RUNNING HEAD: FAILURES TO REPLICATE BLOCKING ARE SURPRISING

Failures to replicate blocking are surprising and informative – Reply to Soto (in press)

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Abstract

The blocking effect has inspired numerous associative learning theories and is widely cited in the literature. We recently reported a series of 15 experiments that failed to obtain a blocking effect in rodents. Based on those consistent failures, we claimed that there is a lack of insight into the boundary conditions for blocking. In his commentary, Soto (in press) argues that *contemporary associative learning theory* does provide a specific boundary condition for the occurrence of blocking, namely the use of same- versus different-modality stimuli. Given that in ten of our 15 experiments same-modality stimuli were used, he claims that our failure to observe a blocking effect is unsurprising. We cannot but disagree with that claim, because of theoretical, empirical, and statistical problems with his analysis. We also address two other possible reasons for a lack of blocking that are referred to in Soto's (in press) analysis, related to generalization and salience, and dissect the potential importance of both. While Soto's (in press) analyses raises a number of interesting points, we see more merit in an empirically guided analysis and call for empirical testing of boundary conditions on blocking.

Keywords: blocking, replicability, associative learning theory, moderators

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Blocking refers to the observation that conditioned responding to X after AX+ trials is reduced if AX+ trials are preceded by A+ trials. Since its discovery by Kamin (1969), blocking has inspired many associative learning theories. The effect is widely referred to in the literature, ranging from papers on basic learning research (e.g., Feldman, 1975) to papers on neuroscience (e.g., Corlett et al., 2004; Steinberg et al., 2013) and psychopathology (e.g., Boddez et al., 2012; Moran, Al-Uzri, Watson, Reveley, 2003). Moreover, blocking has been documented in numerous species, including mollusks (e.g., Sahley, Rudy, & Gelperin, 1981), rats (e.g., Kamin, 1969), and humans (e.g., Dickinson, Shanks, & Evenden, 1984). We recently reported a series of 15 experiments in which we tried but failed to obtain a blocking effect in rodents (Maes et al., 2016). Although we did not dispute the fact that blocking is a genuine effect, we did argue that the effect is more difficult to obtain than what would be expected based on the published literature. Hence, we concluded that more empirical research is needed to reveal the moderators of this important effect. Notwithstanding that others before us had also hinted at the unreliability of blocking (e.g., Batson & Batsell, 2000; Beesley & Shanks, 2012; Blaser, Couvillon, & Bitterman, 2006; Guerrieri, Lachnit, Gerber, & Giurfa, 2005; LoLordo, Jacobs, & Foree, 1982; Taylor, Joseph, Balsam, & Bitterman, 2008; Vadillo & Matute, 2010; Yamada, 2010), our article spurred discussions on the status of the blocking effect (Skibba, 2016; Soto, in press; Urcelay, in press).

In his commentary, Soto (in press) argues that our failures to observe a blocking effect are rather unsurprising. First, at the theoretical level, he claims that most of them could be predicted on the basis of what he refers to as *contemporary associative learning theory*. Second, at the methodological level, he argues that the remaining failures could simply reflect a lack of statistical power. In this paper, we respond to both arguments put forward by Soto, starting with the theoretical arguments. Based on Soto's (in press) analysis, we also discuss some considerations that might be taken into account when designing blocking experiments.

Contemporary associative learning theory does not predict our failures to find blocking with same-modality stimuli

