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ORIGINAL ARTICLE

On costs and benefits of n−2 repetitions in task switching: towards a behavioural marker of cognitive inhibition

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Abstract Inhibition in task switching is inferred from slower reaction times returning to a recently performed task after one intervening trial (i.e. an ABA sequence) compared to returning to a task not recently performed (CBA sequence). These n−2 repetition costs are thought to reflect the persisting inhibition of a task after its disengagement. As such, the n−2 repetition cost is an attractive tool for the researcher interested in inhibitory functioning in clinical/neurological/neuroscience disciplines. In the literature, an absence of this cost is often interpreted as an absence of inhibition, an assumption with strong implications for researchers. The current paper argues that this is not necessarily an accurate interpretation, as an absence of inhibition should lead to an n−2 repetition benefit as a task's activation level will prime performance. This argument is supported by three instances of a computational cognitive model varying the degree of inhibition present. An inhibition model fits human n−2 repetition costs well. Removal of the inhibition—the activation-only model—predicts an n−2 repetition benefit. For the model to produce a null n−2 repetition cost, small amounts of inhibition were required—the reduced-inhibition model. The authors also demonstrate that a lateral-inhibition locus of the n−2 repetition cost cannot account for observed human data. The authors conclude that a null n−2 repetition cost provides no evidence on its own for an absence of inhibition, and propose reporting of a significant n−2 repetition benefit to be the best evidence for a lack of inhibition. Implications for theories on task switching are discussed.

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Introduction

Task switching research has become increasingly popular in recent years, driven by the desire to elucidate the processes that enable maintenance and flexible switching of cognitive processes (Kiesel et al., 2010). One mechanism thought to allow such flexible switching is inhibition of recently performed tasks. Evidence for inhibition in task switching comes from the Backward Inhibition paradigm (Mayr & Keele, 2000; Koch, Gade, Scuch, & Philipp, 2010; Mayr, 2007 for reviews), where participants are required to switch between three tasks, with the currently relevant task being signalled by a task cue (e.g. the word "Magnitude" to judge whether a number stimulus is higher or lower than 5). Findings from the backward inhibition paradigm show that performance is slower and more error prone returning to a task recently performed after just one intervening trial (i.e. an AB*A* sequence) compared to returning to a task not recently performed (CB*A* sequence). This *n−2 repetition cost* is thought to reflect the persisting inhibition of task representations in working memory (WM) when switched away from, thus hampering its reactivation if the task is required soon after (as is the case in an AB*A* sequence).

The n−2 repetition cost is attractive as a psychological phenomenon as it is—to date—robust against

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non-inhibitory accounts (Koch et al., 2010; Mayr, 2007). This is noteworthy, as inhibition is an important but controversial concept in cognitive psychology (Gorfein & Brown, 2007; Juvina, 2011a, b; MacLeod, Dodd, Sheard, Wilson, & Bibi, 2003); the controversy exists as many extant inhibitory phenomena can successfully be explained with non-inhibitory accounts (see e.g. Tipper, 2001). Therefore, the backward inhibition paradigm is an important tool for assessing inhibitory functioning in different populations. Indeed, some studies have begun to look at differences in n−2 repetition costs across different clinical/neurological populations (Fales, Vanek, & Knowlton, 2006; Mayr, Diedrichsen, Ivry, & Keele, 2006; Moritz, Hübner, & Kluwe, 2004; Whitmer & Banich, 2007). For example, Mayr et al. (2006) investigated n−2 repetition costs in patients with lesions to either the left- or right-prefrontal cortex (compared to age-matched controls). The main finding of importance was that—compared to left-prefrontal patients and control subjects, who showed the standard backward inhibition effect—right-prefrontal patients showed no n−2 repetition cost. The authors took this absence of n−2 repetition cost to reflect an inhibitory deficit in patients with damage to the right-prefrontal cortex, and by inference, that this cortical region is critical for inhibitory control. Thus, the study of the n−2 repetition cost has theoretical and practical/applied implications beyond that of the purely cognitive task switching literature.

