Notes and Comment

Locus of the redundant-signals effect in bimodal divided attention: A neurophysiological analysis

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We reanalyzed the data from the study of Lamarre, Busby, and Spidalieri (1983). In that study, the activity of single neurons in area 4 of the motor cortex was recorded during a bimodal detection task in which a monkey (Macaca mulatta) had to respond as quickly as possible to a visual or an auditory signal or to both (redundant trials). Manual responses on redundant trials were speeded by the presence of both signals, as is typically found. The times between signal onsets and the first changes in neuronal activity were also speeded by redundant signals, but there was no difference between redundant-signals and single-signal trials in the time between the change in neuronal activity and movement onset. These results suggest that late motor processes are not speeded by redundant signals in bimodal detection tasks.

Bimodal detection tasks have often been used to study the mechanisms operating when attention is divided between the auditory and the visual modalities (see Stanislaw, 1988, for a review). Raab (1962) was one of the first to study a speeded version of this task. Observers were required to respond as quickly as possible to a visual signal, an auditory signal, or both (i.e., redundant signals), and Raab found that responses to redundant signals were faster, on average, than those to either the visual or the auditory signals by themselves. This redundant-signals effect (RSE) has since been replicated many times (e.g., Corballis, 1998; Diederich, 1995; Diederich & Colonius, 1987; Giray & Ulrich, 1993; Hughes, Reuter-Lorenz, Nozawa, & Fendrich, 1994; Marzi et al., 1996; Marzi, Tassinari, Aglioti, & Lutzemberger, 1986; Miller, 1982, 1986; Schwarz & Ischebeck, 1994).

Quantitative analyses of the RSE have provided insights into the explanation of the speed-up in redundant trials. Raab (1962) suggested that the effect might be ex-

plained by a race model. According to this model, the response in each trial is initiated by detection of either the visual signal or the auditory one. In single-signal trials, the response cannot be initiated until the presented signal is detected, but in redundant-signals trials, the response can be initiated as soon as *either* signal is detected. Raab showed that such a model could explain the RSE, essentially because the winner of a race goes faster, on average, than either of the runners in the race.

Although Raab's (1962) race model provides an intuitively plausible account of the reduced mean reaction time (RT) in redundant-signals trials, analyses of RT distributions indicate that this model cannot explain the full speed-up in redundant-signals trials. Miller (1982) showed that Raab's model requires RTs on visual, auditory, and redundant trials to obey the following inequality:

$$F_r(t) \leq F_v(t) + F_a(t), \quad (1)$$

where $F_v(t)$, $F_a(t)$, and $F_r(t)$ are the cumulative probability distributions of RT in visual, auditory, and redundant trials, respectively. This inequality holds for all *separate-activations* race models in which the visual and the auditory detection processes operate separately and each operates at the same speed regardless of whether the other signal is presented (Ashby & Townsend, 1986; Luce, 1986). $F_r(t)$ can approach $F_v(t) + F_a(t)$ for small values of t if the detection times of the two single signals have a strong negative correlation (Colonius, 1990), but even in that case Inequality 1 must still hold. Observed RT distributions often violate Inequality 1, however, with the fastest RTs in redundant-signals trials being faster than the fastest of either the visual or the auditory single-signals trials (e.g., Diederich & Colonius, 1987; Giray & Ulrich, 1993; Miller, 1982, 1986). Although it is possible to elaborate race models so that they are compatible with violations of Inequality 1 on the basis of the presence of certain contingencies in many redundant-signals experiments (e.g., Mordkoff & Yantis, 1991), the observed violations of Inequality 1 are generally thought to support an alternative class of coactivation models (e.g., Grice, Canham, & Boroughs, 1984; Schwarz, 1989; Townsend & Nozawa, 1995; Ulrich & Giray, 1986; Ulrich & Miller, 1997), in which visual and auditory signals jointly influence the process of generating a response, rather than racing separately, as Raab's (1962) model proposed.

