as breastfed by their mothers except during the short experimental separation period. These animals had not been exposed to any other pharmacologic agents prior to this study.

Procedure

Prior to the electrophysiological assessments, a measure of freezing was established for each of the subjects used in this experiment. The infant was first separated from its mother and placed in a different room in a cage (79 × 76 × 71 cm). The infant was then allowed to adapt to the new environment for 10 min before testing. After adaptation, an adult male human entered the room and remained motionless 2.5 m from the cage, avoiding eye contact by presenting his profile to the infant for 9 min. During this time, the infant's behavior was recorded on videotape. The videotape was later analyzed by experienced coders. Freezing duration was scored beginning 3 s after the infant maintained a tense body posture without vocalizing and in the absence of any head movements. Subjects were assessed in this paradigm on four separate occasions, with approximately 2 months between assessments, beginning at 4 months of age and continuing until the infants reached the age of 12 months. The mean duration of time in seconds that the animal froze across the four separate occasions was taken as the index of freezing and used in all subsequent analyses.

Each animal was tested in an electrophysiological recording session that occurred between 8:30 a.m. and 11 a.m. Animals were separated from their mothers and brought to the electrophysiology laboratory. During the session, the animal was manually restrained while electrodes were attached (see below). Brain electrical activity was then recorded for approximately 10 15-s trials prior to injection. Each animal was then injected im with 1 mg/kg diazepam in a 5 mg/ml concentration.[1] The injection occurred approximately 10 min following initial electrode application. Slight variations in time occurred as a function of the quality of the initial pre-injection recordings. Following a period of 20 min after injection (during which the animals were continuously restrained), brain activity was recorded for approximately 10 15-s trials. We determined the exact number of recording trials during each condition of the experiment on the basis of the quality of the recorded electroencephalogram (EEG). We wished to ensure that subjects had at least 20 artifact-free s of EEG across trials within each condition. We chose to use trials that were short (i.e., 15 s) so that we could monitor the quality of the EEG on-line and collect only as many trials as we needed to ensure a sufficient amount of artifact-free data. The interval between trials was usually about 2 s, unless artifact was visibly present in the EEG. In such cases, the next trial began when the EEG normalized. In a second electrophysiology session, which was conducted 3 months following the initial session, animals were tested at the same time of day and were exposed to the identical sequence of procedures, the only difference being that the injection consisted of vehicle only in a volume equal to that of the diazepam. Brain electrical activity was recorded before the injection and following a 20-min period after the injection. Animals were continuously restrained during the entire procedure as in the diazepam session.

EEG Recording and Data Reduction

Left and right frontal (F3 and F4) and left and right parietal (P3 and P4) electrodes were placed according to the standard 10/20 system. Silver–silver chloride electrodes were used, and the four active leads were referenced to linked mastoids.[2] The scalp surface was initially

[1] Injections were given im rather than iv to make this study comparable to other data from the Kalin laboratory on the behavioral effects of diazepam where im injections have been used (e.g., Kalin & Shelton, 1989).

[2] Although an anecdotal observation reported in Nunez (1981) implied that physically linking the ears or mastoids might attenuate the

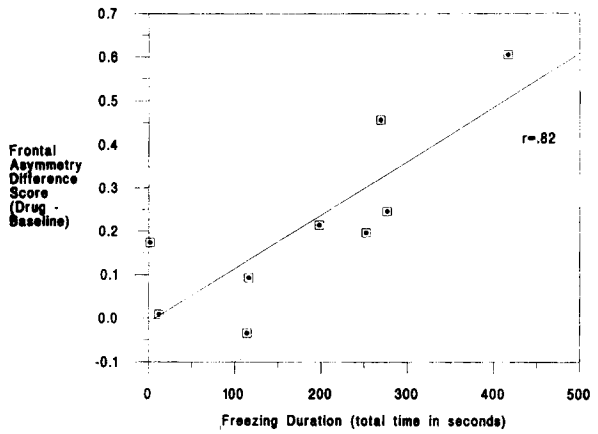

Figure 1. Scatter plot of the relation between asymmetrical frontal response to diazepam (frontal asymmetry score in response to diazepam minus frontal asymmetry score during baseline) and total duration of freezing in response to a human profile (averaged across four testing occasions).