On costs and benefits

Due to the importance of the n−2 repetition cost as a robust measure of cognitive inhibition and inhibitory ability in clinical/neurological populations, it is essential to establish to what extent an absence of n−2 repetition cost reflects absence of inhibition. Several studies to date have reported an absence of n−2 repetition costs in certain situations, either due to clinical/neurological reasons (e.g. Fales et al., 2006; Mayr et al., 2006; Moritz et al., 2004) or theoretical reasons (e.g. Arbuthnott, 2008b; Arbuthnott & Woodward, 2002; Druey & Hübner, 2007; Grange & Houghton, 2009, 2010, 2011a, b; Houghton, Pritchard, & Grange, 2009; Schuch & Koch, 2003). However, the interpretations of such findings are somewhat mixed (see "Appendix A"): Some suggest it reflects a total *absence* of inhibition, whereas some suggest it reflects *reduced* inhibition. If the latter were true, the n−2 repetition cost would be useful to assess the magnitude of the presumed inhibition deficit. For example, in the case of the Mayr et al. (2006) study, was the lack of n−2 repetition costs for right-prefrontal patients reflective of a total lack of inhibitory ability—and thus, does damage to the right-prefrontal cortex completely disrupt inhibitory control—or merely a reduction of inhibition? Due to a lack of consensus in the literature of what an absence of an n−2 repetition might reflect, it is essential that this question be addressed by researchers.

In the present article, the argument is formalised that an absence of n−2 repetition costs cannot exclusively be used to assume an absence of inhibition, a case of the familiar maxim that "absence of evidence is not evidence of absence" (e.g. Mari-Beffa, Estevez, & Danziger, 2000). Indeed, in an ABA sequence, if no inhibition is applied to task A at n−2, one would predict an n−2 repetition *benefit*, as the activation of task A should persist and prime behaviour (e.g. Altmann & Gray, 2008). There are several examples of a significant n−2 repetition benefit in conditions hypothesized to involve no inhibition (see e.g. Arbuthnott, 2005; Arbuthnott, 2008b; Schneider & Verbruggen, 2008). In the current paper it is proposed that when n−2 repetition costs in a study do not differ significantly from zero, some degree of inhibition must still be occurring; however, it is not reliably detectable as a reaction time cost as it is in effect cancelling out the expected benefit of repetition. If no inhibition occurs during a task switch, a significant n−2 repetition benefit should be found.

To investigate the plausibility of our proposal, we developed a series of explicit computational cognitive models. Oppositionists of inhibition often cite vagueness in descriptions of inhibitory theories (e.g. MacLeod, 2007), making them too flexible and hard—if not impossible—to falsify. Formal modelling addresses this issue by making explicit the processes under consideration, aiding development of a concise theory of inhibition that is transparent (MacLeod, 2007).

We should note that the models reported here do not claim to be a complete account of inhibitory processes in task switching. Rather, the models are developed to highlight and formalise an important caution to the researcher that an absence of an n−2 repetition cost does not automatically imply an absence of inhibition.

Modelling inhibition in task switching

For the purposes of demonstrating the possibility of inhibition being present in the absence of an n−2 repetition cost, we implemented our assumptions into a computational cognitive model in the adaptive control of thought–rational (ACT–R) modelling architecture (Anderson, 2007). ACT–R is a hybrid cognitive architecture, comprising symbolic structures (e.g. discrete memories and rules) and sub-symbolic structures (mathematical equations that control the symbolic processes, such as activation of memories and utility of rules).

ACT–R was chosen for several reasons. Most notably, a recent computational model built within the ACT–R architecture has been successful in producing many task switching phenomena (Altmann & Gray, 2008). However, as ACT–R is primarily an activation-based architecture, n−2 repetition costs were not modelled. Nevertheless, inhibition can be instantiated within ACT–R. For example, Juvina and Taatgen (2009) modelled response suppression in a modified Stroop task to explain a plethora of within- and between-trial effects by assuming responses are inhibited once performed so as to avoid perseveration.