Briefly, Soto's (in press) theoretical argument relies on two main assumptions: (1) the more the AX compound is processed in a configural manner, the weaker blocking will be, (2) the more similar A and X are, the more readily they will be processed configurally when presented in compound. On that basis, Soto states that "contemporary associative learning theory predicts that more similar stimuli, and particularly those coming from the same modality, should produce a weaker blocking effect" (Soto, in press, p. 5-6). Given that in ten of our 15 experiments, stimuli from the same modality were used, Soto argues that "in the light of contemporary associative learning theory, most of the failures to obtain blocking obtained in this paper are unsurprising" (Soto, in press, p. 8). However, we cannot but disagree with the theoretical argumentation provided by Soto (in press), for four reasons that we will summarize here before addressing them in more detail below: 1) It is not warranted to refer to contemporary associative learning theory as a unitary entity. 2) Even when focusing on a subset of models, it would be inaccurate to state that those models *predict* in an a priori manner our failures to observe blocking. 3) Focusing on the two main assumptions supporting the theoretical argument of Soto (in press), namely that stimuli from the same modality will be processed more configurally and that more configural processing leads to a weaker blocking effect, empirical evidence forces us to question the assumption that same-modality stimuli are necessarily processed configurally. 4) Finally, the relationship between configural processing and blocking as put forward by the three models discussed by Soto (in press) is also to be questioned, based on the data from our studies (Maes et al., 2016) and other data in the literature.

Ad 1. To begin, Soto's (in press) argument implies that *contemporary associative learning theory* can be thought of as a unitary entity. This idea contrasts with the fact that there is an enormous variety of associative learning theories, a variety that is so large that it is impossible to make general claims about contemporary associative learning theory as a class (Miller & Escobar, 2001). For every prediction of a particular associative theory, there is another theory or the same theory with different parameters that would not make that prediction or that would even make the opposite prediction. Consider the assumption put forward by Soto (in press) that within-compound

associations develop more easily between more similar stimuli, and that this should reduce blocking. One might argue that the development of a within-compound association between A and X would indeed reduce the expression of blocking, because at test X will activate the representation of A which in turn will activate a representation of the outcome, yielding strong conditioned responding despite a weak direct association between X and the outcome. However, in spite of this intuition, prominent formal theories of learning, like the comparator hypothesis (e.g., Miller & Matzel, 1988), actually make the opposite prediction that stronger within-compound associations would lead to stronger blocking. This is because a stronger A-X association is expected to produce a stronger downmodulation of responding to X by the indirect X-A and A-outcome links.

Even within the subset of models discussed by Soto (in press), important differences exist. Although those models all predict reduced blocking for same-modality A and X stimuli, the reduction in blocking occurs for different reasons: Two of those models, the Replaced Elements Model (Wagner, 2003) and the Extended Configural Model (Kinder & Lachnit, 2003), assume weak conditioned responding to X in both the experimental (A+, AX+) and control group (B+, AX+), whereas the Latent Causes model (Soto, Gershman, & Niv, 2014) assumes strong conditioned responding to X in both groups. Hence, it is simply incorrect to make general claims about how contemporary associative learning theory sees the relation between blocking and stimulus modality.

Ad 2. It is one thing to say that a subset of contemporary associative learning theories could accommodate our results, but quite another to say that (a subset of) contemporary associative learning theories would have *predicted* the ten failures to obtain blocking with same-modality stimuli, as Soto (in press) argues. Given that the literature is replete with observations of blocking involving same-modality stimuli (e.g., Beckers, Miller, De Houwer, & Urushihara, 2006; Blaisdell et al., 1999; Wheeler, Beckers, & Miller, 2008), models that predict a lack of blocking with same-modality cues would in fact be incompatible with the literature. Soto's argument, of course, is that the likelihood of observing a blocking effect is reduced and not completely eliminated when same-modality stimuli are used. The subset of learning models he discusses incorporate this flexibility by

assuming that same-modality stimuli lead to *more* configural processing. The Replaced Elements Model (Wagner, 2003) and the Extended Configural Model (Kinder & Lachnit, 2003) predict an absence of blocking only when making the additional assumption that same-modality stimuli lead to extreme configural processing and a complete absence of generalization from A to AX, and from AX to X. Likewise, the Latent Causes model (Soto et al., 2014) predicts the absence of a blocking effect only if it is assumed that extreme configural processing leads to complete generalization between A, AX and X. However, given that there is no a-priori rule for deciding whether or when same-modality stimuli induce extreme configural processing or merely induce moderate configural processing, the models are not able to *predict* in an a priori manner whether in a specific blocking experiment using same-modality stimuli the effect will or will not occur.

