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**Specifying Task 2 Response Activation in Response-Based  
Backward Crosstalk**

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## Abbreviations

<b>AIC</b>	<i>Akaike Information Criterion</i>
<b>BCE</b>	<i>Backward Crosstalk Effect</i>
<b>BIC</b>	<i>Bayesian Information Criterion</i>
<b>DDM</b>	<i>Drift-Diffusion Model</i>
<b>DMC</b>	<i>Diffusion Model for Conflict Tasks</i>
<b>ECTVA</b>	<i>Executive Control of Theory of Visual Attention</i>
<b>KFE</b>	<i>Kolmogorov Forward Equation</i>
<b>PDF</b>	<i>Probability Density Function</i>
<b>PRP</b>	<i>Psychological Refractory Period</i>
<b>R1</b>	<i>Task 1 Response</i>
<b>R2</b>	<i>Task 2 Response</i>
<b>RSB</b>	<i>Response Selection Bottleneck</i>
<b>RT1</b>	<i>Task 1 Response Time</i>
<b>RT2</b>	<i>Task 2 Response Time</i>
<b>S-R</b>	<i>Stimulus-Response</i>
<b>S1</b>	<i>Task 1 Stimulus</i>
<b>S2</b>	<i>Task 2 Stimulus</i>
<b>SOA</b>	<i>Stimulus Onset Asynchrony</i>
<b>T1</b>	<i>Task 1</i>
<b>T2</b>	<i>Task 2</i>

**Abstract**

In dual-task experiments, overlapping response characteristics of two consecutive tasks affect performance not only in Task 2 (T2) but also in Task 1 (T1). In particular, T1 responses are faster and less error-prone when the subsequent T2 response indicates the same relative to a different response. This phenomenon is known as the response-based backward crosstalk effect (BCE) and is often explained by pre-activated T2 response information influencing T1 response selection. The present dissertation focuses on the temporal development of T2 response activation during T1 response selection and its fate thereafter. Three studies are presented, combining modern cognitive modeling with more traditional experimental approaches. In Study 1, we used a diffusion model as a first approach to investigate whether T2 response activation increases monotonically or is rather transient. In this study, we also modeled to what extent (and if at all) T2 response activation carries over into T2 response selection. In Study 2, we examined whether T2 response activation is always equally strong, regardless of whether the T2 and T1 responses match or mismatch. In other words, we investigated whether T2 response activation equally facilitates and interferes with T1 response selection, as was implicitly assumed in Study 1. In a final Study 3, we re-examined the time course of T2 response activation, exploring alternative activation functions with a more sophisticated methodology using the developed R package dRiftDM (see "Study" 4). Overall, the studies show that T2 response activation increases rather monotonically for most individuals, and that it equally facilitates and interferes with T1 response selection. It also appears that T2 response activation does not carry over into T2 response selection. Instead, T2 response selection starts with a trace of all the response information accumulated during the T1 decision.

## Zusammenfassung

In Doppelaufgaben-Experimenten beeinflussen sich zwei aufeinanderfolgende Aufgaben mit überlappenden Antwortmerkmalen gegenseitig, wobei nicht nur die Leistung in Aufgabe 2 (A2), sondern auch die in Aufgabe 1 (A1) beeinflusst wird. Genauer gesagt ist eine Reaktion auf A1 schneller und weniger fehleranfällig, wenn die nachfolgende Reaktion auf A2 konzeptuell mit der ersten Reaktion identisch ist, als wenn dies nicht der Fall ist. Diese Beobachtung ist als "response-based backward crosstalk effect (BCE)" bekannt und wird häufig durch eine voraktivierte A2-Reaktion erklärt, die die Auswahl der A1-Reaktion beeinflusst. Die vorliegende Dissertation spezifiziert diese voraktivierte A2-Reaktion, mit Fokus auf ihre zeitliche Entwicklung während der A1-Reaktionsauswahl und ihren Einfluss auf die nachfolgende A2-Reaktionsauswahl, sobald die A1-Reaktionsauswahl abgeschlossen ist. Drei Studien werden vorgestellt, die moderne Ansätze der kognitiven Modellierung mit traditionelleren Ansätzen der experimentellen Psychologie kombinieren. In Studie 1 haben wir in einem ersten Ansatz mittels eines Diffusionsmodells untersucht, ob die voraktivierte A2-Reaktion in ihrer Stärke eher monoton ansteigt oder nur transient ist. In dieser Studie haben wir auch modelliert, ob und inwieweit diese voraktivierte A2-Reaktion die nachfolgende A2-Reaktionsauswahl beeinflusst. In Studie 2 wurde untersucht, ob die voraktivierte A2-Reaktion immer gleich stark ist, unabhängig davon, ob die A1 und A2 Reaktionen konzeptuell übereinstimmen oder nicht. Mit anderen Worten, wir untersuchten, ob die voraktivierte A2-Reaktion die A1-Reaktion sowohl erleichtert als auch erschwert, wie in Studie 1 implizit angenommen. In einer letzten Studie 3 griffen wir die Frage des zeitlichen Verlaufs der voraktivierten A2-Reaktion erneut auf und untersuchten alternative zeitliche Verläufe mit einer ausgefeilteren Methodik. Dazu verwendeten wir das von uns entwickelte R Paket dRiftDM (siehe "Studie" 4). Insgesamt zeigen die Studien, dass die voraktivierte A2-Reaktion bei den meisten Personen eher monoton ansteigt und die A1-Reaktionsauswahl gleichermaßen erleichtert wie beeinträchtigt. Außerdem scheint die voraktivierte A2-Reaktion nicht in die nachfolgende, eigentliche A2-Reaktionsauswahl überzugehen. Stattdessen beginnt die A2-Reaktionsauswahl mit einem Teil der gesamten Antwortinformation, die während der A1-Verarbeitung akkumuliert wurde.

**Enclosed Manuscripts and Author Contributions****Study 1, published**

Koob, V., Ulrich, R., & Janczyk, M. (2023). Response activation and activation-transmission in response-based backward crosstalk: Analyses and simulations with an extended diffusion model. *Psychological Review*, *130*(1), 102–136.

Author	Author Position	Scientific Ideas %	Programming %	Analysis & Interpretation %	Paper Writing %
Koob	1	20	80	60	60
Ulrich	2	30	5	15	10
Janczyk	3	50	15	25	30

**Study 2, published**

Koob, V., Sauerbier, C., Schröter, H., Ulrich, R., & Janczyk, M. (2024). Separating facilitation and interference in backward crosstalk. *Journal of Experimental Psychology: Human Perception and Performance*, *50*(3), 295–312.

Author	Author Position	Scientific Ideas %	Data Generation %	Analysis & Interpretation %	Paper Writing %
Koob	1	50	50	55	50
Sauerbier	2	0	25	0	10
Schröter	3	20	0	15	10
Ulrich	4	10	0	15	10
Janczyk	5	20	25	15	20

**Study 3, under revision**

Koob, V., Ulrich, R., Ahrens, A., & Janczyk, M. (under revision). The time course of Task 2 response activation in dual-tasking: Modeling results, interindividual differences, and practical recommendations. *Invited for a revision by the Journal of Experimental Psychology: General.*

Author	Author Position	Scientific Ideas %	Programming %	Analysis & Interpretation %	Paper Writing %
Koob	1	50	100	60	50
Ulrich	2	15	0	10	10
Ahrens	3	5	0	10	10
Janczyk	4	30	0	20	30

**Study 4, in preparation**

Koob, V., Richter, T., Ulrich, R., & Janczyk, M. (in preparation). An introduction and tutorial to fitting (time-dependent) diffusion models with the R-Package dRiftDM. *Pre-Print at <https://osf.io/preprints/osf/3t2vf>.*

Author	Author Position	Scientific Ideas %	Programming %	Analysis & Interpretation %	Paper Writing %
Koob	1	30	80	60	50
Richter	2	30	15	10	10
Ulrich	3	10	0	10	10
Janczyk	4	30	5	20	30



## 1 General Introduction

On a daily basis, people face multiple task demands that require them to either interleave tasks, perform multiple tasks more or less simultaneously, or switch back and forth between tasks in relatively rapid alternation (Salvucci et al., 2009). For example, people may drive a car while chatting with their passenger. Such behavior is generally referred to as "multitasking." Although there is no precise definition of multitasking, it can be summarized as all actions or cognitive processes aimed at accomplishing two or more tasks within a limited amount of time, requiring the respective cognitive representations to be present simultaneously (see also Koch et al., 2018, p. 558). Interestingly, while people sometimes have to multitask, for example at work, they may also voluntarily seek to multitask. Modern technology has made it easier than ever to engage in multiple activities simultaneously, providing us with the opportunity, and perhaps even the temptation, to engage in multiple tasks. For example, we may be tempted to respond to an email at work just to avoid another, more frustrating task (e.g., Adler & Benbunan-Fich, 2013). Given its ubiquity, it is not surprising that multitasking has attracted diverse research from different areas of psychology. This research ranges from studies on interruptions on work performance in more applied contexts (e.g., Foroughi et al., 2014; Mark et al., 2008) to research addressing the underlying cognitive processes and structures in the context of cognitive psychology (e.g., De Jong, 1995; Pashler, 1984; Rogers & Monsell, 1995).

Although some studies suggest that a subset of individuals may actually achieve performance benefits from multitasking in certain cases (e.g., Brüning et al., 2020; Jersild, 1927), both applied and basic research regularly demonstrates that multitasking results in reduced overall task performance and a variety of negative side effects. For example, talking on the phone while driving significantly delays responses to important traffic signals (e.g., Levy et al., 2006; Strayer et al., 2006), making it a potential cause of traffic accidents (Backer-Grøndahl & Sagberg, 2011). While the importance of multitasking research can be easily highlighted with examples from complex task environments (such as driving a car or operating an airplane), measures of performance in these contexts require aggregating performance over long time intervals from less controlled tasks. As a result,

performance measures in complex task environments lack the precision to pinpoint the underlying cognitive mechanisms. To elucidate the basic cognitive structures and processes during multitasking, we need to use simpler tasks with a well-defined relationship between discrete stimuli and responses. An important approach in this regard are dual-task experiments, in which participants perform two relatively simple but speeded tasks sequentially or simultaneously within a single trial.

The present dissertation stands in the tradition of dual-task research and advances the understanding of a specific interference effect, known as the *Backward Crosstalk Effect* (BCE), which occurs when individuals perform two tasks with overlapping response characteristics. The dissertation is characterized by a theory-driven use of modern mathematical modeling approaches combined with classical experimental methods. In the next Chapter 2, I will first present the assumptions, key results, and theoretical models relevant to dual-task experiments. In Chapter 3, I will then discuss the BCE as an important empirical phenomenon in the context of dual-tasking. In Chapter 4, I will turn to the overarching goals of this dissertation and summarize how they have been addressed by the enclosed studies. Finally, the answers provided by each publication will be discussed in a larger context in Chapter 5.

## 2 Dual-Tasking: Research and Theoretical Models

The two most common paradigms in cognitive psychology to study multitasking are *task switching* and *dual-tasking* (for reviews, see Fischer & Janczyk, 2022; Kiesel et al., 2010; Koch & Kiesel, 2022; Koch et al., 2018). Whereas in task switching, all tasks are performed in a more or less isolated but alternating manner, dual-tasking is characterized by an overlapping presentation of tasks that are performed either simultaneously or in rapid succession.<sup>1</sup>

A common approach in dual-task research are *Psychological Refractory Period* (PRP) experiments, with one of the first PRP-like experiments conducted by Telford (1931). By systematically varying the onset of two stimuli, PRP experiments provide a window

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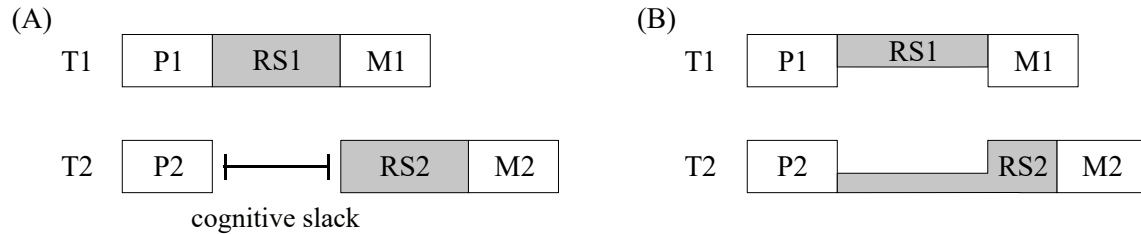
<sup>1</sup>This is only a rough distinction, and sometimes it is not easy to draw a clear line, for example when participants switch between tasks that are presented simultaneously (Reissland & Manzey, 2016).

into the processes and microstructures underlying dual-task performance (McCann & Johnston, 1992; Pashler, 1984, 1994; Welford, 1952). In a typical PRP experiment, participants perform two tasks (T1 and T2) in rapid succession. Specifically, participants first respond (R1) to one stimulus (S1) and then respond (R2) to another stimulus (S2). Typically, these tasks are arbitrary, albeit simple. In addition, to avoid perceptual and motor interference, stimuli are often presented in different modalities and responses are somewhat separated. For example, participants may first classify a tone as high or low in pitch using their left hand (T1), and then classify a visually presented digit as smaller or larger than five using their right hand (T2). The crucial manipulation in PRP experiments is the time interval between S1 and S2, known as the *Stimulus Onset Asynchrony* (SOA). A shorter relative to a longer SOA lengthens T2 response times (RT2s), while T1 response times (RT1s) are often (though not always) unaffected. The increase in RT2 is known as the PRP effect, and the label "psychological refractory period" associated with its effect and the experimental approach is an analogy to the refractory period of neurons from physiology (Telford, 1931). It is a well-established and robust finding (although for some exceptions, see Janczyk et al., 2014; Lien et al., 2006) that reflects a processing limitation in the human cognitive system.