Despite the robustness of the RSE and coactivation in bimodal detection tasks, the processing level at which coactivation occurs has not yet been identified. One possibility is that bimodal interactions occur during sensory detection. There is evidence that such sensory interactions are at least partly responsible for the RSE obtained when redundant signals are presented within the same

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sensory modality (Miniussi, Girelli, & Marzi, 1998). Furthermore, some cortical areas involved in sensory processing do contain neurons that respond to the convergence of both visual and auditory inputs (e.g., Meredith & Stein, 1986), and these would presumably fire sooner and more rapidly in redundant trials than in single-signal trials. Thus, coactivation could arise at the level of sensory detection. Contrary to this hypothesis, Schröger and Widmann (1998) found that visual and auditory single and redundant signals produced the same event-related potentials (ERPs) over the first 190 msec after stimulus onset, and they concluded that auditory–visual interactions did not begin until after sensory processing was completed. They used a go/no-go task, however, and their conclusions may not be applicable to a simple detection task. In particular, they presented both auditory and visual stimuli on every trial, with go stimuli differing from no-go stimuli only in position. Thus, sensory interactions that occurred prior to the position discrimination would have been equal in the single-signal and redundant-signals conditions.

An alternative possibility is that the RSE arises substantially—or even entirely—in motor processes. Diederich and Colonius (1987) and Giray and Ulrich (1993) have found evidence supporting this account. Diederich and Colonius required participants to respond with both the left and the right index fingers whenever a signal was presented. Following Ulrich and Stapf (1984), they assumed that the time difference between the two fingers' responses depended only on the duration of separate motor processes for the left and the right hands. They found that the distribution of this difference depended on the inter-stimulus interval in redundant trials and concluded that the RSE arose at least partly because of facilitation in the motor component. Giray and Ulrich measured response force as well as RT and found that responses to redundant signals were not only faster, but also more forceful, than responses to single signals.¹ Given that response force is a direct measure of motor output, this result is *prima facie* evidence that motor processing depends on the number of signals and hence led Giray and Ulrich to conclude that coactivation in the motor system could contribute significantly to the RSE. On the basis of an analysis of motoric ERPs, however, Mordkoff, Miller, and Roch (1996) challenged the account of coactivation in terms of motor processes. Their evidence suggested that motor processes consumed the same duration in both single- and redundant-signal trials. Mordkoff et al.'s analysis used a task requiring discrimination of targets from nontargets, however, and they acknowledged that motor coactivation might still be present in tasks requiring only stimulus detection.

The present article provides a new test of the hypothesis that the RSE found in bimodal detection tasks arises partly because of a speed-up in late motor processes. Specifically, we report analyses of the latencies for changes in the activity of single neurons in the primary motor cortex (Brodmann's area 4) in a bimodal simple RT task with monkeys. Neurons in this area appear to be involved mainly in the late stages of generating a motor response, rather than in the processing of sensory information,

learning of stimulus–response associations, or action planning (e.g., Germain & Lamarre, 1993; Jasper, 1982; Lamarre, Busby, & Spidalieri, 1983). As has been reviewed by Ghez (1991), a relatively large percentage of the neurons in the primary motor cortex contribute to the corticospinal tract, and electrical stimulation of this area produces simple contralateral movements. Moreover, the activity of neurons in this area seems to depend on the forcefulness of required movements, and lesions in this area weaken movements. In contrast, neurons of the premotor cortex and supplementary motor areas, from which the primary motor cortex receives inputs, seem to code aspects of a required movement plan, and lesions of these areas result in inaccurate or inappropriate movements.

Differences between single- and redundant-signals trials in the latencies of motor cortex neurons can be used to localize the RSE rather directly. In brief, if the RSE arises earlier than the primary motor cortex, the effect should already be present in the latencies of single-neuron activity. Alternatively, if the RSE arises only after late motor processing begins in area 4, the advantage for redundant-signals trials should be present in RT, but not in these single-neuron latencies. Summaries of these data were originally reported in Lamarre et al. (1983). In particular, they found that the intensities and durations of these neurons' responses did not differ for single- versus redundant-signals trials. That report did not, however, contain distributional analyses of RT and single-cell onset latencies using Inequality 1, which are needed to check for coactivation and localize the RSE.