Ad 3. Having established that contemporary associative learning theories or a subset thereof do not predict our consistent failure to obtain blocking, we now turn to examining whether Soto's (in press) analysis at least provides a plausible post-hoc explanation. We do not think so, because there are empirical reasons to doubt that there is a consistent relation between stimulus modality and configural processing in general. Soto's analysis is based on the idea that "more similar stimuli, such as those coming from the same modality, produce more configural processing" (p. 5). As support for this idea, Soto refers to an experiment conducted in rabbits showing that summation is observed with different-modality stimuli but not with same-modality stimuli (Kehoe, Horne, Horne, & Macrae, 1994). In summation experiments, the response to a compound of two stimuli that were both previously paired with the US is measured. A lack of summation is indicative of a generalization decrement from the elements to the compound, which some models (but not all; Kehoe et al., 1994) indeed explain by invoking configural processing (e.g., the Replaced Elements Model of Wagner, 2003, the Extended Configural Model of Kinder & Lachnit, 2003, and the Latent Causes model of Soto et al., 2014). From that perspective, the experiment of Kehoe and colleagues (1994) does provide support for the idea that stimuli from the same modality produce more configural processing. However, when taking into account other studies investigating summation with same-modality

stimuli (Aydin & Pearce, 1995, 1997; Kehoe et al., 1994; Redhead & Pearce, 1995; Rescorla & Coldwell, 1995; Thein, Westbrook, & Harris, 2008) the picture becomes far less clear. First, summation has been observed, at least to some extent, in experiments with pigeons and rats using same-modality stimuli (Aydin & Pearce, 1995, 1997; Thein et al., 2008). Moreover, factors that influence whether summation with same-modality stimuli is observed have been described. Aydin and Pearce (1995) observed summation with two visual stimuli when stimulus duration was long (30 s), but not when stimulus duration was short (10 s). Another factor that influenced whether summation with visual stimuli was observed, was the illumination of the background on which the stimuli were displayed (Aydin & Pearce, 1997). According to Aydin and Pearce (1997), this last factor might also explain some of the other failures to observe summation with visual stimuli in pigeons (Aydin & Pearce, 1995; Rescorla & Coldwell, 1995). Thus, studies aimed at investigating when summation takes place with same-modality stimuli point out that stimulus duration and illumination play an important role. Lastly, in some experiments a failure to observe summation was observed even when stimuli from different modalities were used (Pearce, George, & Aydin, 2002). Taken together, the available evidence indicates that stimulus modality might not be the most important factor to determine whether summation, and by extension configural processing, takes place (an idea also put forward by Couvillon, Arakaki and Bitterman, 1997). In conclusion, rather than accepting Soto's (in press) current argument, we agree with Soto's earlier statement that "similarity between elements is not truly a unifying principle that explains the effect of stimulus factors in compound generalization" (Soto et al., 2014, p. 528).

Ad 4. The overview presented above indicates that, based on empirical data, the argument that stimuli from the same modality produce more configural processing is not supported by the literature. However, putting aside the modality of the stimuli, it is still possible that other (unknown) factors resulted in our stimuli being processed *more* configurally. Even then, our failures to find blocking would still be surprising, but at least the models discussed by Soto (in press) would be able to account for our results. However, neither the assumptions nor the predictions of two of the three

