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A computational account of altered error processing in older age: Dopamine and the error-related negativity

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When participants commit errors or receive feedback signaling that they have made an error, a negative brain potential is elicited. According to Holroyd and Coles's (in press) neurocomputational model of error processing, this error-related negativity (ERN) is elicited when the brain first detects that the consequences of an action are worse than expected. To study age-related changes in error processing, we obtained performance and ERN measures of younger and high-functioning older adults. Experiment 1 demonstrated reduced ERN amplitudes in older adults in the context of otherwise intact brain potentials. This result could not be attributed to uncertainty about the required response in older adults. Experiment 2 revealed impaired performance and reduced response- and feedback-related ERNs of older adults in a probabilistic learning task. These age changes could be simulated by manipulation of a single parameter of the neurocomputational model, this manipulation corresponding to weakened phasic activity of the mesencephalic dopamine system.

Over the past 10 years, several researchers have tried to clarify the mechanisms by which error processing, at both the functional and structural levels, is realized in our brains (for reviews, see Coles, Scheffers, & Holroyd, 1998; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). Much of this research has been stimulated by the discovery of an electrophysiological marker of error detection, the error-related negativity (ERN; Gehring, Goss, Coles, Meyer, & Donchin, 1993; or Ne, Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991). When participants commit errors in reaction time (RT) tasks or receive feedback signaling that they have made an error, the ERN appears as a negative deflection in the electroencephalogram (EEG). It is believed that the signal giving rise to the ERN serves an evaluative control function, in the sense that it can be used to learn adaptive behaviors or to determine the need for adjustments of control settings in other parts of the cognitive system (e.g., Botvinick, Braver, Barch, Carter, & Cohen, 2001; Gehring et al., 1993; Holroyd & Coles, in press).

Recently, the ERN has also been employed as a tool for studying error processing in various neuropsychologically impaired populations. These include patients with Parkinson's disease (Falkenstein, Hielscher, et al., 2001; Holroyd, Praamstra, Plat, & Coles, in press), schizophrenia (Ford, 1999; Kopp & Rist, 1999), obsessive-compulsive disorder (Gehring, Himle, & Nisenson, 2000), and lateral prefrontal damage (Gehring & Knight, 2000), all of which have been reported to show abnormal ERNs. The general notion is that the study of the ERN and error-related behavior in such populations may be informative about the functional deficit underlying the specific cognitive and behavioral manifestations associated with each population. In addition, such studies, in combination with existing knowledge of the dysfunctioning brain circuits in specific patient groups, may help to further establish the source of the ERN signal and the areas to which the signal is projected. In particular when complemented with a formal model of ERN function, this type of research may also contribute to the functional validation of the ERN and to theories of error processing.

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In this paper, we will apply this research logic to the study of error processing in healthy older adults, a group that has been extensively documented as having an executive control deficit (for reviews, see Moscovitch & Winocur, 1995; Phillips & Della Sala, 1996; van der Molen & Ridderinkhof, 1998). It is as yet unknown whether this executive control deficit may, at least in part, arise as a result of deficient monitoring functions such as the error-detection function associated with the ERN signal. On the basis of two experiments, we attempted to address this important general issue by investigating why the ERN is smaller in older adults (Band & Kok, 2000; Falkenstein, Hoormann, & Hohnsbein, 2001) and what this tells us about changes in error processing occurring in older age. But before we turn to these questions, we briefly review the characteristics of and existing theory regarding the ERN.

The Error-Related Negativity

The ERN can be seen in the event-related brain potential (ERP) waveform, derived by averaging EEG epochs that are time locked to the incorrect response. The ERN is a negative component with maximum amplitude over frontocentral recording sites. Its peak amplitude is reached about 80 msec following an incorrect overt response, but its onset may coincide with the first incorrect electromyographic (EMG) activity associated with the erroneous response (Gehring et al., 1993). Using source localization techniques, several researchers (e.g., Dehaene, Posner, & Tucker, 1994; Holroyd, Dien, & Coles, 1998) have localized the source of the ERN in or very near the anterior cingulate cortex (ACC), which is part of the brain's limbic system. Neuroimaging studies (e.g., Carter et al., 1998; Kiehl, Liddle, & Hopfinger, 2000) have provided corroborating evidence for the activation of the ACC in association with errors.

The ERN is elicited after errors irrespective of response modality (Holroyd et al., 1998; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001) and occurs after both errors of choice (e.g., pressing an incorrect response button) and errors of action (e.g., responding when one is not supposed to; Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996). Subthreshold activation of the incorrect response (as measured by EMG) accompanied by the correct overt response can also elicit an ERN (e.g., Coles, Scheffers, & Holroyd, 2001). ERN amplitude varies with error awareness when the degree of certainty about the required response varies due to data limitations (Scheffers & Coles, 2000), but it appears to be unaffected by the level of awareness when there is uncertainty about the *actual* erroneous response (Nieuwenhuis et al., 2001). These and other results are consistent with the notion that the ERN reflects the activity of a generic error-detection system that has access to information that is not necessarily available to conscious error monitoring. There is also evidence that the amplitude of the ERN increases with increasing muscle activity in the incorrect response hand (Scheffers et al., 1996; but see Gehring et al., 1993) and with increasing number of incorrectly chosen response parameters (Bernstein, Scheffers, & Coles, 1995). This type of result suggests that the ERN amplitude may be sensitive to the size of the error or degree of incorrect response activation.

Theories of the functional significance of the ERN have, at least until recently, centered around the notion that the ERN reflects a process that compares a representation of a correct response with a representation of the actual response (e.g., Bernstein et al., 1995; Coles et al., 2001; Falkenstein et al., 1991; Scheffers & Coles, 2000). The ERN amplitude reflects the degree of mismatch between these representations. This type of account is referred to as the "mismatch theory" of the ERN. The representation of the actual response is likely to derive from a central feedback system, since latencies of the ERN onset are too short to allow for external feedback. To obey speed instructions, a person does not always wait to respond until all possible information about the appropriate response is available, but rather uses whatever information is available at the time of the response. However, the comparator system makes use of the fact that continued stimulus processing after the actual response tends to lead to an increasingly reliable representation of the correct response. Finally, the mismatch signal—generated when the comparator detects a mismatch between the appropriate and the actual response—is hypothesized to form the input to a remedial action mechanism that is responsible for immediate error correction and strategic adjustments that reduce the likelihood of further errors (e.g., Coles et al., 2001; Gehring et al., 1993).

A more refined theory of the ERN, combining elements of mismatch theory with reinforcement learning principles, has recently been proposed by Holroyd and Coles (in press; Holroyd, Reichler, & Coles, 1999). According to this theory, the ERN is generated when a negative reinforcement-learning signal is conveyed to the ACC via the mesencephalic dopamine system. This signal is utilized by the ACC to modify performance on the task at hand. A further sketch of Holroyd and Coles's theory and of a formal model implementing this theory is given in the introduction to Experiment 2, in which the model was used to simulate behavioral data and ERN amplitudes of younger and older adults.

A final general issue of importance for the present study is the finding of an ERN-like negativity after feedback when such feedback indicates that an error was made (i.e., *negative* feedback; Holroyd & Coles, in press; Miltner, Braun, & Coles, 1997). For instance, in the ERP study of Miltner et al., participants were required to estimate a 1-sec interval. Participants received visual, auditory, or somatosensory feedback that indicated whether the interval they had produced was correct (where "correct" was defined in terms of a time window around 1 sec). Following negative feedback, a negative component occurred that peaked around 200–300 msec after the onset of the negative feedback stimulus. Its phenomenology corresponded closely to that of the response-related ERN, and source localization analyses suggested that, for all three modalities,

the scalp distribution of the negative potential was consistent with a source in or very near the ACC—the same locus as that reported for the response-related ERN. Miltner et al. concluded that the response-related and feedback-related ERN (henceforth response ERN and feedback ERN) are manifestations of the same, generic, error-detection system.

Aging and Error Processing

Two previous studies have examined the effect of aging on the response ERN (Band & Kok, 2000; Falkenstein, Hoormann, & Hohnsbein, 2001). Band and Kok investigated the error-related ERPs of younger (18–28 years old) and older (60–76 years old) adults in a mental rotation task that required speeded responding to the identity (G or R) and parity (mirrored or normal) of a rotated (45° or 135°) stimulus. Older adults made many more errors than younger adults but only in the more difficult (135°) rotation condition. For the analysis of the ERN, all conditions were pooled so that the average ERNs were largely determined by the ERNs in the difficult rotation condition. The ERNs for older adults were significantly smaller than for younger adults. Importantly, the pattern of immediate error corrections—compared with younger adults, older adults corrected more of their errors in the easy rotation condition but fewer in the difficult rotation condition—led Band and Kok to suggest that older adults' smaller ERN might have been caused by their uncertainty about the required response in the difficult rotation condition. This raises the possibility that older adults show a smaller ERN only in tasks in which the representation of the correct response is easily compromised.

This possibility can be tested by looking at age effects on the ERN in a basic choice RT task with simple stimulus–response mappings and minimal data limitations. From such choice reaction time (RT) tasks, it is known that when participants are asked to immediately correct their errors, or to signal any recognized errors by some arbitrary, other response, older adults correct and signal as many of their errors as do younger adults (Rabbitt, 1979). Interestingly, Falkenstein, Hoormann, and Hohnsbein (2001) compared error-related ERPs—associated with performance in such a four-choice RT task—of a group of younger (19–25 years old) and older (54–65 years old) adults. Despite the absence of age differences in error rates or error correction rates, older adults' ERN amplitudes were significantly smaller. This pattern of results—much smaller ERNs in older adults in the context of statistically similar error rates and correction rates—was replicated in a second experiment by using the arrow version of the Eriksen flanker task, in which distractor stimuli tend to activate the incorrect response. Thus, it seems that the decrease in ERN amplitude with aging is not necessarily related to a weakened representation of the correct response.

On the basis of our Experiment 2, we will offer a neurocomputational explanation of the reduced ERN in older adults using an instantiation of Holroyd and Coles's (in press) model of the ERN. First, we present Experiment 1, in which we intended to replicate the basic phenomenon—reduced ERNs in older adults—in a task often used to study error-related processing. This experiment also enabled us to further discount two alternative explanations of this phenomenon: the possibility, already discussed above, that the reduced ERN is due to a compromised representation of the correct response for older adults, and the possibility that the reduction in ERN amplitude is accompanied by a general decrease in ERP component amplitudes in older adults, and might therefore have a nonspecific cause.

EXPERIMENT 1

In Experiment 1, a group of younger adults and a group of healthy, high-functioning, older adults performed a letter version of the Eriksen flanker task (see Coles, Gratton, Bashore, Eriksen, & Donchin, 1985; Eriksen & Eriksen, 1974). This is a choice RT task in which the target letter is flanked by distractor letters. These flankers can be associated with either the same (i.e., congruent) response as the target or with the opposite (i.e., incongruent) response. It is well known that when participants respond quickly to such incongruent stimulus arrays, they tend to make many errors (e.g., Coles et al., 1985; Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988). Thus, we pressed our participants to respond very quickly.