THE EXPERIMENT OF LAMARRE ET AL. (1983)

The present results are based on more detailed analyses of the data presented by Lamarre et al. (1983, Figures 10–12), and a complete description of the method is given in that article. In addition, Spidalieri, Busby, and Lamarre (1983) report more detailed information concerning the locations of the recorded neurons within area 4.

In brief, a monkey was trained to make speeded elbow flexion movements in response to a white light, a 35-dB pure tone at 800 Hz, or both. After training, a headpiece allowing for microelectrode recordings was surgically implanted, and data collection commenced, following a suitable recovery period. In each trial, neuronal spike intervals were recorded to the nearest millisecond, starting with a baseline period 500 msec before stimulus onset and lasting for 2 sec. Angular displacement of the elbow was also recorded during this interval, digitized at 200 Hz.

Recordings with both single- and redundant-signals trials were obtained from only six neurons, and detailed analyses of the results obtained with these neurons are presented here. Each of these neurons was tested in 20 trials with each signal (visual, auditory, and redundant bimodal). Two neurons were tested with simultaneous auditory and visual bimodal signals (stimulus onset asynchrony $\Delta = 0$). On the basis of preliminary findings that responses were faster to the auditory signal than to

Table 1
Summary Descriptive Statistics for Latencies of Neuronal and Motor Responses

Neuron and Signal Condition	RT		CL		MT		Correlation		
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	RT&CL	RT&MT	CL&MT
Neuron 1									
Visual	266	22.3	180	22.3	86.4	15.6	.75	.35	-.35
Auditory	202	17.4	130	19.6	72.1	13.1	.76	.20	-.49
Bimodal, $\Delta = 0$	197	14.9	119	14.9	77.3	17.2	.33	.58	-.58
Neuron 2									
Visual	279	29.8	186	26.8	92.5	12.9	.90	.44	.01
Auditory	201	14.9	109	18.6	92.0	16.3	.54	.29	-.64
Bimodal, $\Delta = 0$	203	14.0	108	17.1	94.8	16.8	.43	.39	-.66
Neuron 3									
Visual	280	20.4	193	36.3	86.8	29.7	.58	-.02	-.83
Auditory	213	24.3	124	29.2	88.7	21.6	.69	.19	-.58
Bimodal, $\Delta = 39$	228	16.4	147	26.2	80.7	21.7	.56	.08	-.78
Neuron 4									
Visual	276	29.9	188	32.7	87.1	15.4	.88	.07	-.41
Auditory	200	19.0	101	26.1	98.6	23.4	.50	.25	-.71
Bimodal, $\Delta = 87$	246	22.4	149	18.9	97.4	17.5	.65	.58	-.24
Neuron 5									
Visual	275	23.3	189	28.7	86.0	20.9	.70	.16	-.60
Auditory	201	15.5	127	22.6	74.3	22.6	.34	.34	-.76
Bimodal, $\Delta = 114$	243	19.2	163	25.5	80.2	20.3	.62	.17	-.67
Neuron 6									
Visual	270	27.2	187	33.6	83.5	22.1	.75	.08	-.59
Auditory	209	18.3	118	31.3	90.5	21.8	.73	-.21	-.82
Bimodal, $\Delta = 139$	264	23.0	185	30.9	78.3	21.3	.72	.03	-.67
Means across neurons									
Visual	274	25.5	187	30.1	87.0	19.4	.79	.19	-.50
Auditory	204	18.2	118	24.6	86.0	19.8	.61	.18	-.68
Bimodal, mixed Δ s	230	18.3	145	22.3	84.8	19.1	.57	.32	-.62

Note—For each neuron and signal condition, the mean (*M*) and standard deviation (*SD*) are given for reaction time (RT), the onset latency of neuronal activity (CL), and the motor time (MT = RT – CL). Also given are the pairwise correlations between these three measures. Fisher's *r*-to-*Z* transformation was used in averaging correlations across neurons.

the visual signal, four other neurons were tested with the visual signal being presented earlier in redundant trials (Δ s equal to 39, 87, 114, and 139 msec, respectively).