models discussed by Soto (in press) are in agreement with the behavior observed in our studies (Maes et al., 2016). According to the Replaced Elements Model (Wagner, 2003) and the Extended Configural Model (Kinder & Lachnit, 2003), more configuring would lead to less generalization from A to AX and from AX to X. However, there is little indication that generalization from A to AX or from AX to X in our experiments was limited. In seven of our experiments (Experiments 4 and 10 to 15), conditioned responding was measured during Pavlovian training sessions (i.e., response data were collected not only at test but also during training). Those data are available online on the Open Science Framework (https://osf.io/fcwnr/). Of those experiments, Experiments 10 to 14 employed same-modality stimuli. From Table 1, it is clear that only two out of those five experiments (Experiments 11 and 12) revealed a (non-significant) decrease in responding from A to AX in the experimental group. The only significant decrease was observed for Experiment 15, in which different-modality stimuli were used. Further, Appendix N of our original paper (Maes et al., 2016) shows that there was no difference in responding between the last A or B trial and the first X trial. In sum, there is no empirical indication for limited generalization between the stimuli, at least not in the experiments that employed same-modality stimuli. This observation questions the validity of the idea that the absence of blocking in these studies was due to the use of same-modality stimuli inducing pronounced generalization decrement from A to AX trials or from AX to X trials.

(Table 1 about here)

Clearly, the assumptions of the Replaced Elements Model and the Extended Configural Model are not in agreement with the behavior observed in our studies (Maes et al., 2016), nor are their predictions. The simulations of the Replaced Elements Model and the Extended Configural Model provided by Soto (Figure 1 in Soto, in press) indicate that the models predict weak responding to X (low associative strength) in both the experimental and control group. Although there is no gold standard as to what qualifies as “strong” or “weak” responding (or to determine how associative strength is mapped to conditioned responding), responding in almost all our experiments can be considered as strong (except in Experiments 2, 3, and 15, in which the absence of a blocking effect

can be attributed to a floor effect, as we acknowledged in our original paper). Thus, those two models cannot account for the actual behavior observed in most of our experiments. Moreover, in his earlier work, Soto correctly pointed out that those same two models have problems accounting for other variables that have been demonstrated to influence blocking (Soto et al., 2014).

The discussion above strongly suggests that there is limited merit in models that rely on restricted generalization between similar stimuli (such as the replaced elements model of Wagner, 2003, and the extended configural model of Kinder and Lachnit, 2003) for explaining our failures to observe a blocking effect. However, Soto (in press) also provides a simulation of a third model: the Latent Causes model proposed by Soto and colleagues (2014). The latter model assumes that similar stimuli (A, X, AX) activate the same latent cause. As a consequence, responding to X is predicted to be strong in both the experimental and control condition (see Figure 1, Soto, in press). As said, the responses to X in most of our experiments (and all of our experiments with same-modality stimuli) can be considered to be strong and are thus in correspondence with the predictions of the model. However, whereas Soto et al. (2014) simply assumed that highly similar stimuli, such as same-modality stimuli, would lead to configural processing, it is necessary to assume extreme configural processing in order to predict the absence of a blocking effect. To the best of our knowledge, this was not an a-priori assumption of the Soto et al. (2014) model. Therefore, and in contrast with Soto's claim (Soto, in press, p. 6), using the simulations from Soto et al. (2014) to account for part of our results should be considered as post-hoc explaining. Further, by assuming that similar stimuli activate the same latent cause, it is unclear to us how this model would explain (1) the numerous observations of blocking using same-modality stimuli (see above) and (2) the observation of overshadowing with stimuli from the same modality (e.g. Jones & Haselgrove, 2011; Urcelay & Miller, 2009; overshadowing is the observation that a stimulus C elicits less conditioned responding after being trained in compound with another stimulus D than after receiving an equivalent amount of elemental training). If CD and C both elicit the same latent cause, no generalization decrement, and hence no overshadowing, is to be expected.

In conclusion, the statement that "the prediction from the literature is clear: such stimuli [stimuli from the same modality] foster configural processing and reduce the likelihood of observing blocking" (Soto, in press, p. 7), is only defendable based on a selective reading of the literature.