Our approach to modelling inhibition in task switching was slightly modified from that of Juvina and Taatgen (2009) for simplicity. Specifically, we used a modified version of the base-level learning (BLL) equation that governs the activation of chunks (i.e. discrete memories) within ACT–R's declarative memory. The BLL equation assumes that once a chunk is activated, it decays passively as a power function over time making it less accessible. Activation is thus expressed formally as:

$$A_i = \log \sum_{j=1}^{n} t_j^{-d} \quad (1)$$

where A_i denotes the activation of a chunk i, n is the number of presentations of i, and t_j is the time since the jth presentation of the chunk; d is a decay parameter. Each time an item is presented, its activation increases, but decays as a power function of the time since the last presentation; these processes are then summed and passed through a log transformation. The activation of a chunk determines the probability of a chunk to be retrieved and the duration of its retrieval. Chunks that are more frequently and more recently reactivated are more likely to be retrieved and their retrieval is faster. In addition, a stochastic component (noise) has a minor influence on the probability and speed of retrieval.

To model inhibitory effects, where a chunk's activation is suppressed after its use, a modification of the BLL equation is required. One example was introduced by Lebiere and Best (2009), where an inhibitory component was added to the BLL equation in Eq. 1:

$$A_i = \log \sum_{j=1}^{n} t_j^{-d} - \log\left(1 + \left(\frac{t_n}{t_s}\right)^{-d_s}\right) \quad (2)$$

Equation 2 considers the time since the most recent reference of a chunk, t_n, an inhibition-scaling parameter, t_s, and an inhibition-decay parameter, d_s. The 1+ component of this equation ensures the result of the latter part of the equation is positive, meaning the log transformation is always positive, which produces an inhibitory effect when it is subtracted from the component that is identical to Eq. 1 (Lebiere & Best, 2009). The effects of these different activation equations are shown in Fig. 1, with arbitrary parameters, showing BLL (i.e. no inhibition) and the inhibition equation.

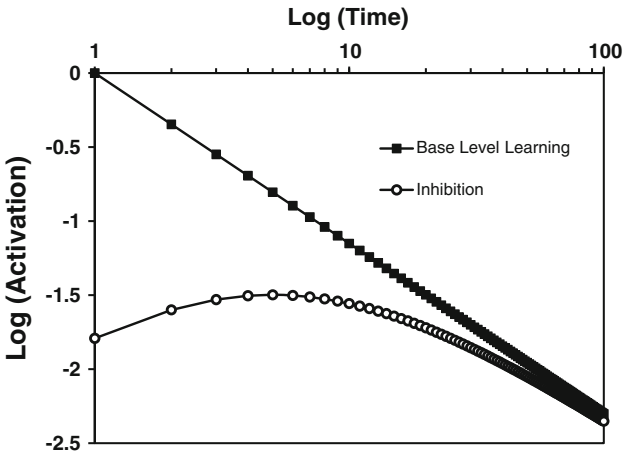

Fig. 1 Activation functions of different base-level learning equations in log–log space with the following arbitrary parameters: $d = 0.5$; $t_s = 5$; $d_s = 1$ (adapted from Lebiere & Best, 2009)

As can be seen, once a chunk has been activated according to the BLL equation, its activation decays—and hence becomes harder to retrieve (Eq. 1)—as a power function, linear in log–log space. Thus, the more recently a chunk has been activated the higher its activation. Conversely, the inhibition equation—Eq. 2—ensures a chunk is hard to retrieve immediately after its use, but this inhibition dissipates slightly over the next few trials before merging with the BLL function. In other words, a chunk does not reach its maximum activation immediately after its reactivation because of the short-lived inhibition component that is added at the time of reactivation (see the inverse U curve in Fig. 1).