abraded with Omni-Prep (Boulder, CO), and electrodes were then attached with Grass EC-2 cream. All electrode impedances were below 10K ohms. EEG was amplified using Grass Model 12 EEG amplifiers (Grass Instruments, Quincy, MA), with a gain of 20000 and a band-pass of .1 to 200 Hz. The EEG was passed through active, low-pass anti-aliasing filters set at 85 Hz with a 48 dB/octave roll-off. The filtered output was sampled at 250 samples/s on a PDP-11/34A computer. Prior to analysis, all EEG waveforms were visually edited to remove all portions of the record containing eye movement or other artifact. When artifact was detected in any channel, data from all channels were removed. A minimum of 20 artifact-free s of EEG was required per condition, per subject. All subjects met this criterion. There was a mean of 70.95 artifact-free s of EEG per condition (SD = 16.91; range = 29.44–159.74 s). There were no significant differences in EEG duration among conditions.

A fast Fourier transform (FFT) was performed on all artifact-free chunks of data 2 s in duration. The 2-s epochs were extracted with a Hamming window, overlapped by 50%. The FFT output was converted to power density (in $\mu V^2/Hz$); these values were then log-transformed to normalize the data (Gasser, Bacher, & Mocks, 1982).

In our recent report on the effects of diazepam on EEG asymmetry (Davidson et al., 1992), we found that the most robust lateralized changes occurred in the 4–8 Hz frequency range. Therefore, this band was used in the present study. This band was originally chosen because the majority of power in the EEG spectrum was in this band (see Davidson, Chapman, Chapman, & Henriques, 1990). Moreover, this band is consistent with that used in studies with young children in the 3–4 year age range (Davidson et al., 1993). For each subject, we aggregated all artifact-free periods during the baseline (initial) recording period and during the period following the injection. To express the direction and magnitude of asymmetry, a log difference was created (log right minus log left power). Assuming that decrements in power are associated with activation (for reviews see Davidson, 1988; Davidson & Tomarken, 1989), higher numbers on this difference score are associated with more relative left-sided activation.

magnitude of observed asymmetry, in a systematic study we have not found any evidence for this claim (Senulis & Davidson, 1989). Two recent studies have also found no evidence of asymmetry attenuation as a function of linking the ears (Andino et al., 1990; Miller, Lutzenberger, & Elbert, 1991).

Results

Behavioral Data

Total duration of freezing was computed for each of the four behavioral test occasions in response to the 9 min episode of exposure to the human profile. Prior to aggregating across these four separate assessments to derive a single index of freezing, we computed Cronbach's coefficient alpha to determine whether sufficient internal consistency reliability was obtained across the four testing occasions to justify aggregation. Cronbach's coefficient alpha was .80. We then aggregated across the four testing occasions to derive a single index for each animal. The mean duration of freezing across assessments was 183.9 s (SD = 135.6), with animals ranging from 1.3 to 416.6 s. Thus, as expected, there was tremendous variability across animals in their freezing behavior in response to the profile challenge.

Electrophysiological Data

The full description of the electrophysiological changes produced by diazepam and its comparison with vehicle are presented elsewhere (Davidson et al., 1992). As we previously reported (Davidson et al., 1992), diazepam resulted in a significant shift toward relative left-sided frontal activation in the 4–8 Hz frequency band in this group of animals. We did not find significant change in parietal asymmetry assessed at the same times, nor did we find significant changes in either frontal or parietal asymmetry produced by vehicle. For the purpose of the present report, we computed a change score to reflect the magnitude of the asymmetrical response to diazepam for each animal. After deriving an asymmetry score (log right minus log left power) for the baseline and the diazepam conditions, we subtracted the score for the baseline condition from the score for the diazepam condition, separately for the frontal and parietal electrode sites. Eight of the 9 animals showed a positive frontal change score denoting greater relative left-sided frontal activation during diazepam compared with baseline. The mean frontal change score was .217 (SD = .20). Overall, this indicated greater relative left-sided frontal activation in response to diazepam (asymmetry score = .164) compared with baseline (asymmetry score = −.053; $t = 3.20, df = 8$, $p < .02$).

We next examined the relation between the frontal change score (frontal asymmetry during diazepam minus frontal asymmetry during baseline) and individual differences in freezing. The scatter plot for the group of 9 animals is presented in Figure 1. The correlation between these variables was .82, $p < .01$. As can be seen from this figure, the longer the duration of freezing displayed by the animal, the greater the frontal change score, indicating more left-sided and less right-sided activation in response to diazepam compared with baseline.