Method

Participants. Sixteen younger (8 women) and 16 older (8 women) adults participated in this experiment. The younger participants, ranging in age from 18 to 23 years (M = 20.4 years, SD = 1.6 years), were undergraduate students at the University of Amsterdam and received course credits for their participation. The older participants were recruited by advertisements in a weekly political journal. They ranged in age from 60 to 80 years (M = 69.6 years, SD = 6.0 years) and received a fixed payment for their participation. According to self-report, all older adults were healthy and alert, free of psychoactive medication, and living independently in their own homes. A standard health questionnaire was used to select participants with no history of perceptual, motor, or neurological disorder. Twelve older participants had received higher education. A computerized Raven Standard Progressive Matrices test (Raven, Court, & Raven, 1988) was administered to compare the two age groups with respect to fluid intelligence. Raw Raven scores were 51.6 items out of 60 (SD = 4.3) for the younger adults and 46.7 items out of 60 (SD = 6.1) for the older adults. All of the younger and older participants had far and near visual acuities of at least 0.4 as measured by Snellen charts.

Stimuli. Stimuli were presented in black against a white background on a computer screen placed at a distance of 60 cm from the participant. Each trial started with the onset of a centrally presented fixation dot, subtending 0.3°, which remained on the screen until the participant's response was registered. Five hundred milliseconds after fixation onset, a five-letter array (HHHHH, SSSSS, HHSHH, or SSHSS) was presented for 100 msec in such a way that the central letter of the array was placed approximately 0.7° below the fixation dot. The angle subtended by the whole array was approximately 2.8° × 0.6°. The response was followed by a blank screen for 500 msec, after which a new trial was initiated.

Design and Procedure. Participants received 100 practice trials before entering the experimental phase, which consisted of eight blocks of 100 trials each. Each block contained 25 trials with each of the four possible stimulus arrays. Presentation order of the stim-

ulus arrays was randomized. Participants were instructed to respond to one of the two target letters (central H or S) with one hand and to the other target letter with the other hand. The assignment of responding hand to target letter was consistent for each participant and counterbalanced across participants. A distinction was made between congruent (i.e., target letter and flankers associated with the same response, e.g., HHHHH) and incongruent (i.e., target letter and flankers associated with different responses, e.g., HHSHH) stimulus arrays. Participants were instructed to maintain an average level of accuracy of 80%–85%. If necessary, participants were verbally encouraged at the end of each block to increase or decrease their speed of responding. There were short breaks between successive blocks.

Psychophysiological recording. An ECI electrocap was used to obtain EEG recordings from electrodes placed at Fz, Cz, Pz, C3′, and C4′. These recordings were referenced to the activity recorded at an electrode on the left earlobe. The electro-oculogram (EOG) was recorded from tin electrodes placed above and below the left eye (vertical EOG) and from electrodes lateral to each eye (horizontal EOG). A ground electrode was positioned on the forehead. All electrode impedances were below 8 kΩ. The EEG and EOG signals were amplified using a Nihon-Kohden system with a time constant set to 5 sec and a low-pass filter of 35 Hz. The signals were digitized with a sample rate of 250 Hz. Single trial epochs with a duration of 2,048 msec (including a 200-msec prestimulus baseline) were extracted offline.

Data analysis. The single-trial EEG signals were corrected for EOG artifacts, using the algorithm described by Woestenburg, Verbaten, and Slangen (1983), and filtered with a bandpass of 1–10 Hz. A baseline, computed as the average signal activity across the 200 msec prior to stimulus onset, was subtracted for all single trials. Then, for each participant and each condition, the EEG epochs were averaged both with respect to stimulus onset and response onset to obtain stimulus-locked and response-locked ERPs. The amplitude of the ERN was defined as the difference between the most negative value in a window of 0–150 msec following the incorrect response and the mean value of the signal in a window of 200–100 msec preceding the response (i.e., the preresponse baseline) in the response-locked ERPs. Trials with RTs < 150 msec and trials with recording artifacts were not included in the analyses (younger adults: 2.8%; older adults: 0.9%). Performance measures (accuracy and correct RT) for each participant were submitted to a 2 (age group) × 2 (congruency) mixed analysis of variance (ANOVA). ERN amplitudes for each participant were submitted to a 2 (age group) × 2 (congruency) × 3 (electrode site: Fz, Cz, Pz) mixed ANOVA.

Results

Behavioral results. Averaged across the two congruency conditions, error rates were 17.8% (SD = 2.4%) for the younger adults and 14.8% (SD = 4.3%) for the older adults, a difference that was significant [$F(1,30) = 6.0$, $p < .05$]. As was expected, error rates were larger in the incongruent (M = 24.4%, SD = 4.3%) than in the congruent (M = 8.1%, SD = 2.2%) condition [$F(1,30) = 226.0$, $p < .001$]. The effect of congruency was similar for the two age groups: 9.0% versus 26.5% for the younger adults and 7.1% versus 22.3% for the older adults [$F(1,30) = 1.1$, $p = .29$].

The two groups differed from each other in mean correct RT. Mean RT was 353 msec (SD = 35 msec) for the younger adults and 419 msec (SD = 40 msec) for the older adults [$F(1,30) = 24.7$, $p < .001$]. RTs were shorter on congruent (M = 364 msec, SD = 22 msec) than on incongruent (M = 408 msec, SD = 27 msec) trials [$F(1,30) = 152.3$, $p < .001$]. This congruency effect was of similar size for younger (334 vs. 373 msec) and for older (395 vs. 444 msec) adults [$F(1,30) = 1.7$, $p = .21$].

The two groups also differed from each other in mean error RT. Mean error RT was 276 msec (SD = 29 msec) for the younger adults and 362 msec (SD = 50 msec) for the older adults [$F(1,30) = 35.8$, $p < .001$]. Error RTs were shorter on congruent (M = 308 msec, SD = 38 msec) than on incongruent (M = 331 msec, SD = 23 msec) trials [$F(1,30) = 14.9$, $p < .002$]. This congruency effect was of similar size for younger (262 vs. 289 msec) and for older (354 vs. 372 msec) adults [$F(1,30) = 0.6$, $p = .45$].

To evaluate possible age differences in posterror slowing, we compared RT as a function of accuracy on the previous trial. Across age groups, participants responded more slowly following an error than following a correct trial [M = 410 vs. 391 msec; $F(1,30) = 13.4$, $p < .001$], but although there was a trend for increased posterror slowing in the older age group, this trend was not significant [younger: 365 vs. 356 msec; older: 455 vs. 426 msec; $F(1,30) = 3.9$, $p = .058$].

ERN amplitude. The number of errors on congruent trials was too small to obtain reliable ERPs for 3 of the older participants. Therefore, these participants were excluded from the following psychophysiological analyses.

Figure 1 shows response-locked ERPs at Fz, Cz, and Pz for congruent and incongruent trials for the two age groups. As is clearly visible, the younger and older adults showed a negative deflection—the ERN—around the time of the incorrect response. The main effect of electrode site was significant [$F(2,54) = 48.3$, $p < .001$], indicating that the ERN had a frontocentral scalp distribution. Because the ERN overlapped the leading slope of the P3—as is most evident at Pz—the preresponse baseline correction contributed to positive ERN amplitudes in some conditions. Importantly, as in previous studies, ERN amplitude—averaged across the two congruency conditions—was smaller for older adults (.3 μV) than for younger adults (−2.4 μV), as confirmed by a significant main effect of age group [$F(1,27) = 7.4$, $p = .011$]. The ERN was somewhat more pronounced after congruent errors (−1.3 μV) than after incongruent errors (−.8 μV), but this difference was not significant [$F(1,27) = 2.5$, $p = .12$],[1] nor was the interaction between age group and congruency ($p > .8$). None of the interactions involving the factor electrode site were significant.

Following Falkenstein, Hoormann, and Hohnsbein (2001), we also tried to measure the ERN at the single-trial level. By obtaining single-trial measures of the ERN, we could test the possibility that the reduced ERN amplitudes of the older adults are due to a larger latency jitter of the ERN across single-trial epochs. Thus, the ERN in the average ERP waveform might be smaller, whereas the true ERN amplitude might not be reduced in older adults. We selected all incongruent, incorrect trials and subjected them to the same algorithm as that used for determining the ERN in the individual ERP waveforms. For younger and older groups, the distributions of the latencies associated

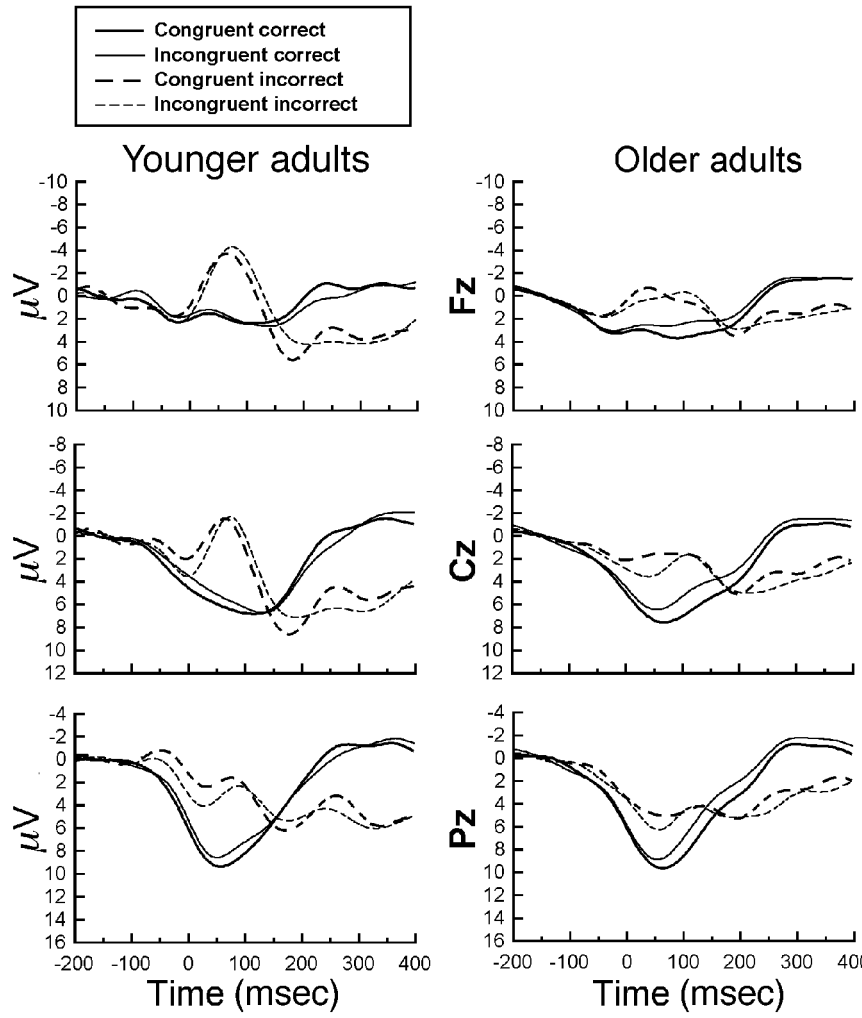

Figure 1. Response-locked, grand-average ERP waveforms from Experiment 1 as a function of accuracy and congruency for younger adults (left panel) and older adults (right panel). Time at 0 msec indicates the timing of the response.

with the "ERN" peak amplitudes selected by the algorithm suggested that the algorithm was unsuccessful. For both age groups, the algorithm selected many "ERNs" at the extreme ends of the specified time window. One reason for this may be that the ERN was often superimposed on the leading or trailing slope of the P3, which biased the most negative signal value in the specified time window toward one end of the window. Apart from these boundary selections, the distribution of younger adults was fairly humped, with a mode around 75 msec following the response. However, the distribution of the latencies for older adults was essentially flat, suggesting that for older adults the ERN did not generally exceed the random background EEG fluctuations, causing the algorithm to select noise. Thus, at least with the present type of algorithm and our data set, it is not possible to reliably measure the ERN in the single-trial epochs recorded from older adults. Nevertheless, if we assume that the amount of EEG noise is comparable in both age groups, then these findings may be an indication that the single-trial ERNs were smaller for older adults.