Our failures (Maes et al., 2016) to observe blocking are statistically surprising

In addition to the theoretical arguments that we refuted in the preceding section, Soto (in press) put forward statistical arguments for why our results were unsurprising, which we disagree with as well. First, Soto argues that for ten out of 15 experiments "the effect might be too small to detect with the relatively small sample sizes used by Maes et al. (2016)" (Soto, in press, p. 7). This argument disregards the power analyses that we provided (Maes et al., 2016, Appendix P). For each experiment (including each of the experiments employing same-modality stimuli), there are counterparts in the literature that matched the procedure and stimuli of our experiments closely (for comparisons see Appendices A to E of Maes et al., 2016). Based on the effect sizes observed in those experiments, we have calculated the power to observe an effect with the sample size used in our studies (Maes et al., 2016, Appendix P). For all the experiments that used same-modality stimuli, the estimated power was at least .70, sometimes much higher. A power of .70 per experiment (which is a conservative estimate, given that power was much higher in four of those experiments) renders the a-priori chance of not observing the blocking effect in even a single of those ten experiments smaller than 0.01%. We would regard an event with an a-priori probability of less than 0.01% of occurring as surprising.

A similar statistical argument can be brought forward when considering the five failures to observe blocking in the experiments that did use stimuli from different modalities. Three of those five failures can be attributed to a floor effect (as we pointed out on p. e57 of the original paper). For the remaining two experiments (Experiments 1 and 4 in Maes et al., 2016), Soto (in press) argues that "statistically we expect some proportion of well-conducted blocking experiments to not produce a blocking effect" (p. 9). When considering the power calculations, which were based on effect sizes of

similar previously published experiments, the a-priori chances of not observing the blocking effect in just those two experiments alone is again smaller than 0.01% (power for each experiment was > .99). We also conducted Bayesian analyses that allow to quantify the strength of the relative statistical evidence for two rivaling hypotheses by calculating the Bayes Factor (BF). Given that it is about relative evidence, a BF of about 1 does not bear any evidence in favor of either one of the hypotheses (e.g., Dienes, 2011; Gallistel, 2009; Rouder, Speckman, Sun, Morey, & Iverson, 2009). As reported in Maes et al. (2016, Appendix H), neither Experiment 1 nor Experiment 4 provided conclusive evidence in itself. However, when considering those two experiments in combination, the BF provides evidence in favor of the null hypothesis (BF_{01} = 3.19). Thus, those two failures to observe blocking are surprising and provide evidence against the robustness of the blocking effect also when using stimuli from different modalities.

Other considerations brought forward by Soto's (in press) analysis

Apart from arguments based on (complex) theories, Soto's (in press) analysis yields two more straightforward explanations for our failures to observe blocking, related to generalization and salience.

Generalization and modality. Although Soto's (in press) analysis mainly focuses on the impact of stimulus modality on configural versus elemental processing (i.e., same-modality stimuli are processed more configurally and increases in configural processing reduce blocking), he also points out that stimulus modality could influence blocking through an impact on generalization. The idea is that strong generalization should produce a smaller blocking effect for two reasons. First, if generalization between A and X is strong a smaller blocking effect might be observed, because generalization from A would result in high conditioned responding to X. Second, Soto (in press) argues that generalization from B to A in the control group might make it even harder to detect a blocking effect (p. 7-8). However, A and B are of the same modality in most if not all blocking experiments, so this latter source of generalization is not specific to our "failed" experiments

involving same-modality A and X stimuli but would affect the chance of observing blocking in mostly any study. Regarding the former type of generalization, from A to X, assuming that generalization from A to X does indeed hamper blocking, Soto's (in press) argument is that using same-modality A and X stimuli enhances generalization from A to X and therefore reduces the chances of finding blocking. We do not agree, however, that this argument threatens the validity of our conclusions. However intuitively appealing the idea may be that, on average, generalization is stronger between stimuli from the same modality, this need not imply that the chances of finding blocking in our experiments were suboptimal. First, it seems reasonable to assume that even stimuli from the same modality can be different enough to prevent strong generalization. In particular, in experiments in rats, generalization from an auditory stimulus C (clicker) to an auditory stimulus T (tone) has been shown to be restricted when the stimuli differ in terms of novelty (as is the case in a blocking experiment) (Honey, 1990; Robinson, Whitt, & Jones, 2017). Moreover, as stated, blocking has been reported repeatedly employing same-modality stimuli (e.g., Beckers, et al., 2006; Blaisdell et al., 1999; Wheeler et al., 2008), indicating that generalization was not an issue in those experiments. Summarized, generalization is *not necessarily* a problem with same-modality stimuli. Furthermore, using different-modality stimuli does not necessarily reduce the potential for generalization. Experiments in rats have suggested that similarity in stimulus duration (Meck & Church, 1982) or intensity (Delay, 1986) can support cross-modal generalization. Hence, using different-modality A and X stimuli in blocking studies is not necessarily more optimal than using same-modality A and X stimuli to prevent generalization from A to X.