A prominent explanation for the PRP effect is provided by Pashler (1984, 1994) and his famous *Response Selection Bottleneck* (RSB) model. A core assumption of this model is that task processing proceeds in discrete stages of stimulus perception, response selection, and motor execution (see Donders, 1868; Sternberg, 1969, for seminal work in this regard). In line with earlier suggestions by Welford (1952), the model assumes that longer RT2s with shorter SOAs result from a bottleneck at the response selection stage. Specifically, peripheral processes of stimulus perception and motor execution can occur concurrently with other tasks (i.e., they are "capacity-unlimited"). However, the central process of response selection, associated with the application of stimulus-response (S-R) rules (Fagot & Pashler, 1992), is limited to one task at a time. Therefore, as long as T1 response selection is currently ongoing, T2 response selection is delayed until T1 is released from the bottleneck (see also Fischer & Janczyk, 2022; Janczyk & Kunde, 2020; Koch et al.,

**Figure 1**

*Illustration of the Response Selection Bottleneck Model (A) and a Capacity-Sharing Model (B)*



*Note.* Both models assume stimulus perception (P) and motor execution (M) to run in parallel with processes of other tasks (i.e., to be capacity-unlimited). The RSB model (left panel) assumes response selection (RS) to constitute a structural bottleneck. RS thus proceeds sequentially, creating a period of "cognitive slack." Capacity-sharing models (right panel) assume RS to be limited in capacity, leading to less efficient, but parallel RS. Note that the vertical size of each box can be taken to reflect processing efficiency.

2018, for comprehensive summaries), and this idle waiting time is known as "cognitive slack" (see Figure 1A).<sup>2</sup>

Although the RSB model provides a parsimonious explanation for the PRP effect and makes precise predictions that can be empirically tested, there are alternative models that do not assume a seriality constraint at the response selection stage (Logan & Gordon, 2001; Meyer & Kieras, 1997; Navon & Miller, 2002; Tombu & Jolicoeur, 2003). Among these, capacity-sharing models are prominent examples (Kahneman, 1973; Navon & Gopher, 1979; Norman & Bobrow, 1975; Tombu & Jolicoeur, 2003; Wickens, 1984). They assume that central processing capacity is limited, but can be flexibly shared like a common resource among the respective central processes of multiple tasks. Thus, in the context of the PRP effect, parallel response selection is possible, but because resources are limited, the processing efficiency of each task is reduced during the time of shared processing

<sup>2</sup>There has been much debate about exactly which part of task processing constitutes the bottleneck, and even multiple bottlenecks have been proposed (De Jong, 1993; Johnston & McCann, 2006; Jolicoeur & Dell'Acqua, 1998; Koch & Rumiati, 2006). Since the present dissertation is concerned with interference at the response level, I will make the simplifying assumption that response selection is the core central limitation of the human cognitive system. Furthermore, not all of the following models describing processing limitations are as specifically concerned with the response selection stage as the RSB model is. Thus, depending on the theory, I will sometimes use the broader term "central processing" when describing the core processes that ensure the correct translation of a stimulus into a response. For the purpose of this dissertation, however, we can use the narrower term "response selection" as a synonym for the broader term "central processing" without much loss in precision.

(e.g., when the SOA is small, see Figure 1B). Capacity-sharing models gain plausibility from the slight increase in RT1s for short relative to long SOAs that can sometimes be observed (Tombu & Jolicœur, 2005). This is because capacity sharing reduces T1 processing efficiency, and the shorter the SOA, the longer the capacity is shared. In contrast, the RSB model expects T1 processing to be independent of a particular SOA. Note, though, that capacity-sharing models include the RSB model as a special case when resources are allocated strictly sequentially (i.e., first 100% to T1, then 100% to T2).

An interesting notion in many capacity-sharing models is that participants have some control over the allocation of resources (Navon & Gopher, 1979). In other words, there is an executive component to the decision about how to schedule and process both tasks. While in some situations participants may allocate resources more sequentially, in others they may be more motivated to share them (e.g., Brüning et al., 2020; Fischer & Plessow, 2015). Such an involvement of executive control processes in dual-tasking is a prominent assumption (see Logan & Gordon, 2001; Luria & Meiran, 2006; Meyer & Kieras, 1997), and by far not unique to capacity-sharing models. For example, the *Executive Control of Theory of Visual Attention* (ECTVA, Logan & Gordon, 2001) assumes that instructions about tasks determine a set of control parameters that shape how stimuli are categorized, selected, and translated into a response. Importantly, ECTVA allows attention to gradually shift between tasks by biasing the processing of each stimulus based on a particular property (e.g., its location or color). Interestingly, given two sets of S-R rules (one for each task) and two stimuli that can be categorized under both sets, ECTVA assumes that it is impossible to determine which stimulus goes with which response, unless attention is selectively directed to a particular stimulus feature of a given task (such as the stimulus' location). Logan and Gordon (2001) refer to this problem as the dual-task binding problem, and suggest that focusing first on S1 until R1 and then on S2 until R2 in a serial fashion naturally solves this problem. In other words, serial response selection, as shown in PRP experiments, is not the result of a strict RSB, but rather a strategic choice (see also Meyer & Kieras, 1997; Miller et al., 2009; Tombu & Jolicœur, 2003, for related ideas and discussions).

According to the original RSB model, T1 and T2 processing are largely independent. However, if parallel central processing is possible, as proposed by capacity-sharing models and alike, it is intuitive to assume that unwanted information exchange between tasks may occur. In fact, such effects are regularly observed and summarized under the term *crosstalk*.<sup>3</sup> On a larger scale, crosstalk between tasks can occur for several reasons. One reason is that their stimuli, responses, or associated sensory effects are represented in overlapping modalities (Hazeltine et al., 2006; Ruthruff et al., 2006; Schacherer & Hazeltine, 2021; see also Wickens, 2002). For example, combining a task with visual stimuli and manual responses with a task involving auditory stimuli and vocal responses results in better performance than combining two tasks that both use only visual stimuli and manual responses.

Crosstalk can also occur when tasks have a dimensional overlap at the stimulus or response level, causing tasks (and their respective S-R rules) to rely on content that may be either compatible or incompatible. For example, if R1 is a key press to the left and R2 is a key press to the right, simultaneous activation of both (incompatible) response codes creates interference between tasks (i.e., crosstalk). Much research has been devoted to crosstalk effects in dual-tasking, because they allow inferences about a task's content being present in parallel to processes of another task. For example, crosstalk at the response level may affect not only T2 but also T1, suggesting that T2 response information must be partially present during T1 processing. Clearly, such a (backward) crosstalk effect of T2 on T1 is an important phenomenon to consider when theorizing about the mechanisms underlying dual-tasking.

### 3 Backward Crosstalk

The following section delves into crosstalk effects that demonstrate an influence of T2 on T1 (i.e., BCEs). In a broader sense, there are various manifestations of BCEs, and the scientific community is just beginning to classify and explain each of them (Durst & Janczyk, 2019); ideally within a unified framework in the future. To set the stage, I will

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<sup>3</sup>Analogous to the technical term "crosstalk" in older telephone connections, where another conversation can be quietly heard through parallel circuits.

first provide a more general overview of earlier studies on the BCE and briefly point out the various conditions under which a BCE can be observed. Following this, I will then focus on the so-called *response-based* BCE and discuss the conceptual models developed to explain it. This then provides an entry point into the overarching goals of the dissertation (Chapter 4).

### 3.1 Previous Studies and Different Types

One of the first systematic studies on the BCE was conducted by Hommel (1998), who aimed to test whether T2 response information is indeed not present until T1 response selection is complete, as proposed by the RSB model. In his first experiment, participants were presented with colored letters. R1 was a left or right button press to the color (S1), and R2 was the utterance of the word "left" or "right" to the identity of the letter (S2). Since this implies a dimensional overlap at the response level in terms of spatial locations, R1 and R2 could be either compatible (e.g., pressing the left button for T1 and saying "left" for T2) or incompatible (e.g., pressing the right button for T1 and saying "left" for T2). The important finding was that not only R2s but also R1s were slower and more error-prone in incompatible trials relative to compatible trials, and this net performance difference constitutes the BCE proper. Moreover, Hommel (1998) showed that the BCE is present even for the longest *interresponse intervals* and fastest R1s. This implies that T2 response information must be present during T1 processing, even before an R2 is actually selected. In other words, an S-R translation for T2 appears to occur despite a putative RSB.<sup>4</sup>

#### 3.1.1 Compatibility-Based Backward Crosstalk

Following Hommel's original result, many researchers have demonstrated influences of T2 on T1 processing in different ways. BCEs created by the dimensional overlap at the

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<sup>4</sup>An intuitive explanation for the BCE, which would still be consistent with an RSB, is that T2 response selection interferes with T1 motor execution. However, such an explanation is more difficult to reconcile with the observation of a continuously decreasing BCE with larger SOAs. Additionally, studies by Janczyk, Renas, and Durst (2018) and Thomson et al. (2015) provided evidence against a locus of the BCE at the motor stage (see the next sections below).

response level, as just introduced, can be referred to as *response-based* or *R1-R2* BCEs. They are well researched and have been demonstrated and replicated with many different types of responses (Durst & Janczyk, 2019; Ellenbogen & Meiran, 2011; Huestegge et al., 2018; Janczyk, 2016; Janczyk et al., 2014; Koob et al., 2020, 2024; Miller & Durst, 2014; Naefgen et al., 2017; Renas et al., 2018; Röttger et al., 2021; Scherbaum et al., 2015; Schonard et al., 2020, 2023; Thomson et al., 2010, 2015; Watter & Logan, 2006).

However, a BCE can also arise when R2 is conceptually (in)compatible with S1 (i.e., an *S1-R2* BCE; Durst & Janczyk, 2019; Ellenbogen & Meiran, 2008; Hommel, 1998; Hommel & Eglau, 2002; Koob et al., 2020; Lien et al., 2007). Although this type of BCE has received relatively little attention in recent years, Hommel (1998) already demonstrated its existence. In his second experiment, participants were again presented with colored letters. This time, however, they made a manual response to the color (S1) and a vocal utterance of "red" or "green" (R2) to the letter identity. Thus, in Experiment 2, R2 was either conceptually compatible or incompatible with S1. The results showed slower and more error-prone responses in (S1-R2) incompatible trials compared to (S1-R2) compatible trials.

S1-R2 and R1-R2 BCEs are summarized as compatibility-based BCEs (Durst & Janczyk, 2019; Janczyk, Renas, & Durst, 2018). They arise from the dimensional overlap of T1 and T2 at the response and/or stimulus level, which results in both tasks operating on content that may be conceptually compatible or incompatible. Although the BCEs as introduced by Hommel (1998, Exp. 1 and 2) are prototypical examples of compatibility-based BCEs, it is not uncommon for BCEs to involve overlap between tasks at multiple levels simultaneously (Fischer et al., 2014, 2018; Logan & Delheimer, 2001; Logan & Schulkind, 2000; Naefgen et al., 2022). For example, both S1 and S2 might be digits, while R1 and R2 indicate magnitude and parity judgments, respectively. If both responses conceptually overlap (e.g., both R1 and R2 refer to left- vs. right-oriented responses), a BCE may arise because S2 is involuntarily evaluated under the S1-R1 rule, or because R2 interferes with R1 at the level of response codes (see Rieger & Miller, 2020, for evidence favoring the former as the primary cause of interference, at least in their particular setup).



### 3.1.2 Backward Crosstalk of T2 Response Quality

In addition to these compatibility-based BCEs caused by dimensional overlap, another set of qualitatively different BCEs has been demonstrated that crucially depends on the characteristics of R2. For example, Miller (2006) used a go/no-go task for T2 and showed that T1 responses are faster for T2 go relative to no-go trials (for replications, see Durst & Janczyk, 2018, 2019; Durst et al., 2019; Janczyk & Huestegge, 2017; Ko & Miller, 2014; Mahesan & Fischer, 2024; Mahesan et al., 2021; Miller & Durst, 2014; Röttger & Haider, 2017; Schonard et al., 2023). The standard interpretation is that the inhibition of a prepared R2 spills over to T1, resulting in slower R1s (e.g., Miller, 2006). Similarly, Miller and Alderton (2006) used force-sensitive keys with T2 requiring either a hard or soft key press. R1s were harder when R2 was a hard compared to a soft key press (see also Ruiz Fernández & Ulrich, 2010, for a similar influence of T2 movement distance on T1). In short, a BCE can thus also refer to (qualitative) T2 response characteristics that spill over to T1.