Other ERP components. Figure 2 shows stimulus-locked ERP waveforms at Fz, Cz, and Pz for both age groups as a function of congruency, for trials when the response was correct. Both age groups exhibit a rather standard stimulus-locked ERP waveform, with clearly recognizable N1, P2, N2, and P3 components. Visual inspection of the waveforms for younger and older participants indicates that age differences in the early ERP components (N1, P2, and N2) were small or absent. Furthermore, the P3 component showed a more diffuse midline distribution for older adults than for younger adults, with older adults' P3 being relatively smaller at Pz and relatively larger at Fz (see Kok, 2000).

To evaluate the reliability of these observations, we submitted the individual peak amplitudes in the latency range of the N1 (70–120 msec), P2 (130–180 msec), and

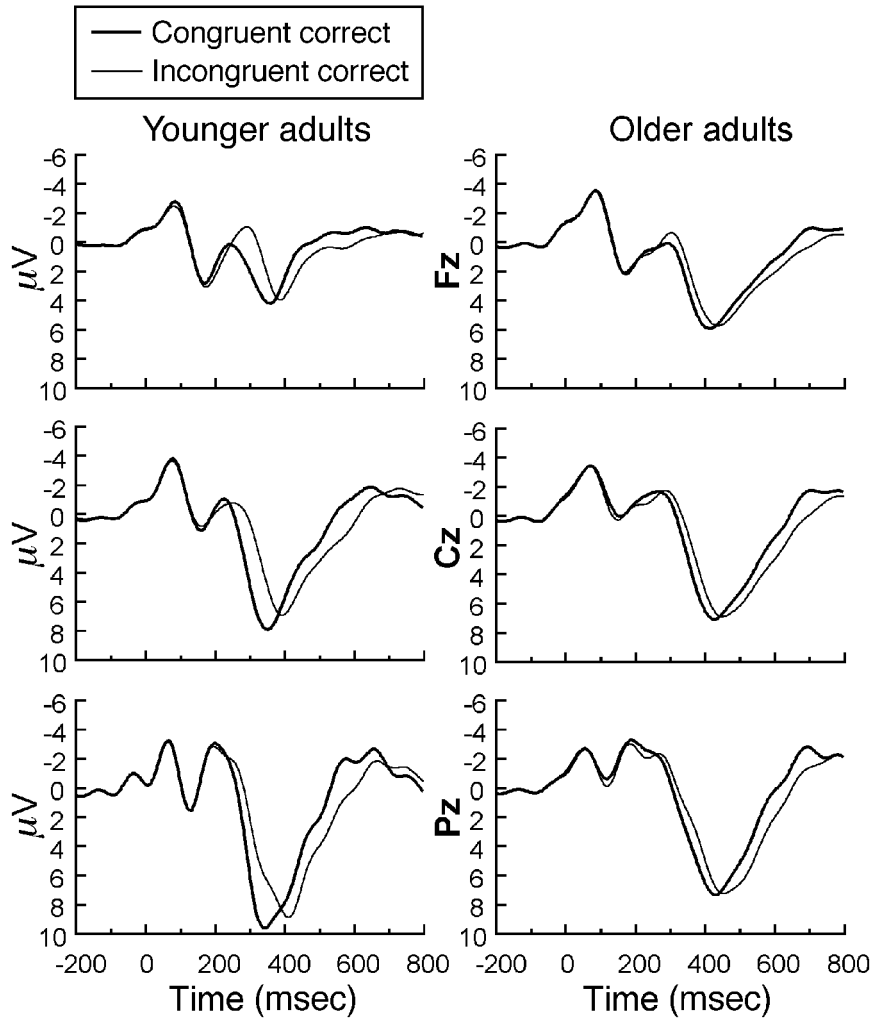

Figure 2. Stimulus-locked, grand-average ERP waveforms from Experiment 1 as a function of congruency for younger adults (left panel) and older adults (right panel). Time at 0 msec indicates the timing of the stimulus onset.

P3 (300–500 msec) to three separate 2 (age group) × 2 (congruency) × 3 (electrode site) mixed ANOVAs. Importantly, none of the main effects of age group were significant [N1, $F(1,27) = 0.00$, $p = .99$; P2, $F(1,27) = 3.5$, $MS_e = 25.5$, $p = .07$; P3, $F(1,27) = .02$, $p = .89$]. As expected, the interaction effect of age group and electrode site on P3 amplitude was highly significant [$F(2,54) = 13.0$, $p < .001$]. An additional ANOVA indicated that the frontal congruency effect in the N2 latency range (250–400 msec; measured as the mean value of the difference signal at Fz) was not significantly different between younger and older adults [$F(1,27) = 2.4$, $MS_e = 0.7$, $p = .13$].

Discussion

Experiment 1 yielded three important results. First, we replicated the empirical phenomenon of interest: reduced response-ERN amplitudes in older adults (Band & Kok, 2000; Falkenstein, Hoormann, & Hohnsbein, 2001). Second, Experiment 1 allowed us to exclude the possibility that the smaller ERN in our older adults was related to an impaired representation of the desired response—for instance, because of uncertainty about the presented stimulus (see Band & Kok, 2000). Scheffers and Coles (2000) have shown that ERN amplitude is positively related to error awareness when the degree of certainty about the required response varies due to data limitations. Thus, the smaller ERN of older adults in the incongruent flanker condition could, in principle, be due to uncertainty about the target letter, invoked by the presence of the distracting, incongruent flanker letters. However, in contrast to the results from two previous aging studies using the letter version of the Eriksen flanker task (Zeef & Kok, 1993; Zeef, Sonke, Kok, Buiten, & Kenemans, 1996), our older adults did not show larger interference effects (on RT and error rate) of incongruent flankers than our younger adults. Note that if older adults were more sensitive to the data

limitations associated with the incongruent condition than younger adults, this should, presumably, have led to increased interference effects for older adults. Perhaps even more convincing is the finding of a similar age-related reduction in ERN amplitude in the congruent condition, in which confusion about the presented target was unlikely since target and flankers were identical. These results and those of Falkenstein, Hoormann, and Hohnsbein (2001) suggest that smaller ERNs in older adults, at least in regular RT tasks, are not related to a decreased certainty about the required response.

Third, consideration of the average stimulus-evoked ERP waveforms suggests that the smaller ERN of older adults stands out as a specific age-related effect. That is, the reduction in ERN amplitude was not accompanied by a general age-related amplitude reduction of other ERP components. Falkenstein, Hoormann, and Hohnsbein (2001) reported a similar finding but presented no stimulus-locked ERPs. Our findings are also consistent with the psychophysiological aging literature. On the basis of a review of this literature, Kok (2000), for instance, noted that P1 and N1 components to stimuli presented in the auditory, visual, and somatosensory modalities are consistently *larger* in older than in younger participants. In line with our data, Kok also observed that P3s tend to have a more diffuse or equipotential midline distribution in older than in younger subjects. The absence of a general decrease in older adults' ERP component amplitudes reinforces the notion that their smaller ERN has a specific cause. Experiment 2 was designed to investigate this specific cause.

EXPERIMENT 2

Experiment 2 was conducted to test a hypothesis that could account for the smaller response ERN in older adults. This hypothesis, inspired by Holroyd and Coles's (in press; see also Holroyd et al., 1999) neurobiological theory of error processing, was tested using a slightly changed version of the probabilistic learning task designed by Holroyd and Coles. In this task, participants were required to press one of two buttons in response to each of a series of stimuli. The participants were told to infer the stimulus–response mappings by trial and error, using the information provided by a positive or negative feedback stimulus presented at the end of each trial. A critical aspect of the task was that the six possible stimuli differed in the degree to which the response was predictive of the value of the feedback (50%, 80%, or 100%). Thus, for some stimuli, the value of the feedback was uncorrelated with the selected response, whereas for other stimuli, the participant could, to varying degrees, learn to control the value of the feedback by acquiring the stimulus–response mapping. Holroyd and Coles formalized their theory in a process model of performance in this task. By using this model, which simulates response accuracy and both response-ERN and feedback-ERN amplitudes in the probabilistic learning task, we were able to test specific predictions derived from our hypothesis. Before we discuss this hypothesis and its predictions, we introduce Holroyd and Coles's theory.

According to Holroyd and Coles's (in press) theory, the basal ganglia learn to predict the expected outcome—in terms of reward and punishment—associated with various events (e.g., stimuli, responses, or stimulus–response ensembles) on the basis of past experience with those events. Violations of these predictions lead to a phasic alteration in firing rate in the mesencephalic dopamine system: a phasic increase if ongoing events are better than predicted, and a decrease if they are worse than predicted. This dopaminergic error signal serves as a reinforcement-learning signal that is used by the basal ganglia to improve the quality of its predictions. It does so according to the "temporal difference learning" algorithm (Sutton & Barto, 1998), a reinforcement learning rule for learning the earliest predictors of future reward or punishment. However, the dopaminergic error signal is also projected to the ACC, which acts as a motor control filter, enabling one of several neural command structures (e.g., dorsolateral prefrontal cortex, orbitofrontal cortex, basal ganglia) projecting to the ACC to take command of the motor system. The error signal is used to train the ACC to recognize the command structure that is best suited to take control of the task at hand. The various neural command structures themselves may also utilize the signal to reinforce the appropriate response strategy associated with each stimulus.

The ERN is assumed to be associated with the arrival of a negative dopaminergic error signal at the ACC. The larger the violation of the prediction, the larger the signal, and the larger the ERN. Thus, if ongoing events are suddenly worse than expected (e.g., because of an incorrect response to a stimulus that usually elicits the correct response), the mesencephalic dopamine system carries a negative reinforcement-learning signal to the ACC, where it elicits the ERN. Feedback ERNs are elicited when the negative feedback stimulus itself is not (or is only partly) predicted by earlier events. This would be the case when the stimulus–response mappings have not yet been learned and hence the negative feedback cannot be predicted on the basis of the response. However, as the system gradually learns the mappings, the phasic dopaminergic activity "propagates back in time," and is elicited as soon as an incorrect response is executed.