To the best of our knowledge, there are also no studies available that have empirically investigated the influence of stimulus modality on blocking in rodents. There is some work on this in bees, but that work suggests that modality (and in extend similarity) is *not* an important modulator (Couvillon, Arakaki, & Bitterman, 1997; Funayama, Couvillon, & Bitterman, 1995; Guerrieri, Lachnit, Gerber, & Giurfa, 2005). What holds for bees need not be true for rats, of course, but it does indicate that stimulus modality and similarity do not *need to* modulate blocking.

Thus, there are no logical or empirical reasons to conclude that the use of same-modality stimuli should reduce the chances of finding blocking by increasing generalization. Therefore, the only meaningful way to determine whether generalization could have hampered blocking in a particular study is to directly assess generalization in that study. It is, however, not straightforward to deduce the amount of generalization between stimuli based on the data obtained in a blocking study alone. A relative lack of difference in responding to A and X might be due to generalization, the formation of within-compound associations or simply strong behavioural control directly gained by X during AX+ training. Likewise, a lack of difference in responding to B and X can be due to generalization from A, AX, and B as well as strong behavioural control gained by X during AX+ training. All of those factors will result in more comparable (or higher) levels of responding to A/B and X and hence, an overestimating of the amount of generalization from A to X. A final approach is to compare responding to B and AX (note that in all blocking experiments A and B are from the same modality). A decrease in conditioned responding from B to AX was observed in five out of the seven control groups (Table 1), suggesting that generalization was limited in those groups. Moreover, comparable levels of responding to B and AX in the other two groups might be the result of generalization based on modality, but equally likely due to generalization based on duration or another factor. Hence, the empirical evidence for the claim that generalization might have been a problem in our studies is weak and it is even less obvious that it could be attributed to the use of same-modality stimuli. All of these theoretical and empirical considerations lead us to conclude that one cannot simply dismiss our failures to find blocking on the basis of the fact that we used A and X stimuli from the same modality in the majority of our experiments. They do reinforce our general conclusion that more empirical research is needed on the moderators of blocking, including the potential moderating role of stimulus modality.

Salience. On p. 9, Soto (in press) argues that relative salience is an important modulator of the blocking effect. Indeed, several studies (Kamin, 1969; Feldman, 1975; Hall et al., 1977) have indicated that blocking is more easily observed when the blocking stimulus, A, is more salient than

the to-be-blocked stimulus, X (as arguably was the case in our Experiments 2, 3 and 15). At the same time, however, as Soto points out, when A is more salient than X, A might also overshadow X in both the control and the experimental group, resulting in low responding to X in both groups and hence, little or no blocking (as observed in our Experiments 2, 3 and 15). So, it seems that A being more salient than X might at once strengthen or attenuate the blocking effect. Further, it is argued by Soto (in press) that when A is less salient than X, X might serve as an external inhibitor such that generalization from A to AX is limited and as a consequence, blocking will be attenuated. In conclusion, while we agree that salience is an empirically supported moderator of blocking, the exact nature of that moderation and the optimal balance of salience between A and X that will serve to best obtain a blocking effect is not well understood. There are reports of the blocking effect that involve stimuli from different modalities that may have differed in salience. In some of those reports, a visual stimulus was shown to block an auditory stimulus (e.g., D. Jones & Gonzalez-Lima, 2001; Mackintosh, Dickinson, & Cotton, 1980; Sanderson, Jones, & Austen, 2016), whereas in others it was the other way around (e.g., Holland, 1999; Sanderson, Jones, & Austen, 2016; Taylor, Joseph, Balsam, & Bitterman, 2008). As such, the mere fact that stimuli from different modalities are used that may differ in salience cannot be considered an impediment for obtaining blocking. As a *post-hoc* explanation for the failure to obtain blocking, we agree that relative salience might a candidate cause for some of our experiments.