It is clear from these equations that the BLL alone would predict an n−2 repetition *benefit*, as the activation of a task will persist—albeit at a decaying rate—until it is needed again at n. This forms the basis of the argument presented here, that in the absence of inhibition, pure activation will produce an n−2 repetition benefit. At the other end of the scale, a sufficient inhibitory component to Eq. 2 will produce an n−2 repetition *cost*, as the short-term inhibition of a task at n−2 will hinder its reactivation at n. It is the basis of the argument presented here that for the model to produce a null n−2 repetition effect of around zero, some inhibition must be required to counteract the persisting activation of a task.

We note here that the model is producing inhibition via a self-inhibition mechanism, whereby an activated chunk is inhibited immediately after its use. There is no consensus in the literature that n−2 repetition costs reflect

self-inhibition—indeed, some authors explicitly state that n−2 repetition costs reflect lateral inhibition (Arbuthnott & Frank, 2000; Arbuthnott, 1995; Koch et al., 2010; Philipp & Koch, 2006) where activation of task A, for example, spreads inhibition to tasks B and C (Arbuthnott, 1995; Houghton, 1993; Houghton & Tipper, 1994). We discuss each type of inhibition further in "General discussion", and provide simulation results of a lateral-inhibition model. We note for the reader at this point that lateral inhibition failed to produce n−2 repetition costs, unlike the self-inhibition models we now report.

Model implementation

These sub-symbolic equations were utilised within an ACT–R model that was programmed to simulate performance on Experiment 1 of Grange and Houghton (2011b, standard cues condition, i.e. first half of the experiment only). In this experiment, participants performed a target-localization task, wherein they were instructed to respond to the location of a cued target, with each target differing on a visual property (either an angled oval, a shaded oval, or an oval which had a thickened border; a fourth neutral oval was always present as a distractor). Participants were cued with one of three shapes (square, octagon, or triangle), with one cuing each target (square = shaded; triangle = border; octagon = angled). Cues were presented for 500 ms (ms), before a blank screen appeared for 250 ms; after this, the four targets appeared, with one centred to each quadrant of the screen. The targets remained visible until a spatially compatible response (i.e. a top-left key press to a target in the top-left quadrant) was made. After the response, a blank screen appeared for 250 ms before the onset of the cue for the next trial. As is typical in studies of n−2 repetition costs, immediate task repetitions were not allowed so as to maximise the number of ABA and CBA sequences for analysis. Participants performed ten blocks of 42 trials.

The implementation of the model followed three stages: we initially fit an inhibition-present instance of the model to data that shows an n−2 repetition cost, to demonstrate that the model can fit such data. Then, the inhibition was removed to show the effects of an inhibition-free model. Then, to investigate what would be needed to produce an n−2 repetition cost of zero, we re-introduced inhibition into the model, but at a reduced amount. To anticipate the findings, the *inhibition model* fit the n−2 repetition costs very well. Removal of inhibition—the *activation-only model*—produced an n−2 repetition benefit, as predicted. Of most interest, to produce an n−2 repetition cost indistinguishable from zero, we needed to re-introduce inhibition, albeit at a lower level than the