The neural network model implementing this theory consists of several modules (Figure 3): (1) an "adaptive critic," corresponding to the basal ganglia, which associates a value with the ongoing events and outputs a "temporal difference" (TD) error, corresponding to a phasic alteration of activity in the mesencephalic dopamine system, when ongoing events are better or worse than expected. The adaptive critic uses the TD error to refine its future predictions; (2) a set of "motor controllers," corresponding to various neural command structures in the brain, which serve a response selection function and use the TD error signal to reinforce response selections that elicit reward; (3) a "control filter," corresponding to the ACC, which delegates con-

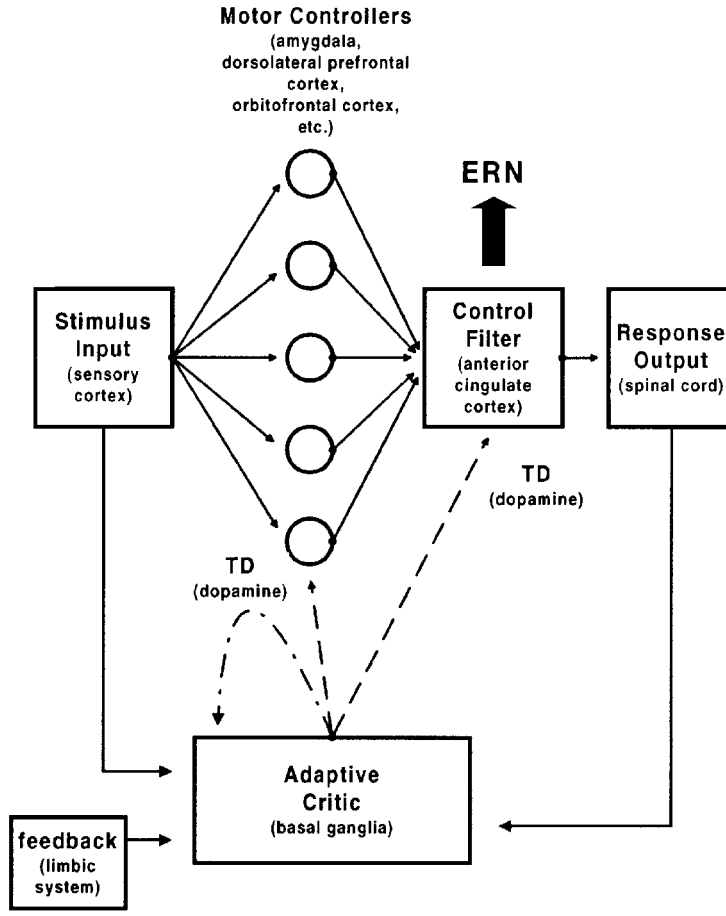

Figure 3. A schematic representation of Holroyd and Coles's (in press) model. Arrows indicate the flow of information between the various modules. The corresponding neural substrate is given in parentheses below each module label. A description of the model is provided in the text. TD, temporal difference.

trol to one of the motor controllers, and is trained by the TD error signal to select the controller that maximizes reward; and finally (4) a set of modules for representing stimulus input and feedback input and for executing the response. Details of the model are described in the Appendix (see also Holroyd & Coles, in press).

We generated a hypothesis regarding why the response ERN is smaller in older adults. According to this hypothesis, older age is associated with a weakened mesencephalic dopamine signal in response to negative (but also positive) violations of the basal ganglia's predictions. This "dopamine hypothesis" can be simulated by a proportional decrease of the TD error at the time of its computation, and is grounded in a substantial neurobiological literature documenting age-related decreases in the efficiency of dopaminergic function (see General Discussion; see also Braver et al., 2001; Li & Lindenberger, 1999). Figure 4 illustrates the predictions of the dopamine hypothesis by plotting, in one graph, simulated data from a "younger" model with an intact TD error and data from an "older" model with a proportionately decreased TD error. When the weakened dopamine signal arrives at the ACC, it should result in a reduced ERN amplitude, irrespective of whether the signal is elicited by negative feedback or by an incorrect response. These predictions of the dopamine hypothesis are illustrated in Figure 4A for the 50% condition, in which the ERN is generated only after negative feedback, and in Figure 4B for the 100% condition, which shows the expected tradeoff, in the course of learning, between the amplitudes of the response ERN and the feedback ERN. Note that the prediction of a smaller feedback ERN for older adults has not been tested in previous studies. The smaller reinforcement-learning signal should also result in reduced response accuracy in the probabilistic learning task, since actions that elicit reward (punishment) are positively (negatively) reinforced to a lesser extent. This prediction of the dopamine hypothesis is illustrated in Figure 4C, which shows simulated learning curves in the 100% condition. Note that reducing the strength of the reinforcement-learning signals should affect the steep-

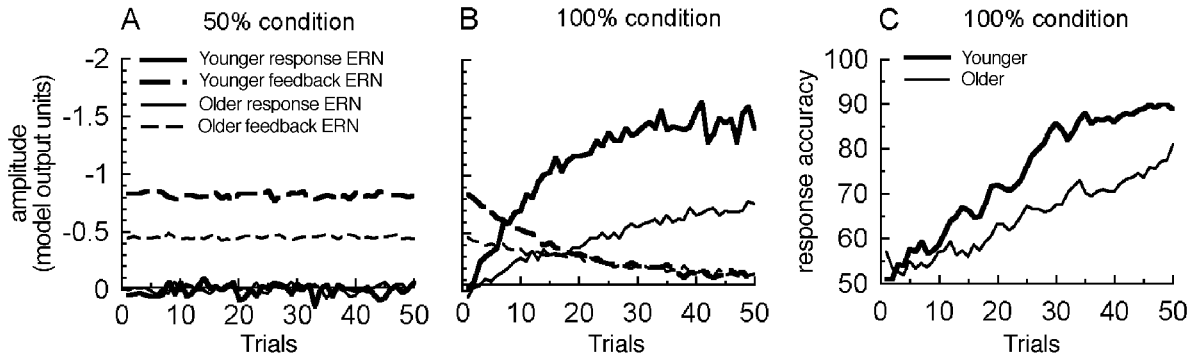

Figure 4. Simulated ERN (A and B) and response accuracy (C) data plotted by trial within block, meant to illustrate the predictions of the dopamine hypothesis. The patterns show the effect of decreasing the temporal difference (TD) error in the "older" model by comparing this model with a "younger" model with an intact TD error. The parameters of the younger and older models used to generate these data were the same as those used to fit the empirical data (see Model Simulations). The abscissa indicates the number of repeated presentations of a single stimulus from the indicated condition.

ness of the learning curve (i.e., the learning rate) but not its asymptote. But if, as in Experiment 2, accuracy is probed before asymptotic performance has been reached, then accuracy should be reduced compared with a system with intact reinforcement-learning signals.

Thus, the primary purpose of Experiment 2 was to test the dopamine hypothesis by measuring performance and ERNs from younger and older adults performing the probabilistic learning task and by fitting "younger" and "older" versions of the model to the data. Another purpose of Experiment 2 was to evaluate whether we could replicate the pattern of results for younger adults reported in Holroyd and Coles (in press, Experiment 1). Consistent with their theory, they observed that the probability of a correct response in a specific condition was, in general, positively related with response-ERN amplitude and negatively related with feedback-ERN amplitude.

Method

Participants. The participants were the same as in Experiment 1. In addition to a fixed payment (see Experiment 1, Method), all participants received a performance-related bonus, as described below.

Stimuli. Stimuli were presented in color against a white background on a computer screen placed at a distance of 60 cm from the participant. Each experimental block involved a new set of six imperative stimuli. These stimuli were images of buildings, animals, musical instruments, and so on. Images of a head of lettuce and of a carrot served as feedback stimuli indicating to the participant that they were rewarded or penalized on that trial. The mappings between reward/punishment and the feedback stimuli were counterbalanced across participants and kept fixed across the experiment. A different feedback stimulus, an image of a cherry, was presented in case a response deadline was missed. All stimuli were part of a public Corel image library and were scaled to a uniform size so that they subtended approximately 9.5° × 9.5°.

Design and Procedure. On each trial, the stimulus events consisted of the presentation of an imperative stimulus for 500 msec, followed by a blank screen for 500 msec, followed by the presentation of a feedback stimulus for 500 msec, followed by a blank screen for 500 msec. Thus, the interval between consecutive imperative stimuli was 2 sec. Participants were required to make a two-choice decision by pressing one of two buttons within 700 msec after the onset of the imperative stimulus. The response deadline was introduced to ensure that participants made some errors due to premature responding in the 100% mappings even after the mappings had been learned. If a response exceeded the deadline, the cherry stimulus communicated to the participant that he/she was penalized 4 cents on that trial, providing motivation for him/her to respond more quickly. Otherwise, the feedback stimulus indicated to the participant that he/she had either earned or was penalized 2 cents of bonus money on that trial.

Participants were not informed about the appropriate stimulus–response mappings but were told to infer these mappings by trial and error and to respond in such a way as to increase their bonus by as much as possible. In each block, one of the six stimuli was mapped to the left button, so that participants were rewarded if they pressed the left button and penalized if they pressed the right button. Another stimulus was mapped to the right button in a similar fashion. Following Holroyd and Coles (in press), we refer to these mappings as the *100% mappings*. For two other stimuli, feedback was delivered randomly, irrespective of the given response. As a result, participants were rewarded on 50% of the trials and penalized on 50% of the trials, and these mappings are therefore called the *50% mappings*. On the same logic, the two remaining stimuli were associated with an *80% mapping*.[2] That is, one stimulus required a left buttonpress on 80% of the trials (referred to as *valid* trials) and a right buttonpress on 20% of the trials (*invalid* trials), and the other stimulus required a right buttonpress on 80% of the trials (valid) and a left buttonpress on 20% of the trials (invalid). Like Holroyd and Coles, we also examined how performance and ERN amplitudes changed over the course of a block of trials. We analyzed such learning effects by comparing the dependent variables in the first and second halves of each block.

The experiment involved five blocks of 300 trials each. Each of the six stimuli was presented 50 times in a random order. Valid and invalid trials from the 80% mappings were randomly intermixed, the only restriction being that the first 25 trials with each of the two stimuli contained 5 invalid trials. Before the experimental phase, participants received written instructions and performed one practice block of 300 trials. Before each experimental block, participants were given the opportunity to study the six imperative stimuli used in the upcoming block and to press a key to start the block when they were ready. At the end of each block, participants were informed about the total amount of bonus money they had earned throughout the experiment. Participants began the experiment with a bonus of HFL 2.50.