### 3.1.3 Interim Summary and Discussion

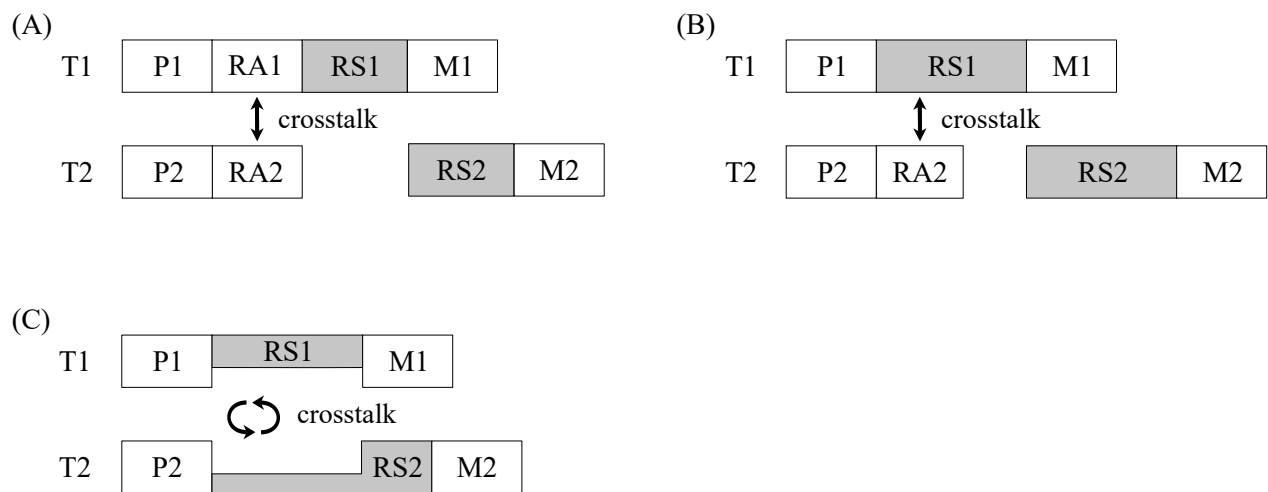
The previous elaboration has shown that, while all BCEs are characterized by an influence of T2 on T1, they can arise from different manipulations and possibly for different reasons. In fact, there has been a recent and still unsettled discussion about whether no-go BCEs and compatibility-based BCEs fundamentally differ in their origin from T2 processes and their locus during T1 processing (Durst & Janczyk, 2018, 2019; Janczyk, Renas, & Durst, 2018; Mahesan & Fischer, 2024; Röttger & Haider, 2017; Schonard et al., 2023). In the present dissertation, however, I am concerned only with the R1-R2 BCE, and will therefore limit the discussion of the underlying mechanisms to this particular BCE. Readers interested in the differences and similarities between no-go and compatibility-based BCEs are referred to the literature just cited. Furthermore, unless explicitly stated otherwise, I will use "BCE" to refer to the R1-R2 BCE for simplicity.

### 3.2 Conceptual Models for Response-Based Backward Crosstalk

Arguably, the BCE seems difficult to reconcile with a strict RSB at first glance, since some R2-related information must be present and interacting with T1 even while T1 processing is still ongoing. At the same time, experiments on the BCE still observe a PRP effect, which is consistent with an RSB. In the literature, two classes of conceptual models are typically considered that can simultaneously explain both the BCE and the PRP effect: Models that distinguish between response selection and response activation, and models that assume (central) capacity sharing. I will start with the former.

Some researchers have suggested extending the RSB model to include an additional stage of response activation. Specifically, Hommel (1998) divided the response selection stage into an initial stage of response activation and a final stage of response selection. While response activation involves the activation of response codes via S-R rules (but not beyond selection), response selection is concerned with the "response activation pattern" (p. 1381) of all S-R rules to guide the final decision. Importantly, according to this model, response activation can occur in parallel (i.e., it is capacity-unlimited), causing crosstalk when the temporal overlap between tasks is high (see Figure 2A, and also Lien & Proctor, 2002; Schubert et al., 2008, for similar descriptions). Consequently, a BCE would result from a longer T1 response activation stage in incompatible relative to compatible trials.

Following the initial suggestion by Hommel (1998), other researchers have attempted to further define the source and locus of the BCE. Although these studies agree with a stage or phase of parallel, capacity-unlimited T2 response activation which runs prior to T2 response selection, they found no evidence that the BCE occurs during T1 response activation. Instead, they located the BCE during T1 response selection. For example, Janczyk, Renas, and Durst (2018) conducted a series of experiments using the so-called locus of slack and effect propagation logic, which, in combination, allows to locate the origin of an effect either prior to, during, or after response selection (see Fischer & Janczyk, 2022, Figures 2 and 3, for instructive illustrations). In Experiments 1-3, they leveraged the locus of slack logic by presenting a third, "unrelated" task prior to T1 and T2, and observed that the BCE remained constant when the SOA was varied between the unrelated

**Figure 2***Illustration of Three Conceptual Models Capable of Explaining the BCE*

*Note.* (A) A model as introduced by Hommel (1998). Crosstalk, and thus a BCE, occurs when T1 and T2 response activation (RA) overlap in time. (B) A modified version by Janczyk, Renas, and Durst (2018, see also, Miller, 2017; Thomson et al., 2015), where crosstalk occurs between T2 RA and T1 response selection (RS). (C) A capacity-sharing model (identical to Figure 1B), where crosstalk is a natural byproduct of parallel running RS. Note that the horizontal width of the "RS box" varies across panels A-C for illustrative purposes only.

third task and T1. This indicates that the BCE has its locus either during or after response selection. In Experiment 4, Janczyk and colleagues then used the effect propagation logic by reversing parts of the task order, presenting T1 and T2 (i.e., the BCE measurement and induction tasks) simultaneously before the "unrelated" third task. The BCE in T1 fully propagated to the third task, regardless of the SOA between them. This suggests that the BCE has its locus either during or prior to response selection. Taken together, Experiments 1-4 by Janczyk, Renas, and Durst (2018) provide compelling evidence that the BCE occurs during T1 response selection, with its source in T2 response activation, and other studies have reached similar conclusions (Durst & Janczyk, 2019; Miller, 2017; Thomson et al., 2015). According to this conceptualization, when T1 and T2 are presented in close temporal proximity, T2 response activation overlaps with T1 response selection, thereby prolonging T1 response selection in incompatible relative to compatible trials (see Figure 2B).

When conceptualizing the BCE as T2 response activation influencing concurrent T1

response selection, then this bears a striking similarity to the mechanisms at work in conflict tasks (B. A. Eriksen & Eriksen, 1974; Simon, 1969; Stroop, 1935), particularly in the flanker task (B. A. Eriksen & Eriksen, 1974). In the flanker task, participants respond to a central target that is flanked by irrelevant, distracting stimuli. In congruent trials, the central letter and adjacent flankers indicate the same response, whereas in incongruent trials they indicate opposite responses. As with the BCE, responses are slower and more error-prone in incongruent relative to congruent trials; known as the *congruency effect*.<sup>5</sup> Importantly, the congruency effect is usually explained as an interaction between controlled target processing and involuntary, automatic distractor (e.g., flanker) activation (De Jong et al., 1994; Kornblum et al., 1990; Ridderinkhof, 2002). Thus, given this similarity in the mechanisms at work for both the BCE and the congruency effect, and given that T2 is not (yet) relevant during T1, it has previously been suggested that S2 may act as a distractor during T1 processing, similar to flankers in the flanker task (see Durst & Janczyk, 2019, p. 133; Janczyk, Renas, & Durst, 2018, p. 273; and also Miller & Alderton, 2006, p. 161).

It is important to note, however, that a simple explanation for the BCE might also come from capacity-sharing models (see Chapter 2). Rather than insisting on a strictly serial response selection process and separating it from T2 response activation, these models allow for simultaneous T1 and T2 response selection. Conceptualized this way, the BCE becomes a natural byproduct of parallel response selection (see Figure 2C). Consequently, the more central capacity is shared between tasks, the more crosstalk occurs on average.

Disentangling capacity-sharing models from those assuming T2 response activation is a notoriously difficult task, and all the experiments reviewed so far are compatible with both models. This is also partially true for the studies included in this dissertation. However, resolving this debate is beyond the scope of the present dissertation. Furthermore, a cornerstone of this dissertation is the specification of T2 response activation and how it

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<sup>5</sup>While the term "compatibility" is commonly used in the BCE literature, "congruency" is often used in the conflict task literature. I will use one or the other depending on the literature under discussion, although both can be considered synonymous in the context of this dissertation.

behaves differently in dual-task versus conflict tasks (such as the flanker task). As such, I will only discuss and summarize the enclosed studies in Chapter 4 in terms of a model that assumes T2 response activation. Nevertheless, I will later relate the overarching results of this dissertation to capacity-sharing models in the General Discussion in Chapter 5.

## 4 Specifying T2 Response Activation: Overarching Research Questions and Summary of Enclosed Manuscripts

In the following chapter, I will motivate three questions related to the R1-R2 BCE. I will then introduce the mathematical framework of *Drift-Diffusion Models* (DDMs) that will be fundamental for answering two of these three research questions. Finally, I will summarize my findings with respect to each research question.

### 4.1 Overarching Research Questions

As reviewed above, many studies have investigated BCEs over the past decades, providing conceptual models against the background of general processing limitations. Surprisingly, however, while the idea of T2 response activation as the source of the BCE seems generally accepted, little is actually known about the temporal development of T2 response activation and its subsequent influence on T2 response selection. The present dissertation aims to provide some of these missing pieces, thereby contributing to a more accurate understanding of the BCE within the context of human multitasking.

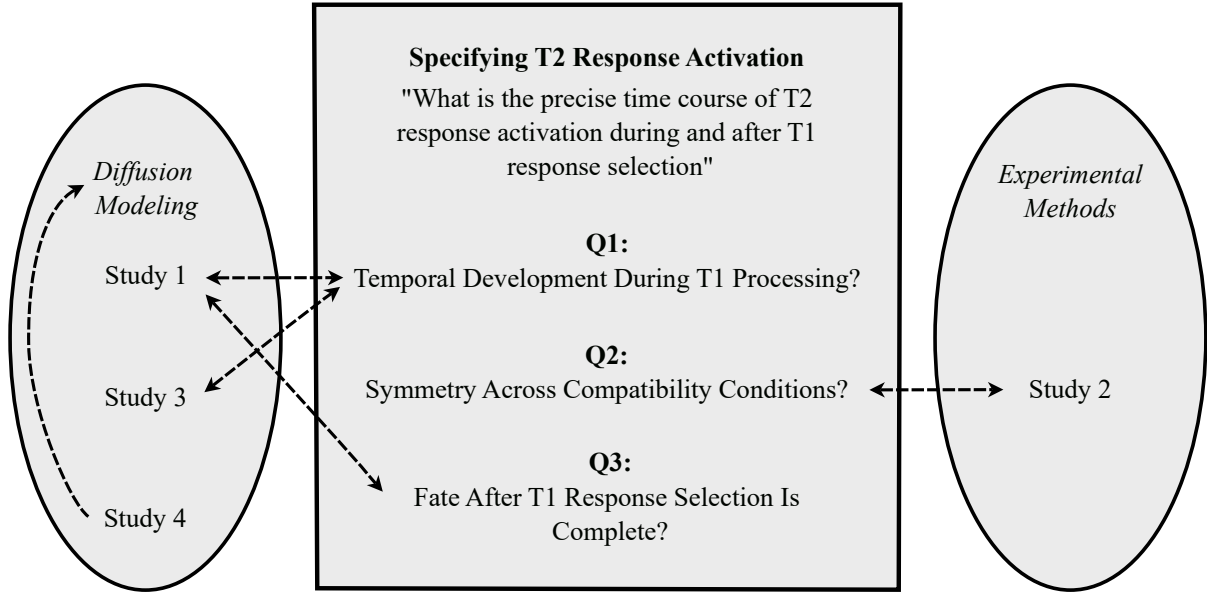
On a broader level, the present dissertation builds on the work by Janczyk, Renas, and Durst (2018, see Figure 2B) and Durst and Janczyk (2019), posing the following main question: What is the precise time course and influence of T2 response activation during and after T1 response selection? This overarching question was related to three subordinate research questions that were addressed in three studies (see also Figure 3 for an illustration of how each study relates to the research questions):

- **Q1:** How does T2 response activation develop over time during T1 response selection? Is this activation more transient, since T2 is not (yet) relevant and potentially detrimental to T1 performance? Or is it more monotonically increasing, as T2 will

soon be relevant and is thus prepared? This question was addressed by explicitly modeling different time courses of T2 response activation within a DDM framework (Study 1 and Study 3).

- **Q2:** Regardless of its time course, can we expect T2 response activation to be similar in compatible and incompatible trials? Or, put differently, does T2 response activation facilitate T1 performance as much as it impedes T1 performance? To this end, we conducted a series of experiments with a "neutral" condition in which R2 did not spatially overlap with any of the R1s (Study 2).
- **Q3:** What happens to T2 response activation when switching from T1 to T2 response selection? Does T2 response activation contribute to subsequent T2 response selection or is it reset once T1 response selection is complete? This question was again addressed in a DDM framework by exploring different theory-driven T2 activation transmission mechanisms (Study 1).

As just outlined, DDMs served as the primary approach to tackle Q1 and Q3. The methods required and developed in the course of this dissertation led to a collection of programs that ultimately resulted in a tutorial paper and the R package *dRiftDM* for fitting DDMs with time-dependent parameters. This tutorial paper is also included in the current dissertation as "Study" 4, although it is not directly related to the BCE. In the next section, I will provide a short introduction to DDMs and quickly summarize *dRiftDM*'s core features and advancements to the field of psychology. Afterwards, each subordinate research question and its related studies will be discussed in separate subsections.