On each trial, three latencies were measured. First, the RT was defined as the time from the onset of the stimulus (the first stimulus, on redundant trials with $\Delta > 0$) to the first of 10 consecutive angular displacement samples that changed in the same direction. Second, the onset latency of single-neuron activity (*cell latency*, CL) was scored by using a version of a method described by Evarts (1974). In essence, this latency was defined as the location of the first of 3 consecutive 10-msec bins for which each of the 3 bins contained more spikes than any of the fifty 10-msec bins during the 500-msec baseline period. Third, the motor time (MT) was computed for each trial as the difference MT = RT – CL. Measures like these have been used successfully in several studies of area 4 neurons, and the good reliability of these measures is apparent from—among other evidence—the systematic nature of the results reported by Lamarre et al. (1983).

ANALYSIS

Table 1 presents some basic summary statistics for each dependent measure, neuron, and signal condition. Several points are noteworthy regarding these basic data.

1. Inspection of the mean MT column reveals that all of the neurons changed firing rate approximately 70–100 msec before the onset of movement, on average. The relatively short time from neuron activity to movement is consistent with the idea that the neurons are involved in relatively late stages of movement activation, as would be expected from their location in area 4.

2. RTs were approximately 70 msec faster, on average, to the auditory signal than to the visual signal. This replicates the advantage in responding to auditory signals found in other conditions reported by Lamarre et al. (1983).

3. The advantage for auditory signals was essentially identical for RT and CL, suggesting that the effect of modality was localized entirely before area 4 neurons. Because MT was defined as RT – CL, it also follows from this finding that MT was the same for both signal modalities, consistent with the idea that motor speed does not depend on input signal modality.

4. RT was more strongly correlated with CL than with MT, suggesting that sensory detection processes are responsible for a larger portion of the total RT variance than are motor processes. There is also a strong negative correlation between CL and MT, but this follows necessarily from other aspects of the data already considered. Given the definition MT = RT – CL, it can be shown that the correlation of CL and MT has the same sign as²

$$\epsilon = \text{corr}(\text{RT}, \text{MT}) \cdot \text{SD}(\text{RT}) - \text{SD}(\text{MT}). \quad (2)$$

It can be seen from Table 1 that ϵ must be negative, given the observed sizes of the values in Equation 2. To grasp the source of the negative correlation, imagine for a moment the extreme case that RT is constant within a condition [i.e., $\text{SD}(\text{RT}) = 0$]. There would still be variability in CL owing to measurement error, and it is easy to see that CL and MT would be perfectly negatively correlated owing to their complementary relation (i.e., $\text{MT} = \text{RT} - \text{CL}$). Of course RT is actually somewhat variable in the real data, but this complementary relation of CL and MT nonetheless tends to induce their negative correlation.

Another question that should be examined using these basic data is whether the neurons were reasonably comparable with one another. Effects of Δ in redundant-signals trials—to be examined shortly—were assessed by testing different neurons with different values of Δ . It is important to consider how much the individual neurons differed from one another, even before they were assigned to different Δ conditions, because large differences between neurons would increase the possibility that the present results were specific to the particular neurons included in the present small sample. Fortunately, it is possible to compare the neurons with respect to their responses in visual and auditory single-signal trials, because these conditions were identical in all respects for all neurons. These comparisons indicate that the neurons were rather comparable in most respects. (1) Both RTs and neuron onset latencies CLs were not significantly different across neurons. To test for differences, analyses of variance (ANOVAs) were carried out separately for RT and CL, using between-subjects factors of neuron and visual versus auditory signal modality and using individual trials as random replications (i.e., “subjects”). Neither the main effects of neuron nor the neuron $\times$ signal interactions were significant in these analyses ($p < .05$). There were highly significant effects of signal modality, however, with faster responses to auditory than to visual signals evident in both RT and CL ($p < .001$). (2) A parallel analysis of MTs indicated that there were significant differences between neurons in MTs, although these were small. The effect of neuron was significant [$F(5,228) = 3.30$, $MS_e = 407.3$,