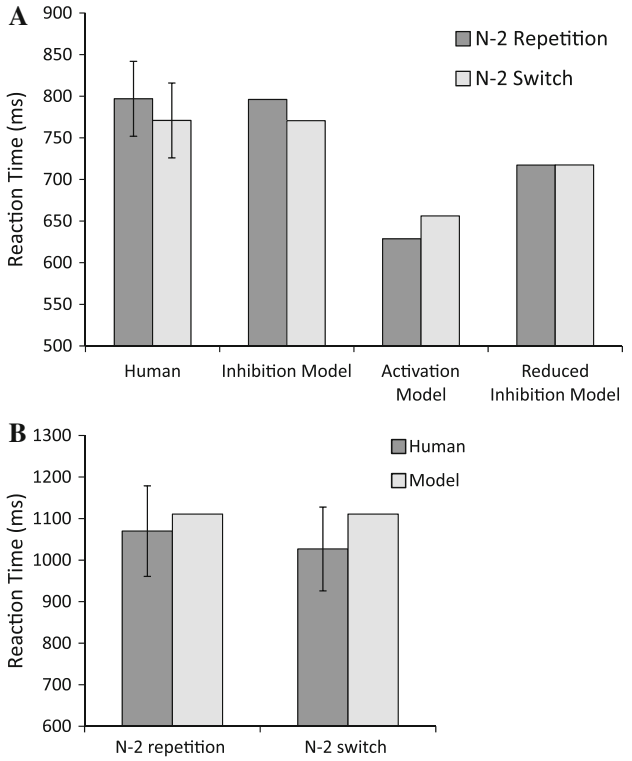

Fig. 2 **a** Model predictions (correct RTs) for all instances of the self-inhibition model for n−2 repetitions and n−2 switches against human performance. **b** Model predictions (error RTs) for inhibition model against human error RT data. *Error bars* denote one standard around the human data's mean

inhibition model (*reduced-inhibition model*). N−2 repetition cost (benefit) predictions of all of the models can be seen in Fig. 2.

We describe here the general structure of the model that is identical in all three instances of the model (inhibition, activation-only, and reduced inhibition). Then each instance of the model will be described in a separate section. To enhance readability, only a description in general terms will be given here; the source code of the model is provided as supplementary material. Table 3 in "Appendix B" provides the reader with all production rules that govern the model behaviour, and their plain-English interpretations. Read chronologically, "Appendix B" provides a clear step-by-step description of how the model behaves. Discussion of all components of the model—most notably the sub-symbolic equations not critical to our argument, such as how production rules are selected based on a calculation of their utility—are beyond the scope of this paper, and have been documented extensively elsewhere (Anderson, 2007).

The goal of the model is to detect the location of a target that matches a given cue and press the key that corresponds to the location of the target. To accomplish this goal, the model attends to, perceives, and encodes the cue in its goal

module. Then the model attempts to retrieve a memory representation of the target that corresponds to the perceived cue. The success and duration of this retrieval attempt depends on the activation of the representation to be retrieved. If a memory representation of the target is retrieved, the model performs a visual search in order to find the screen object that matches the retrieved memory representation. When the corresponding object is found, the model issues the motor response that corresponds to the location of the target object on the screen. If a memory representation of the target cannot be retrieved, the model responds randomly and potentially makes an error.

It is the retrieval of the memory representation of the target that is responsible for the effects discussed here (i.e. costs and benefits of n−2 repetitions). These representations are reactivated with each retrieval attempt that is successful. In the case of the inhibition and reduced-inhibition instances of the model, each representation is also inhibited after being retrieved (see Eq. 2).

Inhibition model

We simulated 100 participants by presenting the experiment procedure to the inhibition instance of the model, which uses Eq. 2 to determine activation levels of chunks. We fit the model to the human data reported in Grange and Houghton (2011a, b; their table 1, standard cues) as it is here that a more detailed presentation of the data exists. Specifically, the model was fit to correct reaction times (RTs), error RTs, and error rates (%). The model was fit by reducing the root mean square deviations (RMSD) and R^2 values between observed correct and error RTs, without straying too far from ACT–R's default parameter values. The model's RT predictions for correct RTs against human performance can be seen in Fig. 2a, and error RT predictions in Fig. 2b. (All model predictions can be found in Fig. 2a; a simplified graph focussing on the predicted n−2 repetition cost for each instance of the model is provided in Fig. 3.) Error rate in the human data was very low ($M = 3.25\%$), mirrored by the model predictions ($M = 3.5\%$), so we focus on the RTs. The best fitting parameters and fit statistics can be found in Table 1.

As can be seen, the model fit the human data very well. For correct RTs—the main dependent variable of interest in studies of inhibition in task switching—the model predicted an n−2 repetition cost of 25.6 ms, which is indistinguishable from the human n−2 repetition cost of 26 ms. Additionally, the model fit the RT (error) data reasonably well (Fig. 2b).