**Figure 3**
*Illustration of the Main and Subordinate Research Questions*


## 4.2 Diffusion Models and the R Package dRiftDM

DDMs are stochastic models that describe the response selection process in binary choice tasks as a noisy response evidence accumulation (i.e., diffusion) process (Ratcliff, 1978; Ratcliff et al., 2016; Voss et al., 2013, 2015). Put simply, they assume that a decision process  $\{X(t), t \geq 0\}$  continuously evolves over a one-dimensional, real-valued time space, accumulating response information for one response or the other. As an approximation, a difference equation provides an accessible way to describe it. Starting from an initial value  $X(0) = x_0$  ( $x_0 \in \mathbb{R}$ ), the process evolves for each time step  $t_n$  ( $n \in \mathbb{N}$ ) with step size  $\Delta t$  as follows:

$$X(t_{n+1}) = X(t_n) + v(t_n) \cdot \Delta t + \sqrt{\Delta t} \cdot \sigma(t_n) \cdot Z(t_n), \quad t_{n+1} = t_n + \Delta t \quad (1)$$

Here,  $X(t_n)$  represents the accumulated response information up to the current step, which is then modified by two parts: First, a deterministic part, reflected by the drift rate  $v(t)$ , "nudging" the process up or down (while taking into account the step size,

see the term  $v(t_n) \cdot \Delta t$ ). Second, a random part, which in the context of a difference equation, is a standard normal distribution (i.e.,  $Z(t) \stackrel{iid}{\sim} N(0, 1)$ ). It will result in random disturbances of the process at every time step. The size of this random disturbance is controlled by the so-called diffusion coefficient  $\sigma(t)$ , which is further appropriately scaled with respect to the step size  $\Delta t$  (see the term  $\sqrt{\Delta t} \cdot \sigma(t_n) \cdot Z(t_n)$ ).<sup>6</sup>

When the process  $\{X(t), t \geq 0\}$  hits an upper or lower (absorbing) decision boundary,  $b(t)$  or  $-b(t)$ , a decision for one or the other response is registered, and a corresponding motor program is executed. While the boundaries may refer to response alternatives, such as "left" or "right," they often code accuracy (e.g., Durst & Janczyk, 2019; Fröber & Lerche, 2023). In this case, the upper decision boundary  $b(t)$  usually reflects a correct response, while the lower decision boundary  $-b(t)$  reflects an incorrect response.

It is important to note that the drift rate  $v(t)$ , the diffusion constant  $\sigma(t)$ , and the decision boundary  $b(t)$  may be time-dependent, expressed by the " $(t)$ " in the notation. For example, a common assumption in the context of conflict tasks is that the influence of the irrelevant stimuli on the decision process diminishes, so that the drift rate of the (overall) decision process varies over the course of a trial (Ulrich et al., 2015; White et al., 2018). Oftentimes, however, especially outside the conflict task literature, the core components of the decision process are assumed to be time-independent (Ratcliff, 1978; Voss et al., 2013), in which case  $v(t) = v$ ,  $b(t) = b$ , and  $\sigma(t) = \sigma$  follows. A visualization of such a basic variant with time-independent parameters is given in Figure 4. Since Roger Ratcliff (1978) was the first to introduce this DDM to psychology, it is sometimes called the "Ratcliff DDM."

In a psychological context, the time it takes for a diffusion process to reach one of the two (absorbing) boundaries reflects the duration of the response selection process. Obviously, responding to a stimulus requires more than just central response selection. Thus, to capture more "peripheral" time requirements, such as stimulus perception and response execution (see Figure 1), a non-decision time  $t_0 > 0$  (sometimes known as  $T_{er}$ )

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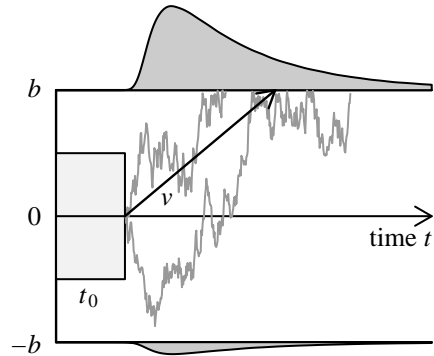
<sup>6</sup>The term  $\sqrt{\Delta t}$  ensures that smaller step sizes lead to relatively smaller increments in the random part. Otherwise, with  $\Delta t \rightarrow 0$ , the variance of the noise would run to infinity for any non-zero time interval.



is included.

**Figure 4**

*Schematic Outline of a Basic Diffusion Model*



*Note.* This diffusion model assumes all parameters to be time-independent, and is thus a special case of the more general diffusion model framework introduced in Equation 1. The slope of the black arrow shows the (constant) drift rate  $v(t) = v$ . The (constant) decision boundaries  $b(t) = b$  and  $-b(t) = -b$  indicate correct and incorrect responses, respectively. Two exemplary trajectories of evidence accumulation starting at  $x_0 = 0$  are shown by the jagged gray lines. The time required for more peripheral processing is shown by the gray square and quantified by the parameter  $t_0$ . The gray distributions above and below the boundaries indicate the probability density functions of correct and incorrect responses, respectively.

A key challenge in modeling data with a DDM is to derive a summary or full *Probability Density Function* (PDF) of the model's predicted RTs (see Figure 4), that is, the sum of the time required for central response selection and peripheral processes. Including the contribution of the latter, reflected by the non-decision time, is usually not a problem. However, deriving the contribution of the stochastic response selection process is both mathematically and numerically challenging. This is because the duration of the response selection process results from the underlying diffusion process, and thus from the time it takes for the (noisy) diffusion process to reach one of the two absorbing boundaries. Technically, this problem refers to deriving the *first passage time* in the form of summary statistics or a full PDF.

While closed-form solutions of the first passage time are available for some cases (Schwarz, 2022), in most cases, especially when deriving the full PDF, such solutions are unavailable. In these instances, complex numerical approximations are required, such

as those implemented in *fast-dm* (Voss et al., 2013). However, previous, more advanced "out-of-the-box" software solutions were limited to models with time-independent parameters and did not apply, for example, to models with time-dependent drift rates, which are common in cognitive psychology (e.g., Ulrich et al., 2015; White et al., 2011). As a result, psychology researchers have often resorted to Monte Carlo simulations (e.g., Evans & Servant, 2022; Luo & Proctor, 2022; Mackenzie & Dudschig, 2021; Mittelstädt et al., 2023; White et al., 2018), where the model is explicitly simulated  $N$  times over the discretized time interval in accordance with Equation 1. While this is the most flexible approach, it has significant drawbacks, most notably its inefficiency and low accuracy.

Fortunately, the dynamics of diffusion models are well understood in mathematics and physics, with origins dating back to the 19th century (Fick, 1855). Thus, there are several sophisticated approaches for deriving the model's first passage time, although their implementation requires advanced mathematical and programming skills. One such approach is based on the *Kolmogorov Forward Equation* (KFE), which relates changes in the diffusion process over time  $t$  to changes in the evidence state  $x$ .<sup>7</sup> A detailed discussion of the KFE and its subsequent numerical discretization is beyond the scope of this dissertation, but interested readers are referred to Richter et al. (2023) and Shinn et al. (2020).

Over the course of my dissertation, an R package (*dRiftDM*; Study 4) was developed for fitting diffusion models based on a numerical discretization of the KFE provided by Richter et al. (2023). It is tailored to the needs of (cognitive) psychology researchers and serves as an entry point for applying DDMs with time-dependent parameters. While similar packages have been released in recent years (e.g., *PyDDM*, Shinn et al., 2020, and *PyBEAM*, Murrow & Holmes, 2024), which also rely on the KFE, these packages require users to be familiar with Python. Additionally, common cognitive psychology models, such as the *Diffusion Model for Conflict Tasks* (DMC) or the *Shrinking Spotlight Model* (Ulrich et al., 2015; White et al., 2011), may first need to be implemented, and the results

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<sup>7</sup>There is also its counterpart, the *Kolmogorov Backward Equation*. From a purely mathematical perspective, the two are interchangeable, but the backward equation allows for highly efficient numerical discretization when the parameters of a DDM are time-independent (which provides the basis for *fast-dm* Voss et al., 2013).

typically required by psychological researchers extracted manually. Nevertheless, both PyDDM and PyBEAM represent significant advances in solving more complex DDMs.

A detailed introduction to dRiftDM can be found in our tutorial paper (see Study 4). In short, it allows users to either select a pre-built model or build and customize their own. The key to its flexibility is that each model depends on a set of functions that provide the model's "components" (i.e., drift rate, boundary, etc.) and a set of parameters. By swapping out the parameters and the underlying component functions, users can create custom DDMs. Each model can then be passed to various functions to explore the model, compute summary statistics, or fit individual data. Note that dRiftDM also served as the basis for Study 3. A limiting factor, however, is that trial-by-trial variability in the drift rate is not yet possible, and the time course of the drift rate and the decision boundary must be calculable prior to a trial.

With this background on DDMs in mind, I will now address each research question and study of this dissertation. A central goal related to Q1 was to jointly model T1 response selection and T2 response activation as interacting evidence accumulation processes, with a particular focus on the time course of T2 response activation.

### **4.3 Q1: Modeling the Time Course of T2 Response Activation**

In many publications, the activation of the T2 response is often considered "automatic" (e.g., Hommel, 1998; Janczyk, Renas, & Durst, 2018; Miller & Alderton, 2006), meaning that S2 automatically, and perhaps involuntarily, activates R2 via its associated S-R rule. In fact, a BCE can be observed even when T2 does not (or no longer) require a response (Hommel, 1998; Janczyk, Renas, & Durst, 2018; Miller & Durst, 2014), which supports the "automaticity" with which T2 information may be activated. From this perspective, and as was already mentioned in Chapter 3, S2 may act similar as flankers do in the flanker task (see Durst & Janczyk, 2019, p. 133; Janczyk, Renas, & Durst, 2018, p. 273; and also Miller & Alderton, 2006, p. 161). In other words, the BCE arises because S2 serves like a not (yet) relevant distractor, creating interfering response activation with respect to the currently relevant T1. Consequently, the time course of T2 response activation might

coincide with that of flanker response activation in conflict tasks.

A prominent model used to quantify the interaction between flanker and target response activation is DMC (Ulrich et al., 2015). It conceptually follows the dual-route logic (De Jong et al., 1994; Kornblum et al., 1990; Ridderinkhof, 2002), which proposes that the target is processed via a *controlled* route and the flankers via an *automatic* route, while the overall decision is based on a merger of both. Importantly, DMC explicitly models this target and flanker processing as diffusion processes with distinct time courses. Response activation resulting from the controlled process increases linearly, as in the Ratcliff DDM (Ratcliff, 1978, see Figure 4). In contrast, response activation from the automatic process is transient, rising, and then falling back to zero, resembling a pulse-like function. The overall decision process, which reflects the interaction between controlled and automatic processing, is the sum of both subprocesses.

Given the similarity between the BCE and the congruency effect in the flanker task, it is reasonable to suggest that DMC, with its proposed interaction between automatic and controlled processing, could effectively model the influence of T2 response activation on T1 response selection. However, this would imply that the automatic process (i.e., T2 response activation in the dual-task context) is transient. In flanker tasks, this assumption is easily justified, as flankers are inherently irrelevant, and their corresponding response information may either passively decay or be actively suppressed (e.g., Hommel, 1994; Miller & Schwarz, 2021; Ridderinkhof, 2002). In contrast, T2 in any dual-task will eventually require a response, making it never completely irrelevant. Indeed, ample evidence suggests that T2 response activation is stronger and longer-lasting with greater T2 relevance (Miller & Tang, 2021; Mittelstädt et al., 2023). Thus, instead of following a pulse-like time course, T2 response activation may increase more monotonically, without interruption or decay. As a first approximation, it may be considered linear.

In two studies (Study 1 and 3), we addressed the question of whether T2 response activation is transient or not. While Study 1 provided first insights, Study 3 provided further results and addressed open questions using a more sophisticated modeling approach.

The DDMs considered in Study 1 relied heavily on DMC’s architecture. We assumed

the expected (overall) decision process for T1, denoted as  $V(t)$ , is the sum of two subprocesses, each starting from zero:

$$V(t) = V_{T1,rs}(t) + V_{T2,ra}(t) \quad \text{with} \quad V_{T1,rs}(0) = V_{T2,ra}(0) = 0 \quad (2)$$

Here,  $V_{T1,rs}$  refers to the expected time course of *T1* response selection, which was always assumed to be linear,

$$V_{T1,rs}(t) = v_{rs} \cdot t \quad . \quad (3)$$

The parameter  $v_{rs}$  reflects the slope of expected T1 response selection. In contrast, expected *T2* response activation,  $V_{T2,ra}$ , was modeled as either linear,

$$V_{T2,ra}(t) = v_{ra} \cdot t \quad , \quad (4)$$

or pulse-like,

$$V_{T2,ra}(t) = A \cdot e^{-\frac{t}{\tau}} \cdot \left( \frac{t \cdot e}{\tau} \right) \quad , \quad (5)$$

and both situations are visualized in Figure 5. For the linear function,  $v_{ra}$  represents the slope of expected T2 response activation. For the pulse-like function,  $A$  and  $\tau$  represent the amplitude and peak latency of T2 response activation, respectively.<sup>8</sup>

Importantly, the sign of T2 response activation depends on the compatibility of a trial. In compatible trials, T2 response activation contributes evidence for the same, correct response (positive sign), while in incompatible trials, it provides evidence for the

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<sup>8</sup>The pulse-like function is a scaled gamma-distribution density function. Equation 5 can be derived from the original equation presented by Ulrich et al. (2015, Appendix B), by setting the shape parameter of the gamma-distribution function to  $a = 2$ .