The mean parietal change score was .108 (SD = .26). When the parietal asymmetry scores during baseline and diazepam

were compared, there was no significant difference ($t = 1.18$, $p > .25$). We then examined the relation between the parietal change score (parietal asymmetry during diazepam minus parietal asymmetry during baseline) and individual differences in freezing. The correlation between these variables was −.36, *ns*. The difference between the correlations of frontal versus parietal asymmetry change and freezing was significant ($p < .01$).

Data from the vehicle session were examined in the same way as the data from the diazepam session. The mean change in frontal asymmetry during vehicle versus baseline was .05 ($SD = .29$; ns); the mean change in parietal asymmetry during vehicle versus baseline was .11 ($SD = .51$; *ns*). The correlation between total freezing time and the change in frontal asymmetry with vehicle was −.14; the correlation between total freezing time and the change in parietal asymmetry with vehicle was −.03. The frontal vehicle change correlation ($r = -.14$) was significantly different from the frontal diazepam change correlation ($r = .82$; $p < .01$). The two parietal correlations were not significantly different.

We also examined the correlations between frontal and parietal asymmetry recorded during the baseline period and duration of freezing. The correlations for both the frontal and parietal regions were clearly in the nonsignificant range ($ps > .35$).

Discussion

We observed a strong association between an animal's asymmetrical response in the frontal region to diazepam and an animal's propensity to freeze in response to a standardized challenge. No such relation was observed between the asymmetrical frontal response to vehicle and freezing, and the correlations for the drug versus vehicle conditions were found to differ significantly. Animals that showed longer freezing times also had larger frontal change scores. The direction of this correlation indicates that those animals that froze the most had less right-sided and more left-sided response to diazepam relative to baseline. It will be important in future research to establish dose-response curves for this effect because in the present study only a single dose (chosen on the basis of behavioral effects previously reported) of diazepam was used. The parietal change score derived from the identical time points showed a nonsignificant inverse relation with freezing and was significantly different from the frontal correlation.

This finding is generally consistent with other literature that has revealed associations between individual differences in fear and anxiety and the benzodiazepine system (e.g., Insel et al., 1984; Robertson, Martin, & Candy, 1978). The fact that animals who showed the most freezing also had less right-sided and more left-sided effects of diazepam raises the possibility that individual differences in behavioral inhibition may be associated with an asymmetrical distribution of benzodiazepine receptors such that those animals who manifest the greatest amount of fear-related behavior have lower receptor densities in the right compared with left frontal regions. The relatively low density of benzodiazepine receptors in a region that has previously been implicated in fear-related behavior (e.g., Davidson & Tomarken, 1989; Silberman & Weinberger, 1986) might result in this region's being less capable of modulating negative affect once it was triggered. This decreased capacity to modulate negative affect would result in such animals exhibiting dispositionally high levels of fearfulness. Clearly, other mechanisms involving different aspects of the benzodiazepine system may also be involved.

In 3-year-old humans, we have found an association between baseline levels of frontal activation asymmetry and behavioral measures of temperamental inhibition (Davidson et al., 1993). In the present study, the correlation between baseline frontal activation asymmetry and freezing was not significant. One reason for the failure to detect such a relation in the present data may be related to the nature of our baseline assessment. Infant monkeys were restrained by a human experimenter while measures of brain electrical activity were obtained. Thus, we really did not have a nonstressed baseline. In fact, the overall group mean of frontal activity during the baseline condition showed right-sided activation, a pattern that is different from that found in human studies with large sample sizes (Tomarken, Davidson, Wheeler, & Kinney, 1992). Because of the overriding stress in the situation, most animals showed right-sided frontal activation during the baseline period. We are currently collecting the same types of brain electrical activity measures from implanted intracortical electrodes using radiotelemetry. With such a system, we can obtain a nonstressed baseline and would expect to find correlations in this case between baseline frontal asymmetry and individual differences in freezing.

In conclusion, this study has demonstrated a robust relation between individual differences in the asymmetrical response to diazepam in the frontal region and the temperamental dimension of behavioral inhibition. It implicates the benzodiazepine system in behavioral inhibition and suggests that individual differences in the asymmetry of certain features of this system may subserve variations in fearfulness.

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