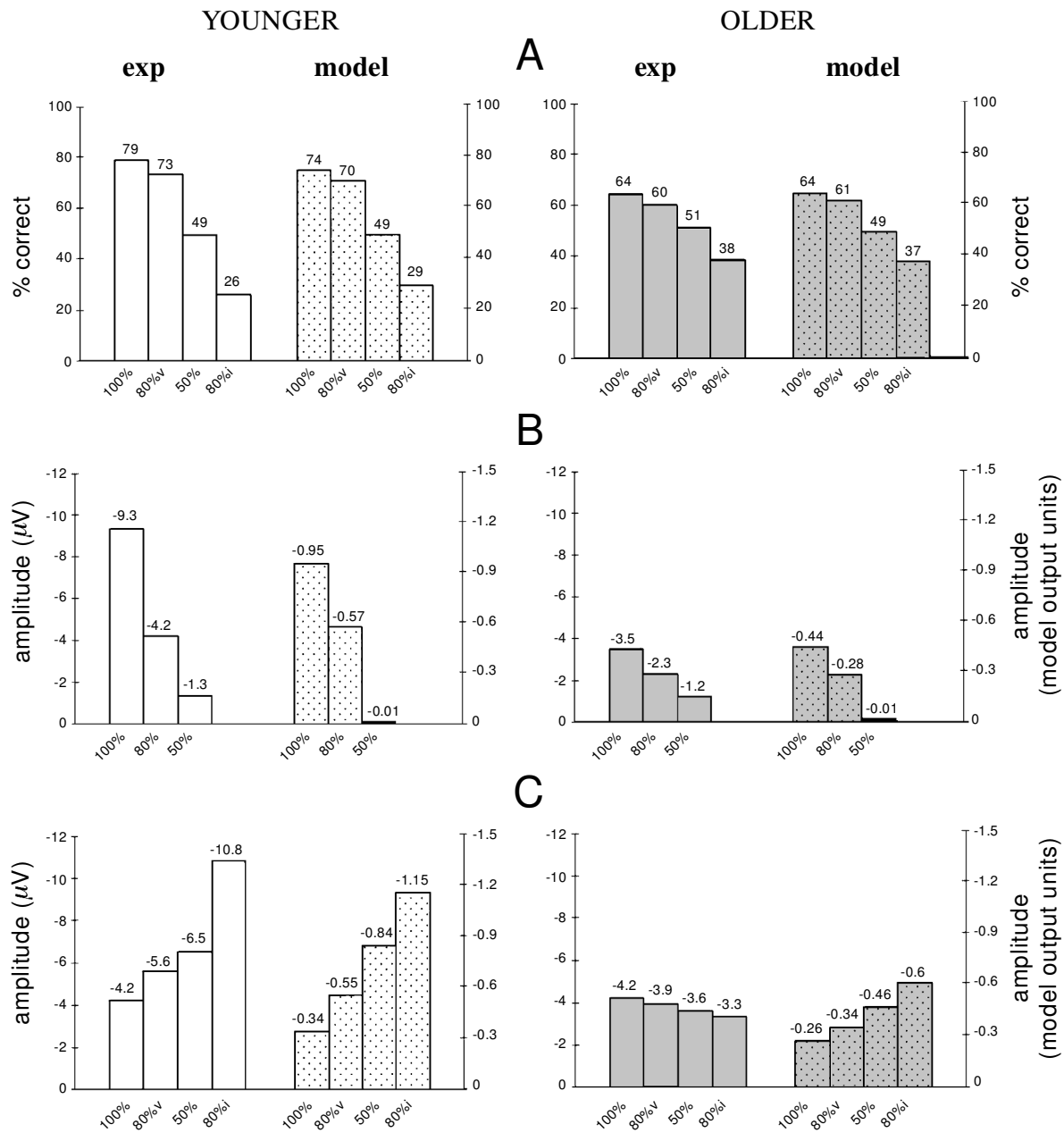

Figure 5. Simulated (model) and empirical (exp) results in Experiment 2 as a function of condition for the younger adults/model (left panel) and older adults/model (right panel). (A) Response accuracy. (B) Response-locked and (C) feedback-locked ERN amplitudes. Amplitudes (in microvolts for the empirical results; in model output units for the simulated results) reflect the subtraction of amplitude magnitudes associated with negative and positive feedback (see text for more detail). 80% v, 80% valid condition; 80% i, 80% invalid condition.

Psychophysiological recording. Details are the same as in Experiment 1. In this experiment, we also collected cardiovascular measures from the younger participants. These data will be reported elsewhere (van der Veen, Nieuwenhuis, van der Molen, Crone, & Jennings, 2001).

Data analysis. The single-trial EEG signals were corrected for EOG artifacts, using the algorithm described by Woestenburg et al. (1983), and filtered with a bandpass of 1–10 Hz. A baseline, computed as the average signal activity across the 300 msec prior to stimulus onset, was subtracted for all single trials. Then, for each participant and each condition, the EEG epochs were averaged with respect to response onset and feedback onset to obtain response-locked and feedback-locked ERPs. The 100 msec preceding feedback onset served as a baseline for the feedback-locked ERPs. Following Holroyd and Coles (in press), difference waveforms were created by subtracting the signal elicited on trials with positive feedback from the signal elicited on trials with negative feedback. The amplitude of the response ERN was defined as the peak negativity

Table 1
Experimental and Simulated Response Accuracy as a Function of Age Group, Condition, and Block Half

	Experiment		Model	
Condition	Younger	Older	Younger	Older
100%	76/83	60/69	62/ 85	57/70
80% valid	69/77	56/63	59/82	56/67
50%	50/48	51/51	49/48	49/49
80% invalid	27/25	42/34	42/15	42/34

Note—Numbers indicate percentage correct in the first/second block half.

of the difference waveform at electrode Cz in a window 0–150 msec following the response. The amplitude of the feedback ERN was defined as the peak negativity of the difference waveform at electrode Cz in a window 200–350 msec (as chosen after visual inspection) following feedback onset. The following trials were discarded from the reported analyses: (1) trials with recording artifacts or RTs < 150 msec—1.7% for younger adults and 2.5% for older adults; and (2) trials in which no response was generated before the 700-msec deadline—5.5% for younger adults and 8.2% for older adults. Performance measures, response-ERN, and feedback-ERN amplitudes for each participant were submitted to separate mixed ANOVAs with age group (younger, older) as a between-subjects factor and condition (100%, 80% valid, 50%, and 80% invalid mappings) and block half (Trial 1–150, Trial 151–300 of each block) as within-subjects factors.

Results

We first describe the empirical behavioral results and ERN amplitudes found for our younger and older participants. After that, we discuss the results of the simulations associated with the dopamine hypothesis. We also consider the possibility of simulating the effects of aging by changing other model parameters.

Behavioral results. Figure 5A shows behavioral accuracy for younger and older adults for each condition, averaged across the first and second halves of each block. As can be seen, the younger adults displayed higher accuracy than the older adults in the conditions in which performance could improve over the course of a block. The lower-than-chance performance in the 80% invalid trials indicates that participants responded mostly according to the dominant (but here incorrect) mapping. The main effects of age group and condition and the interaction between these two factors were all highly significant (all Fs > 17.5, ps < .001).

As expected, participants' accuracy performance in the second block half showed an improvement over the first block half in both the 100% condition and the 80% valid condition (Table 1). Because participants gradually learned the dominant 80% mapping, response accuracy in the 80% invalid condition decreased as a function of block half. Evidently, accuracy in the 50% condition did not change with block half. The interaction between condition and block half was highly significant [$F(3,90) = 26.5, p < .001$] but similar for both age groups [three-way interaction: $F(3,90) = 2.1, p = .13$].

Probably as a result of the use of a response deadline, older adults' RTs—averaged across all conditions—were

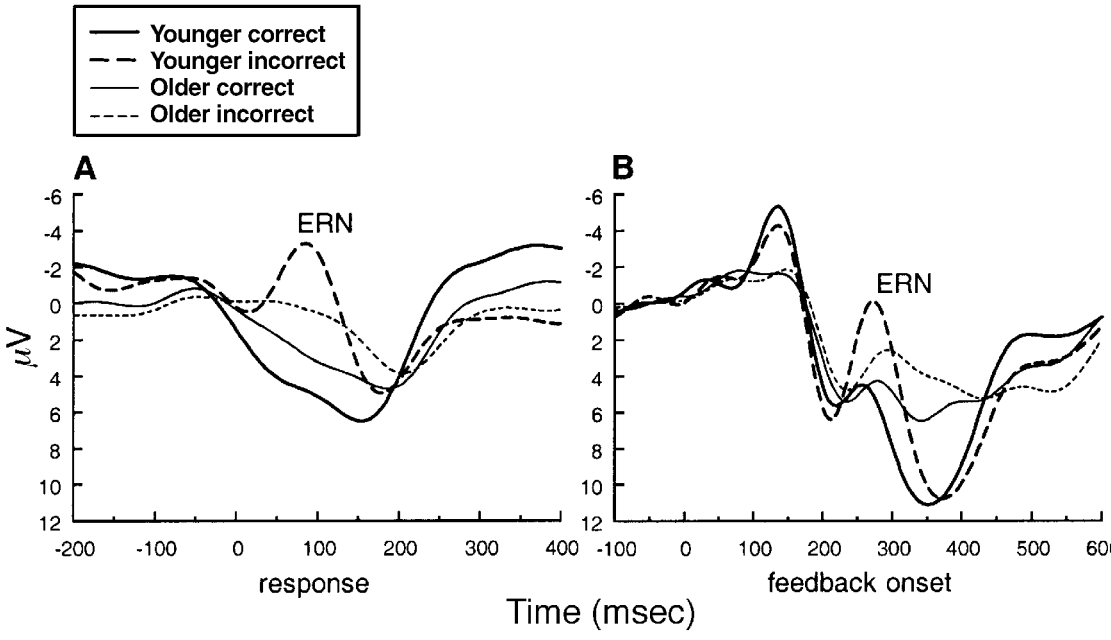

Figure 6. Grand-average ERP waveforms for younger and older adults from two conditions in Experiment 2. Waveforms were recorded from Cz. (A) The ERN elicited by the response in the 100% condition. Time at 0 msec indicates the timing of the response. (B) The ERN elicited by the feedback in the 50% condition. Time at 0 msec indicates the timing of the feedback onset.

not much slower than younger adults' RTs (younger: 387 msec; older: 406 msec), as confirmed by a nonsignificant main effect of age group [$F(1,30) = 2.9$, $p = .099$].

ERN amplitude. Figure 6 shows illustrative ERPs, associated with positive and negative feedback, from two conditions in Experiment 2, for both age groups and averaged across participants and block halves. Figure 6A shows the ERNs elicited by the response in the 100% condition, the condition in which the response ERN is largest for both younger and older adults. The ERN of younger adults is evident as a clear negativity peaking about 80 msec after the response. Importantly, as in Experiment 1, the average older adults' response ERN is substantially reduced. Figure 6B shows feedback ERNs in the 50% condition, the only condition in which feedback is not dependent on the given response. In this condition, the ERN is elicited primarily by the feedback. For younger adults, the ERN peaked roughly 270 msec after the presentation of a negative feedback stimulus (see Holroyd & Coles, in press; Miltner et al., 1997). The ERN of older adults peaked somewhat later, and, importantly, was clearly smaller.

Figure 5B shows response-ERN amplitudes as a function of age group and condition. Note that valid and invalid trials from the 80% condition were pooled, because for the participants these were not distinguishable until the presentation of the feedback stimulus. Response-ERN amplitudes were smaller for the older adults [$F(1,30) = 21.7$, $p < .001$]. Consistent with Holroyd and Coles's (in press) theory, response-locked amplitudes increased with the probability of a correct response in a certain condition [$F(2,60) = 53.3$, $p < .001$], and this effect of condition was more pronounced for younger than for older adults [$F(2,60) = 16.3$, $p < .001$]. Note that the "true" response ERN in the 50% condition should be absent. The negative values reported for this condition in Figure 5B reflect unsystematic fluctuations of the ERPs associated with negative and positive feedback.

Figure 5C shows feedback-ERN amplitudes as a function of age group and condition. Here, the difference between valid and invalid trials from the 80% condition is clearly important; as predicted by Holroyd and Coles's (in press) theory, the largest feedback ERNs, at least for younger adults, were observed in the 80% invalid condition, in which a clear expectation of positive feedback was violated by the actual feedback. Averaged across conditions, feedback-ERN amplitudes were smaller for older adults [$F(1,30) = 9.9$, $p < .005$]. The main effect of condition was also significant [$F(3,90) = 4.9$, $p < .03$], but whereas younger adults showed a pronounced effect of condition in the predicted direction (i.e., an inverse relation between the ERN and the probability of giving a correct response), the older adults showed, if anything, a small trend in the opposite, unexpected direction. This was expressed in a significant interaction of age group and condition [$F(3,90) = 8.4$, $p < .005$]. An additional analysis including only the older participants indicated that the surprising effect of condition for the older adults was not significant [$F(3,45) = 1.5$, $p = .25$]. Inspection of the older individuals' data patterns revealed that the absence of an effect of condition in the expected direction was consistent across participants.