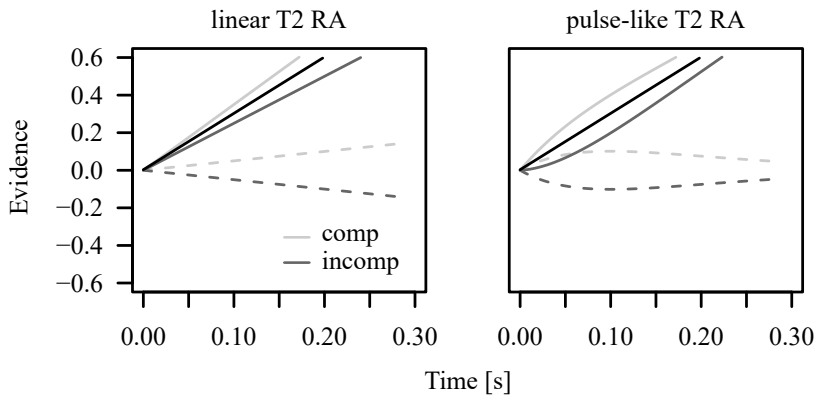
other, incorrect response (negative sign):

$$V(t) = \begin{cases} V_{T1,rs}(t) + V_{T2,ra}(t) & \text{in compatible trials} \\ V_{T1,rs}(t) - V_{T2,ra}(t) & \text{in incompatible trials} \end{cases} \quad (6)$$

Therefore, regarding the overall T1 decision process, performance is better in compatible relative to incompatible trials, as indicated by the solid gray line reaching the upper boundary earlier in time than the dark gray line in Figure 5.

**Figure 5**

*A Visualization of the Expected T1 Decision Process Modeled in Study 1*



*Note.* In all plots, the upper and lower limits indicate the constant decision boundaries  $b = 0.6$  and  $-b = -0.6$ . Dashed lines refer to the (expected) time course of T2 response activation (RA;  $V_{T2,ra}$ ), which provides evidence for either the correct response (positive sign) or the incorrect response (negative sign), depending on the compatibility of a trial (see Equation 6). While T2 response activation is linear in the left panel (see Equation 4), it is pulse-like in the right panel (see Equation 5). The solid black line shows T1 response selection ( $V_{T1,rs}$ ; Equation 3). The solid gray lines describe the overall T1 decision process, derived by adding both subprocesses. For simplicity, this figure does not show any random disturbances in the diffusion processes (see Equation 2). Parameters underlying this plot (with time unit in seconds):  $v_{rs} = 3$ ,  $v_{ra} = 0.5$ ,  $b = 0.6$ ,  $A = 0.1$ ,  $\tau = 0.1$ .

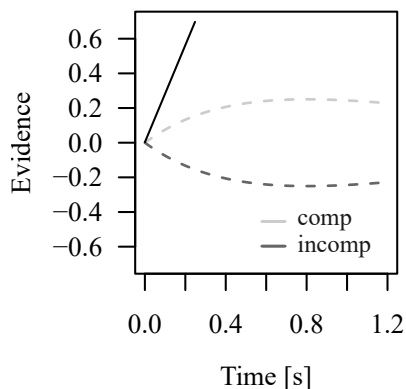
For completeness, we assumed a constant decision boundary and a normally distributed non-decision time for the remaining model components. Additionally, note that the first derivative of  $V(t)$  refers to the drift rate  $v(t)$  as introduced in Equation 1 (see also Janczyk et al., 2024, for a discussion on the interpretation of the drift rate).

To decide between the two models, we fitted them to five data sets from previously published experiments focusing on R1-R2 BCEs (Durst & Janczyk, 2019; Hommel, 1998;

Janczyk, Mittelstädt, & Wienrich, 2018; Janczyk et al., 2017; Koob et al., 2020). Surprisingly, a qualitative comparison of the models revealed more similarities than differences, especially for fast to moderate responses. Only for slow responses did the linear T2 response activation function systematically overestimate the BCE, likely because the linear function—and thus its associated activation—became too large. A closer examination of the estimated pulse-like function revealed why the model predictions of both activation functions were so similar: The pulse-like function peaked so late that activation increased almost linearly early in the trial and remained fairly constant for the rest of the trial (see Figure 6). In other words, the estimated pulse-like function exhibited an asymptotic nature, even resembling a continuous linear activation function when considering the average duration of T1 response selection. Thus, we concluded from Study 1 that T2 response activation was not transient and that its temporal development differed from the typical response activation pattern observed for flankers in a flanker task.

**Figure 6**

*A Visualization of Study 1’s Results*



*Note.* The plot shows the average (expected) T1 response selection and T2 response activation processes estimated in Study 1. The solid black line represents T1 response selection. The light and dark gray dashed lines show T2 response activation in compatible and incompatible trials, respectively. For simplicity, no overall T1 decision process is shown. Parameters underlying this plot (with time unit in seconds):  $v_{rs} = 2.8$ ,  $b = 0.7$ ,  $A = 0.25$ ,  $\tau = 0.8$ .

Although Study 1 provided first insights, there were several reasons to revisit Q1 in Study 3. First, because the more sophisticated numerical discretization of the KFE by Richter et al. (2023) was not yet available, model predictions in Study 1 were derived

using Monte Carlo simulations. Consequently, models were compared only qualitatively. Second, because the pulse-like function resembled an asymptotic function, it was worth exploring whether it could actually be replaced by such a function. If so, this would offer a more accurate model of T2 response activation. Third, in the original study, we noted a peculiarity regarding the peak latency of the pulse-like function (Koob et al., 2023, Footnote 6). For most participants, the parameter reflecting the peak latency was at the upper limit of the parameter space, while for others it was near the lower limit. Clarifying whether this bimodal distribution reflects individual differences or issues with the model itself is crucial for future modeling efforts. Finally, we aimed to provide practical guidance for quantifying the BCE using DDMs, particularly regarding the measurement properties of each model.

With this in mind, we conducted several follow-up analyses in Study 3. In the first part, we again compared a pulse-like and a linear T2 response activation function, but this time we also included an asymptotic function. This asymptotic function mirrored the charging curve of a capacitor from physics and is given by

$$V_{T2,ra}(t) = A \cdot \left(1 - e^{-\frac{t}{\tau}}\right) \quad . \quad (7)$$

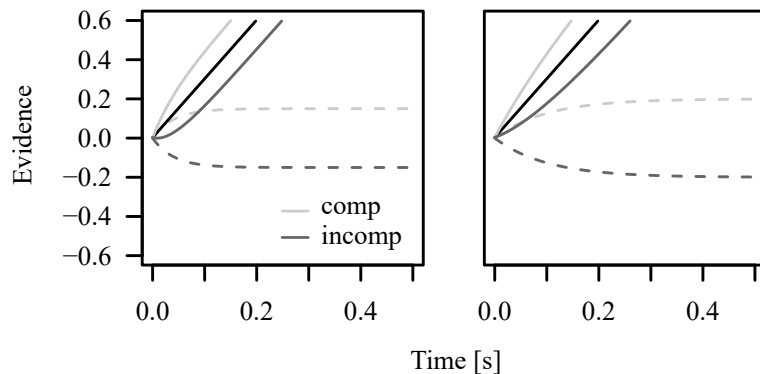
Here,  $A$  and  $\tau$  have conceptually the same role as in the previous Equation 5 for the pulse-like function. Larger values of  $A$  and  $\tau$  lead to a higher and later-occurring asymptote of T2 response activation (see Figure 7 for a visualization).

To formally compare the linear, pulse-like, and asymptotic T2 response activation function, we employed the more sophisticated approach implemented in dRiftDM. This allowed for maximum likelihood estimation and subsequent formal model comparison using the *Akaike Information Criterion* (AIC) and the *Bayesian Information Criterion* (BIC) as fit statistics. *Lower* AIC and BIC values indicate a *better* relative fit, taking into account both quantitative fit and the number of parameters in the model. The results clearly favored the linear function, although it slightly overestimated the BCE for slow responses as in Study 1. We reasoned that this was likely due to the small misfit not being enough to outweigh the additional parameter required by the pulse-like or asymptotic



**Figure 7**

*A Visualization of Asymptotic T2 Response Activation Considered in Study 3*



*Note.* The basic structure of this figure follows that of Figure 5. The main difference is that both panels show exemplary expected time courses of asymptotic T2 response activation according to Equation 7. In the left relative to the right panel, the asymptote is reached earlier and the maximum strength of T2 response activation is slightly smaller. Parameters underlying this plot (with time unit in seconds):  $v_{rs} = 3$ ,  $b = 0.6$ ,  $A = 0.15$  (left panel),  $A = 0.2$  (right panel),  $\tau = 0.04$  (left panel),  $\tau = 0.1$  (right panel).

function. Interestingly, the asymptotic function performed well qualitatively, but almost never outperformed the linear and pulse-like functions in terms of quantitative model fit. This may be because the asymptotic function is less flexible than the pulse-like function, while being too similar to the linear function, which requires one less parameter.

In the second part, we addressed the issue of individual differences in the peak latency of the pulse-like function, which reemerged in Study 3. Although the majority of individuals yielded lower fit statistics for a linear function, a subset (approximately 10 – 20%) yielded lower fit statistics for a pulse-like function, indicating a more transient time course of T2 response activation. This might suggest that some individuals treat S2—at least to some extent—as not (yet) relevant, perhaps indeed similarly to a flanker stimulus. To determine whether these differences were genuine or simply statistical noise, we generated synthetic data using a model with a linear T2 response activation function. This model was chosen because it was the most appropriate for the majority of individuals and, therefore, likely represents the “true” population model in the absence of individual differences. Subsequently, we fitted each model (with linear, pulse-like, or asymptotic T2 response activation) once more to these synthetic data. Assuming that the observed

individual differences are due to statistical noise resulting from noisy sampling under a linear model, we would expect the relative frequency with which the pulse-like function yields the smallest fit statistic (i.e., the "best" fit) to be similar for both the observed and synthetic data. However, this was not the case. The pulse-like function yielded the smallest fit statistic more frequently for the observed than the synthetic data. This suggests that individual differences in the time course of T2 response activation may indeed exist. Importantly, more detailed analyses of individual model predictions revealed these differences to be quantitative rather than qualitative. In other words, there is likely a continuum from more pulse-like to more linear T2 response activation, with the observed "subgroups" reflecting a dichotomization of this continuum.

In the third and final part of Study 3, we conducted a parameter recovery to evaluate the measurement properties of the pulse-like and linear T2 response activation functions. This is crucial because the parameters of any DDM should be reliable enough to allow researchers to make valid inferences based on individual parameter estimates. Parameter recovery also offers practical insights, such as the required number of trials, optimal settings for discretizing the KFE, or whether less meaningful parameters can be fixed to create more parsimonious models (e.g., fixing the standard deviation of the non-decision time). Our main findings and recommendations were as follows: A DDM with a linear T2 response activation function exhibits very good parameter recovery properties, even with as few as 100 to 200 trials per compatibility condition. In contrast, parameters associated with the pulse-like function show relatively poor recovery properties in the context of the BCE. Additionally, researchers can safely use a slightly coarser numerical discretization of the KFE when deriving model predictions without compromising the model's measurement properties, which is useful when computational resources are limited. Finally, fixing or dropping the standard deviation of the non-decision time to make the model more parsimonious is not recommended when fitting BCE data.

Taken together, Study 1 and Study 3 suggest that for most individuals, T2 response activation increases monotonically during T1 response selection, best described by a linear function. Therefore, the BCE in dual-task experiments does not behave like the congru-

ency effect in the flanker task, presumably because T2 always becomes relevant. However, there may be a subset of individuals who treat S2 of T2 as not (yet) relevant, and thus similar to a flanker stimulus. Yet, given the results of our parameter recovery study, it seems challenging to reliably quantify these individual differences using a pulse-like function.

While these results suggest that the BCE can be quantified using a DDM, our modeling procedure assumed that the strength of T2 response activation is equal in both compatible and incompatible trials (see Figures 5, 6, and 7). Whether this assumption holds was explored with experimental methods in Study 2.

#### 4.4 Q2: Symmetry of T2 Response Activation

When considering how T2 response activation influences T1 response selection, as shown in Figure 2, it is intuitive to assume that T2 response activation is equally strong for both compatible and incompatible conditions, albeit in opposite directions. In particular, if S2 activates R2 upon onset, it should do so independently of the current compatibility condition. Therefore, in the absence of additional control mechanisms or a compatibility-dependent processing architecture, T2 response activation should equally facilitate and interfere with T1 processing across conditions. In other words, T2 should enhance performance in compatible trials and disrupt it in incompatible trials, producing symmetric *facilitation* and *interference* effects in T1. Surprisingly, this "symmetry assumption" has received little attention, despite being a crucial factor when theorizing about the BCE.

To disentangle the unique contributions of facilitation and interference to the BCE, we need a condition without any crosstalk as a baseline, as it is common in the conflict task literature (e.g., T. L. Brown, 2011; Burle et al., 2002; Craft & Simon, 1970; C. W. Eriksen & St. James, 1986; Ila & Polich, 1999; Mahani et al., 2019; Miller, 1991). In these *neutral trials*, ideally, neither S2 nor its associated R2 overlap with the features that distinguish the R1 alternatives. At the same time, a high degree of similarity with the remaining response and stimulus sets of T1 and T2 has to be maintained. Given such a neutral condition, the interference effect is then the difference in performance between the

incompatible and neutral conditions, while the facilitation effect is the difference between the neutral and compatible conditions. To my knowledge, only one study by Naefgen et al. (2022) included a neutral condition in the context of R1-R2 BCEs. However, while this study demonstrated a greater interference than facilitation effect in T1, it was not explicitly designed to test the symmetry assumption. Notably, there were twice as many neutral trials as (in)compatible trials in this study, so its implications should be treated with some caution.