$p < .01$], as was the interaction of neuron and signal modality [$F(5,228) = 2.55$, $MS_e = 407.3$, $p < .03$]. However, these significant effects accounted for only 6.4% and 5.0% of the variance, respectively, so they do seem to indicate only minor differences among the neurons. (3) Differences across neurons in correlations also appear to be only a little, if any, larger than would be expected by chance. After using Fisher’s r -to- Z transformation, the standard deviation of the transformed correlation scores should be approximately .24 by chance (Hays, 1988). The observed standard deviations of the transformed scores were .30, .22, and .30 for the correlations of RT with CL, RT with MT, and CL with MT, respectively. Because these standard deviations are not much larger than would be expected theoretically even if all true correlations were identical, we can conclude that the neurons probably differed only slightly in their true correlations for all three pairs of variables.

Table 2 summarizes the observed RSEs. For each neuron and latency measure, the observed mean in the bimodal condition is compared against the minimum of the two single-signal means. For MT, the minimum was simply the smaller of the average MTs across the visual-signal and auditory-signal conditions. For RT (CL), the minimum was the smaller of the visual-signal mean RT (CL) and the auditory-signal mean $\text{RT} + \Delta(\text{CL} + \Delta)$, where the Δ value was added in order to adjust for the delayed onset of the auditory signal (cf. Miller, 1986; Ulrich & Miller, 1997). This adjustment procedure is based on the assumption—natural within the race model—that the only effect of changing the Δ is to alter the starting times of the two separate detection processes. Analogous assumptions are common in many other paradigms in which Δ is varied (e.g., Glaser & Glaser, 1982; Hommel, 1994; Pashler & Johnston, 1989; Sternberg & Knoll, 1973).

For RT, RSEs are clearly largest when the auditory signal is delayed by 39–114 msec (neurons 3–5), a pattern also reported by Giray and Ulrich (1993) and Miller (1986). Intuitively, when the two signals are simultaneous, the more slowly detected visual signal arrives too late to have any influence on the response. Conversely, when the auditory signal is presented with a sufficiently long delay (i.e., $\Delta = 139$ msec), it arrives too late to have an in-

Table 2
Mean Redundant-Signals Effects (RSEs) for Each Neuron and Latency Measure

Neuron	Δ	RT			CL			MT		
		Bimodal	Min	RSE	Bimodal	Min	RSE	Bimodal	Min	RSE
1	0	197	202	5	119	130	11	77.3	72.1	−5.2
2	0	203	201	−2	108	109	1	94.8	92.0	−2.8
3	39	228	252	24	147	163	16	80.7	86.8	6.1
4	87	246	276	30	149	188	39	97.4	87.1	−10.3
5	114	243	275	32	163	189	26	80.2	74.3	−5.9
6	139	264	270	6	185	187	2	78.3	83.5	5.2
Mean	Mixed	230	246	16	145	161	16	84.8	82.6	−2.2

Note—For each neuron, the mean is given for the bimodal condition and for the minimum (Min) of the single-signal conditions, separately for reaction time (RT), the onset latency of neuronal activity (CL), and the motor time ($\text{MT} = \text{RT} - \text{CL}$). The RSE is the difference between the single-signal minimum mean and the bimodal mean.

fluence. Redundancy gain is maximal when both signals are detected at about the same time.

Crucially, the RSEs observed in CL are approximately the same size as those observed in RT, and virtually no RSEs are observed in MT. These observations suggest that the RSE occurs entirely before the changes in activity of area 4 neurons and that the duration of the motor component is independent of the number of signals.

As was discussed in the introduction, it is possible to establish a limit on the RSE predicted by separate-activations race models using Inequality 1. For the analyses of neurons 3–6, the inequality must be generalized to allow for the delay of Δ msec in the onset of the auditory signal on redundant trials. Under appropriate independence assumptions, the generalized inequality is

$$F_r(t) \leq F_v(t) + F_a(t + \Delta) \quad (3)$$

(e.g., Ashby & Townsend, 1986; Miller, 1986). In the present data, this inequality can be tested both with RT values and with values of CL. If coactivation occurs before the motor system, violations of the inequality should be present when the inequality is tested with CL, as well as with RT; if coactivation occurs only in the motor system, however, violations of the inequality should not be present when the inequality is tested with CL.