Although we did not explicitly model block-half effects on ERN amplitudes, these effects were generally in line with Holroyd and Coles's (in press) theory. A separate statistical analysis showed that response-ERN amplitudes increased with block half [$F(1,30) = 19.0$, $p < .001$]. This block-half effect increased with the probability of giving a correct response in a particular condition [$F(2,60) = 13.2$, $p < .001$], and was somewhat larger for younger adults than for older adults [$F(1,30) = 4.0$, $p = .056$]. As expected, feedback-ERN amplitudes decreased with block half in the conditions in which accuracy improved, and increased in the 80% invalid condition, in which accuracy declined. However, the interaction of condition and block half showed only a nonsignificant trend in this direction [$F(3,90) = 2.3$, $p = .096$], probably reflecting the fact that the block-half effects on feedback-ERN amplitude were rather small. Age group did not enter in a reliable interaction with condition and block half [$F(3,90) = 1.0$, $p = .40$].

Model simulations. Apart from a slight adjustment of one learning rate parameter (see the Appendix), the younger model, which served as a starting point for our simulations of age-related effects, was identical to the model developed by Holroyd and Coles (in press, Experiment 1). To simulate the data from the 16 younger and 16 older human participants, both the "younger" and "older" models were run 16 times, with each run consisting of five blocks of 300 trials. Like the empirical ERN, the model ERN was determined by subtracting the simulated ERN on trials with positive feedback from the simulated ERN on trials with negative feedback.

To provide support for the dopamine hypothesis, we tried to simulate the empirical results of older adults by weakening the TD error signal. On each time step in the simulated trials, we multiplied the older adults' TD error by a factor of 0.55 immediately after its computation and *before* it was used to adapt the network weights and compute the ERN. It is important to note that this proportional decrease of the TD error was the only aspect in which the older model differed from the younger model. Figure 5 shows the simulation results associated with the dopamine hypothesis. Because the variance of the model data was relatively small, all statistical terms in the ANOVAs were highly significant ($p < .001$) for each of the measures, and we will report no further statistics.

As was the case for the human participants, the model's response accuracy (Figure 5A) was relatively high in the two conditions that benefited from learning and low in the 80% invalid condition, which suffered from learning. Overall accuracy and—more important—the effects of condition were smaller for the older model than for the younger model. The model's performance in the 100% condition and 80% valid condition improved as the blocks progressed, and performance in the 80% invalid condition got worse over the course of a block of trials (Table 1). Although more pronounced, these learning effects were in the same direction as those in the empirical data.

The simulated pattern of response ERNs (Figure 5B) was also very similar to the empirical data pattern. Response-ERN amplitudes showed a positive correlation with learning and were smaller for the older than for the younger model. The simulated pattern of feedback ERNs (Figure 5C) captured the important finding that feedback-related ERN amplitudes were considerably reduced in the older participants. The model also reproduced the negative correlation between feedback-ERN amplitude and learning portrayed by the younger adults, but the model did not (and cannot!) account for the absence of such a negative correlation in the older adults' data. In the Discussion, we will offer a possible explanation for this discrepancy.

The success of a proportional decrease of the TD error in accounting for the age differences in accuracy and ERN amplitude does not exclude the possibility that a manipulation of other model parameters may equally well account for these differences. In a set of additional simulations, we explored the possibility of simulating the older adults' empirical data by decreasing the value of the learning rate parameters associated with the functions for TD-error-driven learning in various modules of the model. That is, the mesencephalic dopamine signal is intact, but the rate of adjustments that the signal brings about through its reinforcing qualities is smaller in older age. Not surprisingly, in these simulations, the slower learning rate led to a decrease in response accuracy. This, in turn, influenced the relative size of response-ERN and feedback-ERN amplitudes. More specifically, because of its slower learning rate, the model was less able to predict reward on the basis of the response, resulting in relatively small response ERNs. Conversely, because the feedback itself remained an important predictor of reward, the simulated feedback ERNs were *larger* than those produced by the younger model—a clear discrepancy with the empirical data. Thus, a manipulation of the learning rate parameters could account for the reduced response accuracy and smaller response ERNs but *not* for the smaller feedback ERNs in older adults.[3] Moreover, because the ERNs are abnormal solely because learning is retarded, this type of model predicts that both the response-ERN and feedback-ERN amplitude should return to normal once its learning curve has reached asymptote or has otherwise caught up with the normal (i.e., unretarded) model's learning curve. However, the finding that older adults may show a reduced response ERN in the context of normal performance (as in Experiment 1; see also Falkenstein, Hoormann, & Hohnsbein, 2001) seems clearly inconsistent with this prediction.

DISCUSSION

The general pattern of results in Experiment 2 was consistent with the dopamine hypothesis of altered error processing in older adults. Through manipulation of a single model parameter, corresponding to a weakening of dopaminergic error signals, we managed to attain a considerable correspondence between the simulated and empirical age differences: decreased response accuracy and smaller response and feedback ERNs in older adults. Because the model proposes that ERN amplitude is an increasing function of the size of the dopaminergic error signal, the smaller response and feedback ERNs of the older adults are consistent with the dopamine hypothesis. In addition, because the signal serves a reinforcement-learning function, the reduced signal in older adults led to impaired performance in situations in which learning was important. The empirical results cannot be explained by an alternative hypothesis, according to which older adults' dopaminergic error signals are intact but are not efficiently used for learning. Although, like the dopamine hypothesis, this hypothesis is able to account for the reduced response ERNs and lower accuracy in our older adults, it falsely predicts that older adults should have larger feedback ERNs than younger adults, and it cannot account for the pattern of results in Experiment 1.

An unexpected and robust aspect of the older adults' data was the absence of a condition effect on the feedback-ERN amplitude. That is, instead of an increase with the probability of an incorrect response in a condition, the feedback-ERN amplitudes showed a nonsignificant trend in the opposite direction. The lower-order error-processing mechanism employed by the model cannot account for such a pattern. One way of preserving the present theory, while accounting for the discrepancy between simulated and empirical data, is to assume that the amount of attention that older adults paid to the feedback stimulus was dependent on the subjective probability of an error. That is, when older adults were relatively sure that they had made an error—as would be the case after errors in the 100% and 80% valid conditions—then they may have wished to confirm this supposition and have paid relatively more attention to the feedback stimulus, thereby increasing its impact on the error-processing system. In contrast, attention for feedback in the 80% invalid condition may have been rather weak on error trials, since on these trials the participant responded according to the dominant (i.e., probably correct) stimulus–response mapping, leading older participants to ignore the feedback.

The pattern of results of younger adults in Experiment 2 replicated that reported by Holroyd and Coles (in press). Response-ERN amplitude was positively correlated with the probability of giving a correct response. That is, the response ERN was larger in conditions in which participants could learn to control the value of the feedback by learning the correct response, and it was larger in the second half of a block—that is, after some degree of learning had taken place. In line with these findings, Experiment 2 also showed that the response ERN was largest in the participant group that learned more efficiently. The opposite pattern, a negative correlation between ERN amplitude and the probability of a correct response, was observed for the feedback ERN. This tradeoff between response-ERN and feedback-ERN amplitude is a central issue in Holroyd and Coles's theory. The younger adults' data in the 80% condition (a condition not examined by Holroyd and Coles) also supported Holroyd and Coles's theory of error processing. First, as predicted by this theory, the amplitudes of the response ERN and feedback ERN in the 80% valid condi-

tion were intermediate between the ERN amplitudes in the 50% and 100% conditions. And second, the 80% invalid condition, which was characterized by the largest mismatch between expected and actual feedback, was associated with the largest feedback ERN.

GENERAL DISCUSSION

The present study was motivated by a recently proposed theory of the functional mechanisms and neural basis of error processing (Holroyd & Coles, in press). According to this theory, the ERN, a psychophysiological marker of error processing, is associated with the arrival of a negative dopaminergic reinforcement-learning signal in the ACC. This dopaminergic error signal is elicited when the brain first detects that ongoing events are worse than expected and is used to adjust the cognitive system for the task at hand. Previous studies (Band & Kok, 2000; Falkenstein, Hoormann, & Hohnsbein, 2001) have reported an age-related reduction in ERN amplitude following incorrect responses in choice RT tasks. In the present study, we have replicated this result and, more importantly, have proposed a dopamine account, based on Holroyd and Coles's theory, of why the response ERN is reduced in older adults. According to this account, the dopaminergic signals thought to play a role in error processing and reinforcement learning are weakened in the older brain. This leads to reduced ERNs in response to errors and negative feedback, and to slower reward-based learning in older adults.

In Experiment 1, we showed that the reduced response ERN of older adults cannot be understood as a reflection of a general attenuation of ERP components in older adults. Furthermore, Experiment 1 demonstrated that an age-related reduction in ERN amplitude can also be observed in situations that minimize data limitations (see Falkenstein, Hoormann, & Hohnsbein, 2001). This indicates that a reduced response ERN is not necessarily related to reduced certainty about the required response in older adults (see Band & Kok, 2000). In Experiment 2, we proposed a dopamine account of altered error processing in older age, and formalized this account using a model proposed by Holroyd and Coles (in press). We confirmed the predictions of the dopamine hypothesis with respect to age changes in performance and ERN amplitudes in a probabilistic learning task. Computer simulations indicated that the empirical pattern of age changes—reduced response accuracy and smaller ERNs following response errors and negative feedback—could be adequately simulated by manipulation of a single model parameter, corresponding to weakened phasic activity in the mesencephalic dopamine system. And finally, we argued that the results in Experiments 1 and 2 are inconsistent with another hypothesis derived from Holroyd and Coles's model—namely, that a slower learning rate constitutes the *primary* cause of age-related changes in error processing.

The dopamine hypothesis could not account for the finding that the older adults' feedback-ERN amplitudes showed no sensitivity to the degree to which the feedback in a particular experimental condition was uniquely predictive of reward. It is important to note that this finding is inconsistent with any model derived from Holroyd and Coles's (in press) theory. Thus, we proposed that an additional, higher order, cognitive process, masking the effects of the theorized lower order mechanism, may have been at work in the older adults' brains. We hypothesized that the amount of attention that older adults paid to the feedback stimulus may have been greater for conditions in which the subjective expectation of negative feedback was higher. As a result, the impact of negative feedback on the error-processing system, and hence on the feedback ERN, should be largest in conditions in which participants learned to recognize an incorrect response most adequately (i.e., the 100% condition and, to a lesser extent, the 80% condition). Future work is needed to test this hypothesis.

If older adults have a deficient error-processing system, this may have implications for the general issue of an executive control deficit in older age (for reviews, see Moscovitch & Winocur, 1995; Phillips & Della Sala, 1996; van der Molen & Ridderinkhof, 1998). Several authors (Botvinick et al., 2001; Coles et al., 2001; Gehring et al., 1993) have proposed that the activity giving rise to the ERN may signal the demand for increased control (e.g, increased focus of attention or more conservative response settings) to other brain systems. Thus, to the extent that older adults show impaired performance in tasks requiring executive control processes, it may be unclear whether such performance deficits are due to inefficiency of the executive control processes (as is usually concluded), to inefficiency in the monitoring processes necessary to recognize the need for executive control, or to both. This issue deserves more attention in future research.