Conclusions

In sum, we dispute that "the results described by Maes et al. are rather unsurprising, and for the most part can be explained in the light of contemporary associative learning theory" (Soto, in press, p. 4), based on theoretical (the proposed models cannot predict when the use of same-modality stimuli will lead to extreme configural processing, which is a necessary assumption for those models to explain the absence of a blocking effect), empirical (existing data do not support Soto's theoretical assumptions) and statistical (from power calculations based on experiments using similar stimuli and procedures, the a priori chances for not observing a blocking effect were very

small) grounds. That being said, we are happy to see the analysis proposed by Soto (in press) added to the literature, as it highlights the potential importance of generalization and relative salience of the blocked and to-be-blocked stimulus for blocking. Also, Soto's (in press) analysis, while unsatisfactory at present, provides a pointer for the investigation of other potential boundary conditions such as similarity of A and X in stimulus modality. That being said, until empirical research has directly evaluated the importance of potential moderators for the blocking effect, we cannot simply assume that we have good insight into what they are. Empirical research has time and again indicated that many reasonable assumptions brought forward by associative learning models could not be supported by empirical data (e.g., Harris, 2006; Miller, Barnet & Grahame, 1995; Soto et al., 2014). Therefore, we strongly encourage further empirical research aimed at directly verifying whether, when and how stimulus modality and other potential moderators actually moderate blocking. We hope that the surprising lack of evidence for blocking in our studies provides the impetus for such a continued theoretical and empirical exploration of the boundary conditions of this important phenomenon.

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Tables

Table 1

Descriptives and results of pairwise parametrical and Bayesian t-tests comparing conditioned responding to last presentation of A or B and first presentation of AX per group

Exp.	Group	Measure	$M_{A/B}$	M_{AX}	SD_A	SD_{AX}	*t*-value	*df*	*p*-value	*d*	BF_{10}
4°	1	SR	0.10	0.03	0.12	0.04	1.64	9	0.14	0.52	0.85
4°	2	SR	0.04	0.05	0.06	0.08	-0.37	9	0.72	-0.12	0.33
10	1	SR	0	0	0.00	0.00	No statistics computed				
10	2	SR	0	0.13	0.00	0.25	-1.00	3	.39	-0.50	0.61
11	1	ES	3.83	3.00	3.19	2.28	0.64	5	.55	0.26	0.44
11	2	ES	4.50	2.67	1.98	4.27	1.29	5	.25	0.53	0.68
12	1	ES	3.33	-0.17	5.28	7.44	1.00	5	.36	0.41	0.55
12	2	ES	2.50	4.50	3.94	2.59	-0.89	5	.41	-0.37	0.51
13	1	ES	3.58	3.58	3.58	2.07	0.00	11	1.00	0.00	0.29
13	2	ES	3.08	3.25	2.35	3.11	-0.15	11	.88	-0.05	0.29
14	1	ES	2.33	4.17	3.68	4.37	-1.21	11	.25	-0.35	0.52
14	2	ES	4.58	1.58	3.18	4.17	2.19	11	.05	0.63	1.63
15	1	ES	5.87	-1.90	5.82	3.40	6.37	29	< 0.001	1.16	>100
15	2	ES	4.30	-2.23	3.79	3.76	6.92	29	< 0.001	1.26	>100

Note. Group 1 = Experimental Group; Group 2 = Control Group; SR = Suppression ratio; ES = Elevation Score. ° For Experiment 4, the means and standard deviations are calculated over the test session because trial-level data were not available.