Besides addressing a neglected question in the BCE literature, an empirical investigation of the relative contribution of the facilitation and interference effects is valuable for two reasons. First, related experiments from the conflict task literature often reveal asymmetric effects, where the neutral condition is not equally distant from the congruent and incongruent conditions. For instance, P. Smith and Ulrich (2024) recently reviewed 52 studies and concluded that the interference effect is often larger than the facilitation effect (see their Table 1). This aligns with the aforementioned findings of Naefgen et al. (2022). Similarly, Evans and Servant (2022) used DMC to show that the amplitude of the automatic process in Simon and flanker tasks tends to be larger for incongruent than congruent trials. Thus, they argue that the automatic process primarily enforces interference, rather than facilitation. Second, the simplified box-like figures commonly used in the dual-task literature (see, e.g., Figure 2) do not account for the precise temporal development of the interference effect, which may fundamentally alter a model's predicted facilitation and interference effects (cf. P. Smith & Ulrich, 2024). Given this, we considered it worthwhile to empirically disentangle the contribution of facilitation and interference to the BCE, as this not only helps to clarify how the BCE relates to conflict tasks, but also provides a key data set for testing future conceptual and computational models.

To create a neutral condition, we combined a two-choice T1 with a three-choice T2. In each trial, participants were presented with a colored letter (S1 and S2) at the center of the screen. For T1, participants made a left or right button press (R1) based on the letter's color, while for T2, they verbally indicated the letter's identity by saying "left," "middle,"

or "right" (originally in German). We argued that the response "middle" for R2 serves as the neutral condition because it does not match with either of the R1s. Additionally, "middle" was conceptually equidistant from both the left and right responses for T1.

Our results from Experiment 1 revealed that the facilitation effect and the interference effect equally contribute to the BCE. Specifically, for RT1s, the neutral condition was equidistant from the compatible and incompatible conditions.<sup>9</sup> Surprisingly, however, this pattern did not hold for T2, where we only observed a facilitation effect but no interference effect in RT2s. Given that T2 response activation influences the duration of T1 response selection, these divergent results between T1 and T2 were puzzling. In particular, if T1 response selection is shorter in compatible trials and longer in incompatible trials, this variation in T1 response selection should logically propagate to T2 (see, e.g., Kunde et al., 2012; Pashler, 1994).

Given the unexpected result for T2, we conducted three additional experiments to (a) replicate the effect and (b) explore possible explanations for it. In Experiment 2, we introduced blocks in which the same colored letter was presented simultaneously in three locations—left, center, and right. This addressed the possibility that presenting the letter only in the center of the screen led to a particularly strong activation of the "middle" response, resulting in shorter RTs in the neutral condition (i.e., when participants uttered the word "middle"). In Experiment 3, we reversed the task order to determine whether the lack of a facilitation effect for T2 was due to the task switch within a dual-task trial or an inherent property of the task. Finally, in Experiment 4, participants performed the vocal task (T2 from Experiment 1) as a single-task, allowing us to examine performance for each response alternative in isolation.

Experiment 2 replicated the results of Experiment 1, even when the letter was presented simultaneously on the right, in the center, and on the left. Experiment 3 showed that the missing facilitation effect for T2 in Experiment 1 was due to a task-specific property rather than the task switch. Experiment 4 further demonstrated that this task-

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<sup>9</sup>For T1 error rates, the facilitation effect was smaller than the interference effect, likely because overall error rates were low and couldn't be smaller than zero (i.e., we likely observed a "floor" effect for the neutral and compatible condition).

specific property may, in part, originate from shorter RTs when uttering the word "middle" compared to "left" or "right." This suggests that a confound between compatibility conditions and response alternatives contributed to the absence of the facilitation effect for T2 observed in Experiment 1. Unfortunately, this confound made it impossible to draw conclusions about the facilitation and interference effects for T2, although it did not affect the conclusions for T1.

#### 4.5 Q3: The Fate of T2 Response Activation

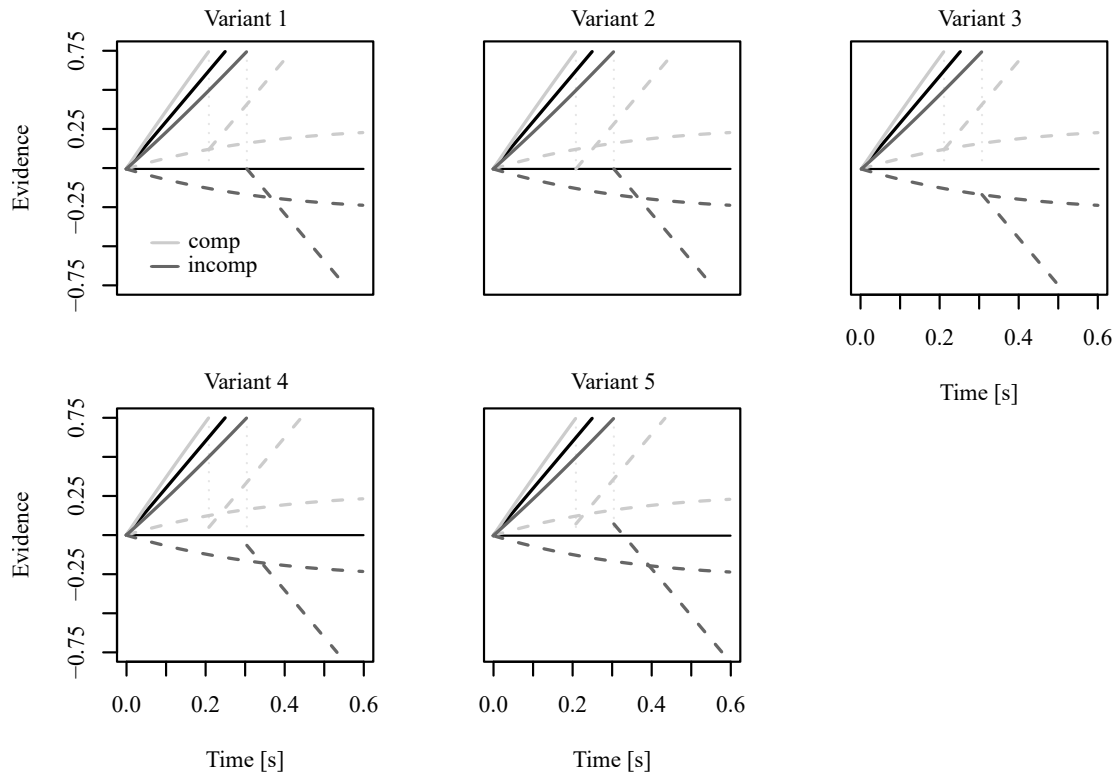
The findings related to Q1 and Q2 suggest that T2 response activation increases almost linearly during T1 response selection and that its influence (and likely its activation) is similar for both compatible and incompatible trials. However, an important theoretical question remains: Does T2 benefit from the previously accumulated response activation? Since T2 becomes relevant after T1 processing, one might expect that any pre-accumulated evidence would carry over and improve actual T2 response selection. Despite the significance of this question, the BCE literature offers limited and inconclusive insights on this issue (cf. Logan & Gordon, 2001; Schubert et al., 2008; Thomson & Watter, 2013). Yet, exploring this topic could potentially clarify why compatibility effects in T2 are consistently larger than the BCEs observed in T1 (e.g., Durst & Janczyk, 2019; Fischer & Hommel, 2012; Fischer et al., 2014; Hommel, 1998; Janczyk, 2016; Logan & Schulkind, 2000). This question was already explored in Study 1, but in a second and independent part of that particular study (which is why I discuss it under Q3).

To investigate the fate of T2 response activation, we explicitly modeled a second decision (i.e., diffusion) process for T2 response selection, initiated after the (overall) T1 decision is complete. We assumed that in compatible trials, T2 response selection strives towards the same decision boundary as the preceding T1 response selection (i.e. both processes have a positive drift rate). In incompatible trials, however, T2 response selection strives toward the opposite decision boundary relative to T1 response selection (i.e., T1 response selection has a positive drift rate and T2 response selection a negative drift rate).

Building on this framework, we examined five theoretically derived mechanisms of T2 response activation transmission (i.e., five model "variants"), inspired by prior studies (see Figure 8 for an illustration of all model variants). First, Thomson and Watter (2013) suggested that T2 response activation could influence T2 response selection, and thus bypass T1 response selection, but only in compatible trials. Thus, in Variant 1, we allowed T2 response selection to start from its previously accumulated activation, but only in compatible trials. In incompatible trials, T2 response selection started from zero, which implies that T2 response activation has no influence in this case. In contrast to Thomson and Watter (2013), Schubert et al. (2008) found no influence of pre-activated T2 response information on T2 response selection, suggesting that it might always be reset to zero (Variant 2; akin to the "response flushing" idea proposed by Logan & Delheimer, 2001). Thus, for Variant 2, T2 response selection never benefited from its previous activation. Inspired by capacity-sharing models (see Chapter 2), a third variant was that T2 activation always carries over into T2 response selection. This Variant 3 followed from the idea that any prior T2 response selection accomplished during T1 processing should reduce the remaining work required for final T2 response selection (Tombu & Jolicœur, 2003). Fourth and fifth, we considered T2 response selection to start with a certain percentage (e.g., 10%), either with respect to its previous response activation during T1 processing (Variant 4), or, alternatively, with respect to the actual T1 decision (i.e., the decision boundary reached by the overall T1 decision process; Variant 5). These latter two variants were inspired by Logan and Gordon (2001)'s ECTVA model, proposing a reset of response counters in dual-task situations to prevent response perseveration.

**Figure 8**

*Schematic Outline of All Five Model Variants Related to the Fate of T2 Response Activation*



*Note.* The general outline follows Figure 5. The newly introduced linear processes (dashed gray lines), starting after T1 processing (solid gray lines), represent T2 response selection. Note that, although we plot T2 response activation as in Figure 5 to illustrate its temporal development, T2 response activation did not drive the actual T2 response. The actual T2 response was based on the process for T2 response selection. Regarding the variants: T2 response selection may benefit from previous activation only in compatible trials (Variant 1), in neither compatible nor incompatible trials (Variant 2), or in both compatible and incompatible trials (Variant 3). The two variants in the second row show mechanisms inspired by ECTVA, where T2 response selection starts with a percentage of either the previously accumulated T2 response activation (Variant 4) or the evidence accumulated for the T1 decision (i.e., relative to the decision boundary reached by the T1 decision process, Variant 5). In the original Study 1 (Part 2), we explored a model with a pulse-like function, as our primary focus at that time was on the qualitative model behavior. Although the displayed function here for T2 response activation is not linear (which might be preferable in light of Study 3), the parameters used in this plot are consistent with those from the simulations in Study 1 (Part 2). Thus, the "pulse-like" function used in our simulation behaved almost like a linear function, with a theoretical peak occurring beyond the x-axis limit. This figure is an adapted version of Figure 3 from Koob et al., 2023.

The subsequent analyses involved fitting all five model variants to both T1 and T2 data, followed by simulations to further explore their behavior. During these simulations,



we also tested how varying the amount of activation transmission (e.g., whether T2 response activation either fully or partially carried over) impacted the model predictions. Overall, most activation transmission mechanisms produced unsatisfactory predictions (see Table 1 for a complete summary): When T2 response activation carried over regardless of a trial's compatibility (Variant 3), the predicted T2 compatibility effect decreased with slower responses (i.e., we observed so-called negatively sloped delta functions; De Jong et al., 1994). This directly contradicted the observed data, which showed increasing T2 compatibility effects with slower responses. Additionally, variants without or with only a small proportion of T2 response activation transmission failed to account for the larger compatibility effect typically observed in T2 compared to T1 (Variants 2 and 4). Furthermore, when T2 activation carried forward only in compatible trials (Variant 1), the model predicted slightly more T2 errors for slow responses in compatible relative to incompatible trials, which was not evident in the observed data.

Ultimately, this led us to conclude that Variant 5 was the most adequate model, followed by Variant 1, although for the latter model one has to be willing to ignore the predictions for T2 accuracy. In Variant 5, T2 response selection begins with a proportion of the evidence accumulated during T1 processing, aligning with the ECTVA model proposed by Logan and Gordon (2001).

**Table 1***Summary of Study 1's Core Results in Part 2*

Variants	Model fits	Simulations		
	Delta Function	Delta Function	CAFs	$\Delta$ Crosstalk
1	yes	yes	no	yes
2	yes	yes	no	no
3	no			
4	yes	yes	no	no
5	yes	yes	yes	yes

*Note.* This table is an adapted version of Table 4 from Koob et al. (2023). In the original study, each variant's behavior was explored by fitting the respective model and through subsequent simulations. "Yes" and "no" indicate whether a variant could or could not qualitatively capture the observed data, respectively. Specifically, we investigated whether a variant could produce increasing T2 compatibility effects with slower T2 responses (columns "Delta Function"), and whether T2 accuracy, conditioned on different levels of T2 response speed, were plausible (column "CAFs," which stands for conditional accuracy functions). For CAFs, we considered a variant plausible if it predicted higher (or at least similar) T2 accuracy in compatible relative to incompatible trials. Additionally, the column " $\Delta$  Crosstalk" indicates whether a larger compatibility effect was predicted for T2 relative to T1. Note that we did not perform model simulations with Variant 3, as this variant already failed when fitting it to the observed data.

## 5 General Discussion

Humans frequently engage in multitasking on a daily basis. Despite our apparent ability to perform multiple tasks in a more or less temporally overlapping manner, decades of research have shown that multitasking is often associated with various costs (e.g., Janczyk & Kunde, 2020; Kiesel et al., 2022; Koch et al., 2018; Pashler, 2000). In addition to task-specific slowdowns caused by reconfiguration requirements or central processing limitations, multitasking also leads to various crosstalk effects that arise as a byproduct when tasks overlap in modality or content.

The present dissertation focused on providing a detailed account of the *response-based backward crosstalk effect*, a specific type of crosstalk arising from a second task influencing the processing of a first task. The goal was to deepen our understanding of the temporal dynamics of T2 response activation during and after T1 response selection. The key to addressing these questions was the combination of modern cognitive modeling approaches

with traditional experimental methods.