Dopamine Dysfunction in Healthy Aging

Our proposal of weakened dopaminergic error signals in older adults is consistent with various reports of age-related disturbances in dopamine function. For instance, using positron emission tomography (PET), Backman et al. (2000; see also Volkow et al., 1998) found a gradual age-related deterioration of dopaminergic receptor binding in striatal structures. Importantly, statistical control of this variable eliminated the age-related variation in performance of a number of cognitive tasks, suggesting that dopaminergic transmission is an important factor in age-related cognitive decline. On the basis of another PET study, Kaasinen et al. (2000) reported a significant age-related loss of dopamine receptors in various brain areas, and especially in the frontal cortex. Furthermore, it has been shown that in monkeys, age-related decreases in neurotransmitter concentration are most pronounced for dopamine in the prefrontal cortex (see Goldman-Rakic & Brown, 1981). And Arnsten and colleagues (e.g., Arnsten, 1993; Arnsten & Goldman-Rakic, 1985) have shown that working memory deficits of aged monkeys can be alleviated by pharmacological agents enhancing the function of their dopamine system. Our simulations suggest that the dopaminergic error signal is weakened at the time of its generation—presumably in the basal ganglia—or between the time of its generation and its impact on other brain systems; in its

present form, our model cannot be used to discriminate between these possibilities or to determine the neurophysiological mechanism through which the signal is weakened. Irrespective of exactly how and when the signal is weakened, an implication of the dopamine hypothesis seems to be that increasing the concentration of dopamine in older adults should lead to a reduction of age-related differences in ERN amplitudes and reinforcement-learning abilities. However, as noted by Braver et al. (2001), such predictions are complicated by the notion that older adults have reduced dopamine receptor concentrations in various brain areas, which may prevent pharmacologic manipulations of dopamine levels from being fully effective.

Braver et al. (2001) have recently argued that the decrease in several working memory functions that occurs with healthy aging is a direct consequence of disturbances in dopamine function in the dorsolateral prefrontal cortex. They based their theory on the empirical confirmation of several predictions regarding age effects in a continuous performance test. The predictions were derived from a computational model of dopaminergic modulation of prefrontal cortex (Braver & Cohen, 2000) that is similar in some important respects to Holroyd and Coles's (in press) model of error processing. In Braver and Cohen's model, the mesencephalic dopamine system carries a temporal difference error to frontal areas of the brain. This phasic dopamine signal serves to regulate the access of information into and out of working memory and also has a reinforcement-learning function that allows the system to learn what information is relevant for the task at hand.

Apart from the purported role of dopamine in cognitive control deficits in older age, it has been hypothesized that disturbances in dopamine function may result in an overall noisier information processing system in older adults. Li and Lindenberger (1999) have reported a set of computational simulations that relate aging-induced deterioration of catecholinergic systems (including the dopaminergic system) to several benchmark phenomena of aging, such as increases in mean RT and interindividual variability of RTs. Such phenomena may occur as a result of a decreased responsivity of neurons to incoming information due to a reduction in catecholamines, which are thought to serve an important neuromodulatory role.

Abnormalities in Error Processing

An interesting question is the extent to which the present findings and conclusions regarding error processing in older adults may generalize to other populations with abnormal ERNs. In particular, reduced response-ERN amplitudes have been observed in patients with Parkinson's disease (Falkenstein, Hielscher et al., 2001; but see Holroyd et al., in press) and patients with schizophrenia (Ford, 1999; Kopp & Rist, 1999). Parkinson's disease is mainly characterized by cell loss in the substantia nigra leading to dopamine depletion in striatal and other brain structures. Schizophrenia has also been extensively documented as involving disturbances in dopamine function. This raises the possibility that, as we have proposed for older adults, the smaller ERN in these populations may be caused by a reduction in phasic dopaminergic error-related activity. The probabilistic learning task of Experiment 2 would be well suited to test these hypotheses because it allows the link between learning and the relative size of response and feedback ERNs, central in Holroyd and Coles's (in press) theory, to be studied in detail. Other tasks, such as the flanker task in Experiment 1, are perhaps less appropriate to test specific hypotheses regarding error processing in various populations because in these tasks learning tends to reach asymptote fairly quickly. As a result, any diagnostic correlational pattern of learning indices and ERN amplitudes is absent in data from such tasks.

Band and Kok (2000) observed not only a reduction in the response-ERN amplitude of older adults but also a substantial ERN-like negativity following *correct* trials (i.e., a CRN) similar to that observed in patients with frontal lesions (Gehring & Knight, 2000) and in schizophrenia patients (Ford, 1999; but see Kopp & Rist, 1999). An ERN-like wave on correct trials may also sometimes be observed for healthy younger adults, but Coles et al. (2001) have argued that this "CRN" either represents the influence of stimulus-evoked components in the response-locked ERP or can be attributed to error processing on correct trials—for instance, when participants have an incorrect representation of the required response (see Scheffers & Coles, 2000). The behavioral results of the mental rotation task used by Band and Kok suggest that their older participants were often uncertain about the presented stimulus, and hence the required response. Therefore, it is likely that the CRN of Band and Kok's older adults can be attributed to a subset of the correct trials on which they wrongly perceived their response as incorrect. This notion is supported by the absence of a CRN in the older adults in Experiment 1 of the present study and in the study by Falkenstein, Hoormann, and Hohnsbein (2001), two studies in which data limitations were relatively small.

Theories of the ERN

The strength of the dopamine hypothesis hinges on the appropriateness of Holroyd and Coles's (in press) model and on the proposed correspondence between elements of the model and neurobiological structures. With respect to the former, it is important to note that the data of the younger adults in the probabilistic learning task replicated those found by Holroyd and Coles. These data indicated that learning of stimulus–response associations (as affected by age group, block half, and condition) correlated positively with response-ERN amplitude and negatively with feedback-ERN amplitude. The resulting trade off between the response ERN and feedback ERN in the course of learning is central to Holroyd and Coles's claim that the ERN is elicited as soon as the expected consequences of ongoing events are worse than expected. The results from a new condition (the 80% condition) were also nicely in line with Holroyd and Coles's theory. In particular, these results showed that the ERN was largest when negative feedback was highly unexpected (i.e., in the 80% invalid condition). Our study only indirectly addresses the issue of how neurobiological structures map onto elements of

the model. However, the idea that the successful aging model (with a proportional decrease of the TD error) could easily be related to the neurobiological literature on age-related dopamine disturbance lends support to the proposed link between TD errors and dopamine signals (for a discussion, see Holroyd & Coles, in press).

Recently, Yeung, Botvinick, and Cohen (2001; Botvinick et al., 2001) have proposed an alternative theory of the response ERN. Yeung et al. argued that errors are usually associated with response conflict in the period following the erroneous response, the time window of the ERN. Rather than indexing erroneous response activation per se, the ERN reflects the neural response of the ACC when it detects such response conflict—that is, the concurrent activation of multiple conflicting responses. This signal is then used by other brain structures to reduce future conflict. Although the conflict-monitoring theory seems able to account for many of the same phenomena that mismatch theories of the ERN can explain (see Yeung et al., 2001), in its present form it does not incorporate a mechanism that can account for the existence of an ERN following negative feedback. Thus, although the conflict-monitoring theory may be able to address the response-related ERNs in this study, it is difficult to see how it would address our feedback-related ERN data. A direct comparison of Holroyd and Coles's (in press) theory and the conflict-monitoring theory is the subject of ongoing research (Holroyd, Yeung, Coles, & Cohen, 2002).

To summarize, on the basis of computational simulations inspired by Holroyd and Coles's (in press) theory of error processing, we have proposed an account of age-related changes in error processing. According to this account, older age is associated with weakened dopaminergic reinforcement-learning signals in response to errors and negative feedback, leading to smaller ERN amplitudes and slower reward-based learning. This dopamine hypothesis is consistent with a substantial literature reporting age-related disturbances in dopamine function. Several alternative hypotheses regarding the reduced response-ERN amplitudes found in older adults were disconfirmed. Our study may inspire similar investigations of altered error processing in other populations. Also, it indicates the importance of considering evaluative control function deficits as a possible explanation for performance deficits in tasks requiring executive control and reward-based learning.

REFERENCES

Arnsten, A. F. T. (1993). Catecholamine mechanisms in age-related cognitive decline. *Neurobiology of Aging*, **14**, 639-641.

Arnsten, A. F. T., & Goldman-Rakic, P. S. (1985). Alpha 2-adrenergic mechanisms in prefrontal cortex associated with cognitive decline in aged nonhuman primates. *Science*, **230**, 1273-1276.

Backman, L., Ginovart, N., Dixon, R. A., Wahlin, T. B., Wahlin, A., Halldin, C., & Farde L. (2000). Age-related cognitive deficits mediated by changes in the striatal dopamine system. *American Journal of Psychiatry*, **157**, 635-637.

Band, G. P. H., & Kok, A. (2000). Age effects on response monitoring in a mental-rotation task. *Biological Psychology*, **51**, 201-221.

Bernstein, P. S., Scheffers, M. K., & Coles, M. G. H. (1995). "Where did I go wrong?" A psychophysiological analysis of error detection. *Journal of Experimental Psychology: Human Perception & Performance*, **21**, 1312-1322.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Evaluating the demand for control: Anterior cingulate cortex and conflict monitoring. *Psychological Review*, **108**, 624-652.

Braver, T. S., Barch, D. M., Keys, B. A., Carter, C. S., Cohen, J. D., Kaye, J. A., Janowsky, J. S., Taylor, S. F., Yesavage, J. A., Mumenthaler, M. S., Jagust, W. J., & Reed, B. R. (2001). Context processing in older adults: Evidence for a theory relating cognitive control to neurobiology in healthy aging. *Journal of Experimental Psychology: General*, **130**, 746-763.

Braver, T. S., & Cohen, J. D. (2000). On the control of control: The role of dopamine in regulating prefrontal function and working memory. In S. Monsell & J. Driver (Eds.), *Attention and performance XVIII: Control of cognitive processes* (pp. 713-737). Cambridge, MA: MIT Press.

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*, **280**, 747-749.

Coles, M. G. H., Gratton, G., Bashore, T. R., Eriksen, C. W., & Donchin, E. (1985). A psychophysiological investigation of the continuous flow model of human information processing. *Journal of Experimental Psychology: Human Perception & Performance*, **11**, 529-553.

Coles, M. G. H., Scheffers, M. K., & Holroyd, C. B. (1998). Berger's dream: The error-related negativity and modern cognitive psychophysiology. In H. Witte, U. Zwiener, B. Schack, & A. Doering (Eds.), *Quantitative and topological EEG and MEG analysis* (pp. 96-102). Jena-Erlangen: Druckhaus Mayer.

Coles, M. G. H., Scheffers, M. K., & Holroyd, C. B. (2001). Why is there an ERN/Ne on correct trials? Response representations, stimulus-related components, and the theory of error-processing. *Biological Psychology*, **56**, 173-189.

Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a neural system for error detection and compensation. *Psychological Science*, **5**, 303-305.

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception & Psychophysics*, **16**, 143-149.

Falkenstein, M., Hielscher, H., Dziobek, I., Schwarzenau, P., Hoormann, J., Sunderman, B., & Hohnsbein, J. (2001). Action monitoring, error detection, and the basal ganglia: An ERP study. *NeuroReport*, **12**, 157-161.

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. *Electroencephalography & Clinical Neurophysiology*, **78**, 447-455.

Falkenstein, M., Hoormann, J., Christ, S., & Hohnsbein, J. (2000). ERP components on reaction errors and their functional significance: A tutorial. *Biological Psychology*, **51**, 87-107.