Figure 1 shows tests of the race model inequality applied to both RT and CL. When applied to RTs, the inequality is clearly violated for neurons 3–5—the same neurons for which an RSE was obtained (cf. Table 2). As will be described in the Appendix, these violations appear too large to have been obtained by chance. More important, it is clear that the inequality was violated for CL as well as for RT, and in fact the extent of inequality violations is approximately the same in CL as in RT for all

Figure 1. Panels 1–6 show tests of Inequality 3 for neurons 1–6, respectively, separately for reaction time (RT) and onset latency of neuronal activity (CL). The solid lines are the observed cumulative distribution functions (CDFs) in the redundant-signals condition, and the dotted lines are the sums of the CDFs for the two single-signal conditions.

neurons. As in the analysis of mean RTs, this supports the conclusion that most or all of the coactivation produced by redundant signals occurs within the first 100–150 msec of stimulus processing, and little or no coactivation occurs during the final approximately 85 msec of motor processing.³

In summary, the present findings provide evidence against late motoric explanations of coactivation in bimodal detection tasks. Under stimulus conditions leading to an RSE in RT (i.e., Δ s of 39–114 msec), the RSE and violations of Inequality 3 were clearly present in the activation latencies of area 4 neurons, and there was virtually no evidence of a reduction in motor latencies on redundant-signals trials, as compared with single-signal trials. Although data are only available from six neurons, the results provide important new information about the locus of coactivation, because there are no previous tests of this inequality with latency measures that partition the RT interval. The lack of effect on motor latencies extends the original report of Lamarre et al. (1983), who showed that the intensity and duration of motor neuron responses were the same for single and redundant signals. These findings also agree nicely with recent evidence that the duration of motor processes is also invariant of stimulus intensity (Miller, Ulrich, & Rinkenauer, 1999) and thus provide additional support for the idea of an invariant motor process consuming the same latency regardless of the stimulus condition, as has frequently been assumed for mathematical convenience in modeling RT (e.g., Luce, 1986). At the same time, these results raise difficulties for continuous-flow models (e.g., Eriksen & Schultz, 1979; McClelland, 1979), in which the duration of motor processing depends on the duration of perceptual processing, because these two types of processing overlap.

The present conclusions about an invariant motor process seemingly conflict with the observation that response force is increased on redundant trials (Giray & Ulrich, 1993). The simplest explanation of the discrepancy is that changes in response force are simply not associated with corresponding changes in the duration of motor processes, when correlated either across single trials (Giray & Ulrich, 1993) or across conditions (e.g., Miller & Low, 2001; Miller et al., 1999). The present conclusions also differ from those reached by Diederich and Colonius (1987), who used an analysis of interresponse times in a task requiring bimanual responses (cf. Ulrich & Stapf, 1984). As was discussed by Miller et al. (1999), conclusions about motor times from this paradigm require the assumption that premotor processes are identical for both response hands. This assumption is weakened by evidence that the two response hands are activated by somewhat different perceptual-motor pathways, rather than by a completely common pathway (Jeeves, 1969).

In conclusion, the present results illustrate the clear advantage of having temporal markers between the stimulus and the response for localizing the source of RT differences between conditions. Although RT researchers

have traditionally attempted to localize experimental effects by using RT itself (e.g., Sternberg, 1969), it is becoming increasingly clear that psychophysiological measures also have an important role to play in this process. Many chronometric approaches developed in RT paradigms can also be applied very profitably to single-neuron latencies, as well as other psychophysiological measures of response latency (e.g., Hanes & Schall, 1996; Riehle & Requin, 1993; Thompson, Hanes, Bichot, & Schall, 1996; for a review, see also Van der Molen, Bashore, Halliday, & Callaway, 1991). For purposes of localizing further the coactivation processes involved in the RSE, it would clearly be helpful to have additional temporal markers somewhat earlier than the motor neurons of area 4.

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NOTES

1. Plat, Praamstra, and Horstink (2000) also found evidence of an effect of redundant signals on motor output. Although they did not replicate the finding that the amount of force varied with the number of signals, they did find that force output increased more abruptly with redundant signals than with single ones.