To address the overarching question regarding the development and fate of T2 response activation, I examined three subordinate research questions (see Figure 3 in Chapter 4.1): (1) How does T2 response activation develop over time during T1 processing: Is this activation more transient or monotonically increasing? (2) Is T2 response activation symmetric, i.e., is it equally strong in compatible and incompatible trials? (3) What happens to T2 response activation after T1 response selection is complete? Does it contribute to subsequent T2 response selection or not?

I will first briefly summarize the findings and then discuss their broader implications for dual-task research. This will be followed by a discussion of the methodological contributions of this dissertation to cognitive psychology and an outline of important limitations that suggest directions for future research.

## 5.1 Summary of Results

To address whether T2 response activation is more monotonically increasing or transient (cf. Durst & Janczyk, 2019), we explicitly modeled different T2 response activation functions within a DDM inspired by DMC (Ulrich et al., 2015). The results clearly indicate that T2 response activation is almost monotonically increasing for most individuals. In particular, formal comparisons rendered a linear T2 response activation as the most adequate function overall, even outperforming an asymptotic T2 response activation function.

However, an interesting observation was that a minority of individuals (approximately 10-20%) still displayed data more consistent with a pulse-like (i.e., transient) rather than linear T2 response activation. Although further analyses indicated that these individuals did not form a distinct subgroup, the apparent individual differences were unlikely to arise by chance when noisily sampling a finite number of trials from a model with linear T2 response activation (which was clearly more appropriate for the majority of individuals). In other words, there may be a continuum of individuals who process T2 in either a more pulse-like or a more linear fashion. However, a follow-up parameter recovery study showed that while the parameter estimates of the linear function were reliable, those of

the pulse-like function were clearly not. Thus, there may be individual differences in the time course of T2 response activation, but precisely quantifying these differences with a DDM is challenging.

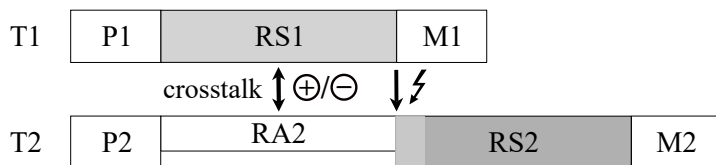
Regarding the symmetry of T2 response activation, we investigated whether T2 facilitates T1 processing in compatible trials to the same extent as it interferes with T1 processing in incompatible trials. In a series of experiments, we introduced neutral trials in which R2 did not overlap with the features distinguishing the R1 alternatives. We found strong evidence that the facilitation and interference effects to the BCE are of equal magnitude, suggesting that T2 response activation is indeed equally strong in compatible and incompatible trials. How this result relates to our modeling work is discussed further below in Chapter 5.4.

The fate of T2 response activation was examined by modeling T2 response selection within a DDM framework, focusing on whether previous T2 response activation affects subsequent T2 response selection. We considered several models in which T2 response selection either starts from previous T2 response activation (Logan & Gordon, 2001; Thomson & Watter, 2013; Tombu & Jolicœur, 2003), always from zero (Schubert, 2008), or with a trace of the previously accumulated T1 response information (Logan & Gordon, 2001). After fitting and simulating each model, our results indicate that only the latter mechanism is consistent with empirical observations. Thus, T2 response activation does not carry over to T2 response selection. Instead, T2 response selection starts with a trace of the evidence accumulated during T1 processing. This then also explains the larger compatibility effect in T2 relative to the BCE in T1.

In Figure 9, I present an updated version of Figure 2B to summarize the results of this dissertation. I argue that this updated version has the advantage of explicitly formulating assumptions with respect to the (R1-R2) BCE. Indeed, looking back at Figure 2B, one might assume that T2 response activation is not continuously present during T1 response selection, that T2 response activation does not influence T2 response selection, or that T1 and T2 response selection are independent. The revised figure aims to convey a more precise picture of the BCE and clearly states its assumptions.

**Figure 9**

*Illustration of a More Precise Conceptual Model for the BCE*



*Note.* This figure is an updated version of Figure 2B, with three major changes: (1) T2 response activation is now shown to be continuously present during T1 response selection, emphasizing that it does not fade. Here, the "box-height" is also slightly smaller, to indicate that T2 response activation is not as efficient in producing a T2 response as subsequent T2 response selection. (2) The use of a plus and minus sign indicates that T2 response activation is comparable in compatible and incompatible trials, equally facilitating and interfering with T1 response selection. (3) The combination of a downward pointing arrow, a lightning bolt, and a brief period of T2 response selection colored identically to T1 response selection illustrates that T2 response selection begins with a trace of the previously selected T1 response.

This Chapter provided a summary of the key findings. The following section discusses how these fit with the existing literature and explores their implications for BCE and dual-task research.

## 5.2 Interpretation of Results and Implications for Dual-Task Research

The monotonic and rather linear increase in T2 response activation suggests that the BCE is phenomenologically different from the congruency effect observed in conflict tasks such as the flanker task. In conflict tasks, activation of the task-irrelevant feature is typically pulse-like (especially for Simon and flanker tasks, see López & Pomi, 2024; Ulrich et al., 2015). The present findings thus contrast with the speculations by Janczyk, Renas, and Durst (2018) and Durst and Janczyk (2019), stating that S2 might behave similarly to flankers in a flanker task due to its "automatic" activation of T2 response information.

Indeed, several authors have argued that shielding T1 from T2 by prioritizing T1 processing, separating task-specific S-R bindings, or inhibiting T2 response activation might benefit performance by avoiding crosstalk, task confusion, or order reversal (Fischer & Plessow, 2015; Fischer et al., 2018; Janczyk, 2016; Lehle & Hübner, 2009; Logan & Gordon, 2001). From this perspective, it is reasonable to assume that participants treat

S2 as a not yet relevant distractor during T1 response selection. The present results do not necessarily contradict this idea, as there may be processes involved that keep T2 response activation in check. However, the results suggest that these processes do not lead to a reduction in T2 response activation for the majority of individuals, as would be expected if participants really treat S2 as a (currently irrelevant) distractor, for instance, like a flanker stimulus.

The persistence of T2 response activation may be explained by a fundamental trade-off between keeping tasks separate to avoid crosstalk and achieving high overall performance on both tasks. Apparently, T2 is always relevant in dual-task scenarios, which means that participants often prepare for both tasks and maintain (at least to some extent) both S-R rules in working memory (De Jong, 1995; Ellenbogen & Meiran, 2008; Meyer & Kieras, 1997; Schubert & Strobach, 2018). Indeed, De Jong (1995, Exp. 3) showed that while participants primarily prepare for T1, they also prepare for T2 to ensure a quick and well-timed task switch. Similarly, Ellenbogen and Meiran (2008) argued that when T1 S-R rules are relatively simple, participants use their remaining working memory capacity to prepare T2 S-R rules, even though this may induce crosstalk. As a result, S2 can continuously prime R2 via its associated S-R rules, which are part of active working memory. Moreover, this continuous activation may neither decay (e.g., Hommel, 1994) nor be actively inhibited (Ridderinkhof, 2002), as this would require T2 S-R rules to vanish from working memory, which is detrimental to performance if these rules are needed shortly thereafter. In essence, it thus appears that participants may be willing to accept some performance decrements due to ongoing crosstalk in exchange for the benefit of a faster task switch once T1 processing is complete.

Of course, the situation changes when T2 becomes less relevant, as is the case in the prioritized-processing paradigm (Miller & Durst, 2014, 2015; Mittelstädt et al., 2023). In this paradigm, participants don't respond to T2 on every trial, but only when T1 requires a no-go response. Because T2 is less relevant, it may be more beneficial to prepare primarily for T1 and less for T2 (see also Durst et al., 2019; Lück et al., 2024; Miller & Tang, 2021). Thus, although T2 response activation may be triggered through previously

formed or memorized S-R rules stored in episodic memory (see also Hommel, 1998, Exp. 5; Janczyk, Renas, & Durst, 2018, Exp. 2; or Logan, 1988), this activation may be more transient because T2 S-R rules are not (additionally) part of active working memory. This would be consistent with the results of Mittelstädt et al. (2023), who explored the role of T2 relevance, demonstrating that T2 response activation is more persistent the more T2 is relevant. In other words, it may be the specific prolongation of T2 response activation following T2 preparation that drives the interference effect in dual-task situations, and which also provides an important distinction from situations in conflict tasks (where the stimulus causing the interference is always task-irrelevant).

Differences in the extent to which participants prepare for T2, or in other words, how much they focus on T1 versus T2, may also contribute to the individual differences observed in Study 3. Indeed, individual differences in multitasking have received more attention in recent years (e.g., Broeker et al., 2022; Brüning & Manzey, 2018; Brüning et al., 2020; Naefgen & Gaschler, 2024; Naefgen et al., 2023), although they have been noted since the beginning of multitasking research (e.g., Jersild, 1927). However, before delving into more detailed analyses of these individual differences, it is important to first determine the extent to which they are reliable. While a few studies have examined individual differences in the size of the BCE (Naefgen & Gaschler, 2024) and their relation to other processing strategies (Brüning et al., 2022), none have examined the reliability of the BCE both within and across sessions. Given the current pessimism regarding the reliability of individual measures from well-established and robust cognitive paradigms (Hedge et al., 2018; Rouder et al., 2023; Schuch et al., 2022), one might expect only moderate reliability for the BCE. Yet, it remains to be seen whether this expectation is justified. Furthermore, one has to keep in mind that the time course of a pulse-like T2 response activation function cannot be estimated with high precision in the context of the BCE (see the parameter recovery properties presented in Study 3).

Another important finding of this dissertation is that T2 response activation likely does not carry over into T2 response selection, matching with conclusions by Schubert et al. (2008), and contrasting with results by Thomson and Watter (2013). Instead, T2

response selection carries a trace of the previous T1 response, as proposed by Logan and Gordon (2001). This fits well with findings of Naefgen et al. (2017, Exp. 3), who showed that free-choice T2 responses are chosen in accordance with force-choice T1 responses, indicating a bias of T2 response selection toward the elicited T1 response.

An interesting aspect arises when we consider the implications for capacity-sharing theories and how we conceptualize the processing of both tasks. If we adhere to the typical "box-like" processing scheme (see, e.g., Figure 2C in Chapter 3), both tasks appear to be processed in relatively separate "pathways" or "channels." In fact, a core assumption of capacity-sharing models is that T1 and T2 response selection can occur in parallel, and that prior T2 central processing reduces the amount of work required to complete T2 response selection once it reaches full efficiency (Lehle & Hübner, 2009; Tombu & Jolicœur, 2003). This requires the cognitive system to track the processing stages of each task in separate channels, because otherwise, it would not be able to determine how much work remains for T2. However, the present results contradict this notion of separate channels, as any T2 response information accumulated during T1 response selection vanishes from the system while T2 response selection somehow carries a trace of the selected T1 response.

To make sense of this, we must abandon the idea of separate processing channels and instead conceptualize task processing in accordance with ECTVA (Logan & Gordon, 2001), in which all stimuli (or their related categories, Ellenbogen & Meiran, 2008; Thomson et al., 2010) map to the same response counters via the instructed S-R rules. In this framework, a clear separation of T1 and T2 processing becomes impossible, since any activation pattern at the response level arises jointly from the stimuli of all tasks and their associated S-R rules. Thus, the reason we generate a response to a given stimulus is not because it has passed through a particular processing channel, but because attention has been directed to that stimulus. Moreover, after selecting a response to T1, it is necessary to reset the response counters to prevent response perseveration. In this sense, ECTVA is more consistent with models that assume that only one task at a time can undergo response selection, and that switching from T1 to T2 involves shifting the "single-channel"



bottleneck to the second task.

Of course, this does not necessarily contradict the concept of "capacity sharing." To reconcile the concept of capacity sharing with ECTVA, one could redefine it in terms of a relative preparation of S-R rules (as was already discussed above). Specifically, instead of assuming that increased capacity sharing leads to more parallel central T1 and T2 response selection within separate channels, one could alternatively suggest that it leads to stronger preparation of the S-R rules of both tasks. Consequently, greater capacity sharing (i.e., S-R rule preparation for both tasks) would lead to a stronger influence of T2 on T1, while yet improving subsequent T2 performance. In other words, attentional resources are not necessarily distributed among processing channels for each task, but rather in terms of an attentional weighting of each task's S-R rules.

### 5.3 Methodological Advances and Remarks

The most significant methodological contribution of this dissertation to the field of (cognitive) psychology is the R package `dRiftDM` (Study 4), which provides easy access to the numerical discretization of the KFE introduced by Richter et al. (2023). The KFE approach is more efficient than the commonly used Monte Carlo simulation approach (e.g., Evans & Servant, 2022; Lee & Sewell, 2024; Luo & Proctor, 2022; Mackenzie & Dudschig, 2021), and should be preferred when deriving the first passage time of DDMS with time-dependent parameters. However, the package cannot replace Monte Carlo simulation in all cases, since the discretization of the KFE requires closed-form solutions for the drift rate, boundary, starting point distribution, and non-decision time distribution. For example, `dRiftDM` cannot be applied to DDMS such as the Dual-Stage Two-Phase Model (Hübner et al., 2010), where the drift rate changes randomly as a function of a second diffusion process. Similarly, the model in Study 1 (Part 2), which includes both T1 and T2 response selection, cannot be modeled within `dRiftDM` because the second diffusion process can only start once the first one reaches a decision boundary.