Falkenstein, M., Hoormann, & Hohnsbein, J. (2001). Changes of error-related ERPs with age. *Experimental Brain Research*, **138**, 258-262.

Ford, J. M. (1999). Schizophrenia: The broken P300 and beyond. *Psychophysiology*, **36**, 667-682.

Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science*, **4**, 385-390.

Gehring, W. J., Himle, J., & Nisenson, L. G. (2000). Action-monitoring dysfunction in obsessive-compulsive disorder. *Psychological Science*, **11**, 1-6.

Gehring, W. J., & Knight, R. T. (2000). Prefrontal-cingulate interactions in action monitoring. *Nature Neuroscience*, **3**, 516-520.

Goldman-Rakic, P. S., & Brown, R. M. (1981). Regional changes of monoamines in cerebral cortex and subcortical structures of aging rhesus monkeys. *Neuroscience*, **6**, 177-187.

Gratton, G., Coles, M. G. H., Sirevaag, E. J., Eriksen, C. W., & Donchin, E. (1988). Pre- and poststimulus activation of response channels: A psychophysiological analysis. *Journal of Experimental Psychology: Human Perception & Performance*, **14**, 331-344.

Holroyd, C. B., & Coles, M. G. H. (in press). The neural basis of

human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*.

Holroyd, C. B., Dien, J., & Coles, M. G. H. (1998). Error-related scalp potentials elicited by hand and foot movements: Evidence for an output-independent error-processing system in humans. *Neuroscience Letters*, **242**, 65-68.

Holroyd, C., Praamstra, P., Plat, E., & Coles, M. G. H. (in press). Spared error-related potentials in mild to moderate Parkinson's disease. *Neuropsychologia*.

Holroyd, C., Reichler, J., & Coles, M. G. H. (1999, April 10–13). Is the error-related negativity generated by a dopaminergic error signal for reinforcement learning? Hypothesis and model [Abstract]. *Cognitive Neuroscience Society 1999 annual meeting*, Washington, DC.

Holroyd, C. B., Yeung, N., Coles, M. G. H., & Cohen, J. D. (2002). *A computational model of learning and error detection in the Eriksen Flankers Task*. Manuscript in preparation.

Kaasinen, V., Vilkman, H., Hietala, J., Nagren, K., Helenius, H., Olsson, H., Farde, L., & Rinne J. (2000). Age-related dopamine D2/D3 receptor loss in extrastriatal regions of the human brain. *Neurobiology & Aging*, **21**, 683-688.

Kiehl, K. A., Liddle, P. F., & Hopfinger, J. B. (2000). Error processing and the rostral anterior cingulate: An event-related fMRI study. *Psychophysiology*, **37**, 216-223.

Kok, A. (2000). Age-related changes in involuntary and voluntary attention as reflected in components of the event-related potentials (ERP). *Biological Psychology*, **54**, 107-143.

Kopp, B., & Rist, F. (1999). An event-related brain potential substrate of disturbed response monitoring in paranoid schizophrenic patients. *Journal of Abnormal Psychology*, **108**, 337-346.

Li, S. C., & Lindenberger, U. (1999). Cross-level unification: A computational exploration of the link between deterioration of neurotransmitter systems and dedifferentiation of cognitive abilities in old age. In L. G. Nilsson & H. J. Markowitsch (Eds.), *Cognitive neuroscience of memory* (pp. 103-146). Seattle: Hogrefe & Huber.

Miltner, W. H. R., Braun, C. H., & Coles, M. G. H. (1997). Event-related brain potentials following incorrect feedback in a time-estimation task: Evidence for a generic neural system for error detection. *Journal of Cognitive Neuroscience*, **9**, 788-798.

Moscovitch, M., & Winocur, G. (1995). Frontal lobes, memory, and aging. In J. Grafman, K. J. Holyoak, & F. Boller (Eds.), *Structure and functions of the human prefrontal cortex* (Annals of the New York Academy of Sciences, Vol. 769, pp. 119-151). New York: New York Academy of Sciences.

Nieuwenhuis, S., Ridderinkhof, K. R., Blom, J., Band, G. P. H., & Kok, A. (2001). Error-related brain potentials are differentially related to awareness of response errors: Evidence from an antisaccade task. *Psychophysiology*, **38**, 752-760.

Phillips, L. H., & Della Sala, S. (1996). Aging, intelligence, and anatomical segregation in the frontal lobes. *Learning & Individual Differences*, **10**, 217-243.

Rabbitt, P. M. A. (1979). How old and young participants monitor and control responses for accuracy and speed. *British Journal of Psychology*, **70**, 305-311.

Raven, J. C., Court, J. H., & Raven, J. (1988). *Manual for Raven's progressive matrices and vocabulary scales: Sec. 3. Standard progressive matrices*. London: Lewis.

Scheffers, M. K., & Coles, M. G. H. (2000). Performance monitoring in a confusing world: Event-related brain activity, judgements of response accuracy, and types of errors. *Journal of Experimental Psychology: Human Perception & Performance*, **26**, 141-151.

Scheffers, M. K., Coles, M. G. H., Bernstein, P., Gehring, W. J., & Donchin, E. (1996). Event-related brain potentials and error-related processing: An analysis of incorrect responses to go and no-go stimuli. *Psychophysiology*, **33**, 42-53.

Sutton, R. S., & Barto, A. G. (1998). *Reinforcement learning: An introduction*. Cambridge, MA: MIT Press.

van der Molen, M. W., & Ridderinkhof, K. R. (1998). The growing and aging brain: Life-span changes in brain and cognitive functioning. In A. Demetriou, W. Doise, & C. F. M. van Lieshout (Eds.), *Life-span developmental psychology: A European perspective* (pp. 35-99). Chichester, U.K.: Wiley.

van der Veen, F. M., Nieuwenhuis, S., van der Molen, M. W., Crone, E. A., & Jennings, J. R. (2001). *Heart rate and error-related negativity reflect different aspects of feedback processing during probability learning*. Manuscript in preparation.

Volkow, N. D., Gur, R. C., Wang, G. J., Fowler, J. S., Moberg, P. J., Ding, Y. S., Hitzemann, R., Smith, G., & Logan, J. (1998). Association between decline in brain dopamine activity with age and cognitive and motor impairment in healthy individuals. *American Journal of Psychiatry*, **155**, 344-349.

Woestenburg, J. C., Verbaten, M. N., & Slangen J. L. (1983). The removal of the eye-movement artifact from the EEG by regression analysis in the frequency domain. *Biological Psychology*, **16**, 127-147.

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2001). *The neural basis of error detection: Conflict monitoring and the error-related negativity*. Manuscript submitted for publication.

Zeef, E. J., & Kok, A. (1993). Age-related differences in the timing of stimulus and response processes during visual selective attention: Performance and psychophysiological analyses. *Psychophysiology*, **30**, 138-151.

Zeef, E. J., Sonke, C. J., Kok, A., Buiten, M. M., & Kenemans, J. L. (1996). Perceptual factors affecting age-related differences in focused attention: Performance and psychophysiological analyses. *Psychophysiology*, **33**, 555-565.

NOTES

1. The numerical effect of congruency on ERN amplitude is probably the result of differential contributions of stimulus-related components to the response-locked waveform for each of the two conditions, rather than to "real" differences between the ERNs on congruent and incongruent trials (for a discussion, see Coles et al., 2001). When the trials in the two categories were matched with respect to RT, using the procedure described by Bernstein, Scheffers, and Coles (1995), ERN amplitude was $-1.3\ \mu V$ for both congruent and incongruent errors.

2. The 80% condition was not part of the design of Holroyd and Coles (in press). Instead, these authors used an "always correct" condition and an "always incorrect" condition, in which the value of the feedback could be entirely predicted on the basis of the imperative stimulus.

3. To reach a satisfying correspondence between empirical and simulated data, we needed to change at least two of the three learning rate parameters of the older model. Decreasing the learning rate parameter value associated with the value layer weights updating function (from 0.1 to 0.025) was necessary. In addition, either one of the parameters associated with the functions for updating the input layer-controller weights (from 0.5 to 0.2) and the controller-filter weights (from 0.2 to 0.1) needed to be changed. Both choices of parameters led to a very adequate correspondence between the empirical and simulated data (essentially similar to that associated with the dopamine model) as far as accuracy and response ERNs were concerned. But the feedback-ERN amplitudes produced by these two models were approximately −.63 (100% condition), −.70 (80% condition), −.83 (50% condition), and −.97 (80% invalid condition), a substantial overestimation of the empirical feedback-ERN amplitudes of older adults.

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APPENDIX
Model Description

Apart from a few explicitly specified exceptions, all details of the model were identical to those specified by Holroyd and Coles (in press, Appendix A). Here, we provide a global description of the model (Figure 3). On each simulated trial, an input layer consisting of six units (one for each stimulus) encoded the presented stimulus. Restrictions to the number and type of stimuli were the same as in the experiment. The activation in the input layer was sent through modifiable feedforward connections to five motor controllers, each composed of two units, one for each response option (left vs. right hand). Each motor controller selected, in a winner-takes-all fashion, one response according to a probabilistic function of the relative net inputs to its two units. However, the function parameters were set so that one controller was best suited to perform the task. Then, a control filter, connected to each of the motor controllers, selected one motor controller as a probabilistic function of the (modifiable) connection weights. The output layer consisted of two units (one for each response) and the control filter activated the output unit associated with the response option selected by the chosen controller. Finally, following the response, one of two units in a feedback layer was activated (according to the same feedback schedule as that used for the participants): one unit in case of reward and the other one in case of punishment.

The units in the input layer, output layer, and feedback layer were connected through feedforward weights to a value layer in the adaptive critic, which was composed of 22 units, representing the possible states of the network: units for representing activation in each of the input (6), output (2), or feedback (2) units, and one unit for each possible conjunction of active input and output units ($6 * 2 = 12$). At any one time, no more than one value unit was activated, and its activation was set equal to 1. The activation of the value units was projected, through modifiable connection weights, to a summation unit, so that at each time step within a trial, the value of this summation unit, $\hat{V}$, was equal to the weight associated with the activated value unit. At each time step t, the adaptive critic also computed the TD error:

$$\delta_t = r_t + \gamma\hat{V}_t - \hat{V}_{t-1} + \text{err},$$

where r was the value of the feedback signal (+1 for reward and −1 for punishment), γ was a constant (i.e., a discount factor, fixed at 1.0), and err was a noise term. All modifiable weights in the network were constantly adjusted as a function of the TD error, with a set of learning rate parameters determining the rate of change. The weights between the value layer and the summation unit were adjusted to improve the predictions of the adaptive critic; those between the input layer and motor controllers to learn the appropriate response to each stimulus; and those between the control filter and the motor controllers to enable the control filter to learn to select the most appropriate controller. Finally, the ERN amplitude was defined as the TD error multiplied by the magnitude of an "eligibility trace"—that is, a decaying representation of the selected controller sent to the adaptive critic (Figure 3). This form of memory trace allows the system to maintain states (here, the selected controller) eligible for learning (see Holroyd & Coles, in press).

Apart from the parameter adjustments meant to simulate the effects of cognitive aging (see Results section), we made one general adjustment to the parameter values used by Holroyd and Coles (in press). For both the younger and older models, we slightly changed the learning rate parameter value associated with the value layer weights updating function (from 0.2 to 0.1). We did this to improve the overall quality of the model fits.

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