2. Equation 2 can be derived as follows, using cov, corr, and *SD* as abbreviations for covariance, correlation, and standard deviation, respectively:

$$\begin{aligned} \text{cov}(\text{CL}, \text{MT}) &= \text{cov}(\text{RT} - \text{MT}, \text{MT}) \\ &= \text{cov}(\text{RT}, \text{MT}) + \text{cov}(-\text{MT}, \text{MT}) \\ &= \text{cov}(\text{RT}, \text{MT}) - \text{cov}(\text{MT}, \text{MT}) \\ &= \text{cov}(\text{RT}, \text{MT}) - \text{var}(\text{MT}) \\ &= \text{corr}(\text{RT}, \text{MT}) \cdot \text{SD}(\text{RT}) \cdot \text{SD}(\text{MT}) - \text{SD}(\text{MT}) \cdot \text{SD}(\text{MT}) \\ &= \text{SD}(\text{MT}) \cdot [\text{corr}(\text{RT}, \text{MT}) \cdot \text{SD}(\text{RT}) - \text{SD}(\text{MT})]. \end{aligned}$$

By definition, the correlation $\text{corr}(\text{CL}, \text{MT})$ has the same sign as its corresponding covariance $\text{cov}(\text{CL}, \text{MT})$. It follows, then, that $\text{corr}(\text{CL}, \text{MT})$ must have the same sign as the term $\text{corr}(\text{RT}, \text{MT}) \text{SD}(\text{RT}) - \text{SD}(\text{MT})$ on the right side of the above expression.

3. In fact, if the motor system were completely responsible for coactivation, one would expect to find violations of a version of Inequality 1 when using the CDFs of the MTs instead of the CDFs of the RTs. No such violations were found in the present data, however.

APPENDIX

Statistical Significance of Inequality Violations

To our knowledge, there is as yet no formal test of the statistical significance of inequality violations appropriate for these data. Therefore, to assess whether the inequality might have been violated by chance, we proceeded to obtain critical values for the size of the violation via computer simulation, using a four-step procedure for each neuron separately. First, for each signal modality separately, we used the observed single-signal RTs to compute maximum-likelihood estimates of the parameters of an ex-Gaussian single-signal RT distribution for that neuron and modality. These provided reasonably good fits; the median correlation between observed and predicted RTs across 20 percentile points was .964. Second, from the two estimated single-signal distributions for each neuron, we constructed the fastest possible RT distribution for redundant trials consistent with the class of separate-activation race models under consideration. Specifically, this distribution, $F_r(t)$, was constructed so that

$$F_r(t) = \begin{cases} F_v(t) + F_a(t) & \text{if } F_v(t) + F_a(t) < 1 \\ 1 & \text{if } F_v(t) + F_a(t) \geq 1 \end{cases}.$$

Third, we simulated 1,000 experiments for the neuron. In each experiment, 20 RTs were randomly sampled from each of the

three conditions (i.e., estimated ex-Gaussian single-signal distributions and constructed redundant-signals distribution). The data from each simulated experiment were used to estimate the redundant-signals CDF and the sum of the single-signal CDFs for percentiles of 5, 15, 25, ..., 95, just as the real data had been (i.e., as shown in Figure 1). Finally, for each percentile, we identified a critical value for the difference between the estimates of redundant-signals and summed single-signal CDFs, which was that the difference value that was exceeded in only 2.5% of the simulated experiments. The observed difference was compared with this critical value for each neuron and each percentile value. Observed differences were larger than the critical differences for at least 5 of the 10 percentile values examined for neurons 3, 4, and 5, but not for any percentile values with the other neurons. Thus, these simulations provide some support that the inequality violations observed with neurons 3–5 were too large to have been observed by chance under a race model. Analogous simulations were conducted to test the statistical significance of inequality violations with CL, and these showed statistically reliable violations only for neurons 4 and 5. The ex-Gaussian model for single-signal latencies also provided reasonably good fits with the CL values, with a median correlation between the observed and the predicted values of .977.

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