Although packages similar to `dRiftDM` have been developed for Python (see Murrow & Holmes, 2024; Shinn et al., 2020), `dRiftDM` is accessible to researchers who primarily use

R. In addition, it already includes models commonly used in cognitive psychology, such as the Shrinking Spotlight Model (White et al., 2011) and DMC (Ulrich et al., 2015), and provides functions to directly compute key statistics (e.g., delta functions that describe congruency/compatibility effects as a function of response speed). In this sense, dRiftDM aims to combine the accessibility of packages like *DMCfun* (Mackenzie & Dudschig, 2021) or *fast-dm* (Voss & Voss, 2007) with the flexibility of more general packages like *PyDDM* (Shinn et al., 2020), while being specifically tailored for psychology researchers. Future versions will offer additional methods to efficiently derive the first passage time, such as the integration method advanced by P. L. Smith (2000). This will allow users to choose the most efficient method for a given model. In addition, we will extend the KFE approach to incorporate trial-by-trial variability in the drift rate, providing access to additional models.

A further advancement of this dissertation is a systematic understanding of the advantages and limitations of DDMs in the context of dual-tasking. The results suggest that DDMs can provide valuable insights into the processes underlying overt T1 and T2 performance. However, Study 3 also revealed that while DMC, a common model for conflict tasks, may adequately describe observed BCE data, it suffers from unreliable parameter estimates in this case. Therefore, for dual-task data, we currently recommend using the more classical DDM (Ratcliff, 1978) with a linear T2 response activation function over DMC (Ulrich et al., 2015). Somewhat fortunately, previous dual-task studies have primarily used the classical DDM (see Durst & Janczyk, 2019; Janczyk, Mittelstädt, & Wienrich, 2018; Janczyk et al., 2017; Mattes et al., 2021), and the present dissertation thus retrospectively supports the validity of its application. For future studies, I offer new and more specific recommendations on how to apply DDMs in the dual-task context, using the modern approach implemented in dRiftDM (see the practical suggestions developed in Study 3).

However, researchers should be aware that the classical DDM may not perfectly capture all aspects of dual-task performance. For example, while a linear T2 response activation function works well and outperforms a pulse-like or asymptotic function, it still

slightly overestimates the BCE for slower responses. In addition, it does not fit all participants equally well and may miss more subtle individual differences in the time course of T2 response activation. On a larger scale, one might ignore the slight misfit if it is not too severe, because Study 3 demonstrated that only a minority of individuals were best described by a non-linear function. It is clear that models are always simplifications and cannot capture every aspect of a dataset. In fact, complex cognitive models that account for many data patterns are often prone to overfitting and exhibit poor measurement properties (cf. Lerche & Voss, 2016). Clearly, a model that could describe the activation of the T2 response along a continuum from pulse-like to more linear would be desirable. However, it remains uncertain whether such a model can be parsimoniously formulated with acceptable measurement properties in the context of dual-tasking.

#### 5.4 Open Issues and Future Avenues

Study 2 showed that both facilitation and interference contribute equally to the BCE, suggesting that T2 response activation is similar in compatible and incompatible trials. However, this assumption was not explored within a DDM, and I explicitly chose not to do so. In the present dissertation, a linear T2 response activation function was found to be the most appropriate for the majority of individuals. Yet, this function was found to slightly overpredict the BCE for slow responses. The pulse-like function, in turn, did not overpredict the BCE, but had mediocre parameter recovery properties. Thus, neither T2 response activation function was satisfactory in all respects. Importantly, however, the predicted magnitude of the facilitation and interference effect differs significantly for both activation functions.

In particular, if the activation decays relatively quickly, as modeled by the pulse-like function for standard conflict tasks, a larger facilitation than interference effect emerges (P. Smith & Ulrich, 2024). This is because the short-lived, interfering information is strong in fast, compatible trials, while it has already decayed to a large extent in relatively slow, incompatible trials. However, when the activation decays very late (or not at all), as it is the case for most individuals in BCE experiments, comparable facilitation and interference

effects are predicted (see also Figure 2 in Koob et al., 2024). Yet again, when the activation follows a linear time course, interference effects tend to be larger than facilitation effects. Thus, given the uncertainty about the true T2 response activation function and given that the conclusions with respect to facilitation and interference sharply depend on this function, no respective modeling exercise was pursued. Nevertheless, future work should of course try to address this gap in order to complement the experimental approach of Study 2 (see also Evans & Servant, 2022, for a discussion in this regard). In doing so, however, it is critical to ensure that the modeled neutral condition does not involve any confounds that would otherwise make it not "truly neutral" (see, e.g., T. L. Brown, 2011; Jonides & Mack, 1984). In addition, any modeling approach that allows the strength of T2 response activation to vary across compatibility conditions must be based on carefully considered theoretical assumptions.

Another important issue for future research is to empirically test the proposed transition mechanism, where T2 response activation is reset and T2 response selection carries a trace of the previous T1 response (see Figure 9, and Logan & Gordon, 2001's ECTVA model). Although such a mechanism produced the most plausible model predictions in Study 1 (Part 2), this does not necessarily confirm it as true. However, the model does offer specific, testable predictions. For example, as long as the amount of evidence accumulated for T1 remains constant (i.e., as long as the decision boundary does not vary), the bias in T2 response selection should remain relatively stable. Thus, the difference in the compatibility effect between T2 and T1 should remain fairly constant as the SOA varies between S1 and S2.

Some support for this prediction can be found in the summary statistics reported by Janczyk (2016, Exp. 1), where the compatibility effect in T2 was consistently about 30 ms larger than the BCE in T1 across SOAs of 50 ms, 150 ms, and 650 ms (see also the 40 ms and 300 ms SOA condition in Röttger et al., 2021). In contrast to this finding, however, Hommel (1998) reported that both the R1-R2 BCE and the corresponding compatibility effect in T2 disappeared at an SOA of 650 ms. Thus, additional well-powered and carefully designed experiments are needed to test the assumption that T2 response selection carries

a trace of the previously selected T1 response. In the same vein, one might try to include a neutral condition for T2 that is not confounded with R2 speed (as it was the case in Study 2). With such a more ideal neutral condition, one can test for the absence of a bias in T2 response selection when the previous T1 response does not overlap with the subsequent T2 response.

Furthermore, it is important to examine how other BCEs, like the S1-R2 BCE, fit into this framework. For the S1-R2 BCE, interference occurs between T2 response activation and T1 stimulus classification (Koob et al., 2020). Interestingly, even in this case, larger compatibility effects have been observed for T2 relative to T1 (e.g., Hommel, 1998; Koob et al., 2020), although R1 and R2 do not conceptually overlap in S1-R2 BCE experiments. Thus, at first sight, it seems difficult to reconcile the larger T2 compatibility effect in S1-R2 BCEs with the proposed reset mechanism of ECTVA.

A final avenue of research could be to explore the similarities and differences between the BCE and the congruency effect in task switching (Meiran, 1996; Rogers & Monsell, 1995). In task switching, trials can be "bivalent," meaning that a stimulus can be evaluated under both the currently relevant and the currently irrelevant task set. For example, digits can be classified as greater or less than five in one task and as odd or even in another. If a digit maps to the same response in both tasks, the trial is considered congruent; otherwise, it is incongruent. As with the BCE, performance on incongruent trials is impaired relative to congruent trials. A common explanation for the congruency effect in task switching is that the S-R rules of the currently irrelevant task are not completely deactivated, allowing them to "linger" in an active state even when not needed (see also Allport et al., 1994). This bears a striking resemblance to the BCE, and several studies have shown parallel findings for both phenomena, such as their susceptibility to the previous trial history (e.g., J. W. Brown et al., 2007; Janczyk, 2016) or the proportion of congruent and incongruent trials (e.g., Bugg & Braver, 2016; Fischer et al., 2014).

Surprisingly, a comprehensive analysis that combines both effects within a common framework is still lacking. Any such attempt, however, must take into account a key difference: In a BCE experiment, both T1 and T2 require a response on every trial,

whereas in task switching only one task per trial is relevant. Consequently, the degree to which a task is perceived as (not yet) relevant may differ between the two paradigms, potentially altering the time course of the interfering response information.

## 5.5 Conclusion

The primary question of this dissertation was: What is the time course of T2 response activation during and after T1 response selection? The answer is twofold. First, T2 response activation is nearly linear during T1 response selection for most individuals, resulting in continuous and symmetrical interference in both compatible and incompatible conditions. However, this activation presumably does not carry over to T2 response selection. Instead, T2 response selection begins with a trace of the total response information accumulated during the T1 decision, that is, T2 response selection is biased by the previously selected response. This contradicts the core assumptions of capacity-sharing models and suggests that the two tasks do not operate in separate channels. Rather, both T1 and T2 rely on the same response codes and response selection acts serially on a shared architecture. In addition, the results suggest that while a linear time course of T2 response activation provides a good approximation, it slightly overestimates the BCE for slow responses and may not capture more subtle individual differences in the time course of T2 response activation.

## 6 References

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## 7 Appendices

### Appendix A: Study 1

Official citation: Koob, V., Ulrich, R., & Janczyk, M. (2023). Response activation and activation-transmission in response-based backward crosstalk: Analyses and simulations with an extended diffusion model. *Psychological Review*, *130*(1), 102–136. <https://doi.org/10.1037/rev0000326>.

Abstract:

In dual-task experiments, overlapping response characteristics of two subsequently performed tasks may not only affect performance in Task 2, but also in Task 1. This phenomenon is often explained through activated Task 2 response information influencing Task 1 response selection, which then possibly propagates again into Task 2. So far, however, only little is known about (a) the time-course of this Task 2 response activation and (b) possible transmission/propagation mechanisms. The present study addressed both issues by testing ten plausible drift-diffusion models with five datasets from dual-task experiments. To this end, we first examined if the temporal course of the response activation is linearly increasing or pulse-like. The pulse-like model turned out to be superior, but the corresponding dynamics of the response activation often described a monotonically increasing function that reached its peak late during Task 1 processing. By extending the pulse-like model with an additional diffusion process, we then examined whether and how the Task 2 response information could affect subsequent Task 2 response selection. Concerning the transmission mechanisms, none of the assumed models proved to be entirely satisfactory. However, additional simulations suggest that Task 2 response activation-transmission does not occur at all. Instead, a model in which Task 2 started with a trace of the previous Task 1 response (i.e., irrespective of the pre-existing Task 2 activation) turned out to be the most promising account.

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**Appendix B: Study 2**

Official citation: Koob, V., Sauerbier, C., Schröter, H., Ulrich, R., & Janczyk, M. (2024).

Separating facilitation and interference in backward crosstalk. *Journal of Experimental Psychology: Human Perception and Performance*, 50(3), 295–312. <https://doi.org/10.1037/xhp0001184>

Abstract:

When two speeded tasks have spatially overlapping responses, pre-activated Task 2 (T2) response information influences Task 1 (T1) response selection, a phenomenon known as the backward crosstalk effect (BCE). Current models of the BCE implicitly assume that T2 response information is equally present in trials requiring compatible or incompatible responses, such that T1 performance both improves when T2 requires a compatible response and deteriorates when T2 requires an incompatible response. Thus, T2 response information should have a facilitatory and an interfering effect on T1. Interestingly, this hypothesis has never been tested, and the present study (conducted between 2021 and 2023) attempts to fill this gap by using neutral trials in which T2 responses did not spatially overlap with those in T1. The results suggest that the BCE (in T1) reflects both facilitation and interference effects of comparable magnitude, thus corroborating current conceptualizations of the BCE. We also observed an unexpected pattern of effects for T2, with only an interference effect, but no facilitation effect. Additional experiments led us to conclude that the T2 result was sensitive to the specific task characteristics. Conclusions about how the crosstalk transfers from T1 to T2 when switching tasks are therefore not possible.

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### **Appendix C: Study 3**

Citation: Koob, V., Ulrich, R., Ahrens, A., & Janczyk, M. (under revision). The time course of Task 2 response activation in dual-tasking: Modeling results, interindividual differences, and practical recommendations.

Abstract:

In dual-task experiments, overlapping response characteristics of two successive tasks affect performance not only in Task 2 (T2) but also in Task 1 (T1). This observation is termed the backward crosstalk effect (BCE) and is often explained by activated Task 2 response information influencing Task 1 response selection. In the present study, we describe and evaluate three diffusion models that specify T2 response activation as linear, asymptotic, or pulse-like. Formal model comparisons and a parameter recovery suggest that linear T2 response activation is most accurate for most individuals. However, we also highlight the potential role of individual differences, as a pulse-like T2 response activation function may better describe a subset of individuals. Finally, we provide practical recommendations for researchers on how to fit the BCE in the larger context of dual-tasking.

**Appendix D: Study 4**

Citation: Koob, V., Richter, T., Ulrich, R., & Janczyk, M. (in preparation). An introduction and tutorial to fitting (time-dependent) diffusion models with the R-Package dRiftDM. *Pre-Print at <https://osf.io/preprints/osf/3t2vf>*

Abstract:

Using mathematical models of human cognition has become an increasingly important and valuable tool in many psychological research fields and neighboring areas. Widely used are drift-diffusion models (DDMs) that can be used to predict probability density functions (PDFs) of binary choice reaction tasks. Often, the parameters of such a model are time-independent (i.e., they do not vary as a function of time within a trial). However, the more general case is that of time-dependent parameters. Several recent models, for example, assume time-dependency for the drift rate and/or the boundaries. Such time-dependent (or non-stationary) models increase mathematical complexity, but several solutions to approximate the PDFs have been advanced. We here present dRiftDM, an R package particularly designed to meet the needs of psychological research to estimate time-dependent models. Importantly, with dRiftDM, users can provide custom drift rates, boundaries, starting points and non-decision times, unlocking a wide range of DDMs. Fitting a model to data can be done participant-wise, and extracting model parameters or calculating summary statistics is straightforward. Hands-on examples for using pre-built and for developing own models are provided.