The good fit of the inhibition model to human n−2 repetition costs suggests that cognitive inhibition may be a necessary component of task switching, which extends the findings of Altmann and Gray (2008), as they did not attempt to model n−2 repetition costs. To our knowledge, this is the first model of n−2 repetition costs—and thus inhibition—in task switching.

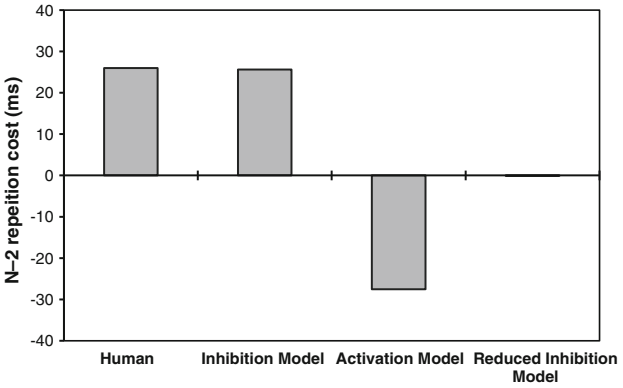

Fig. 3 N−2 repetition costs in milliseconds for human data and all instances of the model

Activation model

With the result of the inhibition model as a foundation, the consequences of removing inhibition from the model were explored in the activation instance of the model. To achieve this, we implemented the same model, but with chunk activation governed by BLL in Eq. 1. All other parameters in the model were held constant and another 100 participants were simulated. The model predictions for n−2 repetitions and switches are shown in Fig. 2a. As predicted, removal of the inhibition component of the activation equation reversed the n−2 repetition cost into a benefit of 27.5 ms. Without inhibition, n−2 repetition benefits arise due to persisting activation of task A across an ABA sequence.

Another result of interest from the model is that removal of inhibition does not only reverse the n−2 repetition cost into a benefit, but it also reduces overall RT considerably. This is driven by the fact that inhibition affects retrieval time for all chunks; therefore, even in a CBA sequence, task A will still be somewhat inhibited, thus slowing its retrieval. As a consequence, the model predicts a speeding of RT in the absence of inhibition. There is some evidence in the literature that lends support to this observation: some conditions that report a significant n−2 repetition benefit also have faster overall RTs than conditions that produce an n−2 repetition cost (e.g. Arbuthnott, 2008b; Schneider & Verbruggen, 2008; but see Arbuthnott, 2005, Experiment 2). Of course, there remains the possibility that the conditions reporting n−2 repetition benefits need no inhibition as they are easier, as reflected by faster RTs. However, Houghton et al. (2009, Experiment 2a) have shown that n−2 repetition costs (and hence, inhibition) are independent of task difficulty as measured by overall RT.

Table 1 The best fitting parameter values for the inhibition model and fit statistics

Parameter	Description	Default value	Fitted value
:lf	Latency factor	1.0	0.4
:bll	Base-level learning [decay *d*] in Eqs. 1 and 2)	nil	0.5
:ans	Activation noise	nil	0.25
:rt	Retrieval threshold	0.0	−0.6
:inhibition decay	Base-level inhibition decay (*ds* in Eq. 2)	1.0	1.1
:inhibition-scale	Base-level inhibition scale (*ts* in Eq. 2)	5.0	7.0

$R^2 = 0.987$, RMSD = 46.74 ms

RMSD root mean square deviation

It is important to note that in the absence of inhibition, the model does not predict an n−2 repetition cost of zero, supporting the argument of the current paper that an absence of inhibition should lead to a significant n−2 repetition benefit.

Reduced-inhibition model

To address the question of whether inhibition is required to observe an n−2 repetition cost of zero, inhibition was re-introduced to the model. The same parameters were used as in the inhibition instance of the model; however, the inhibition-scale parameter in Eq. 2, t_s, was reduced from 7.0 to 4.0. The predictions of the reduced-inhibition model that were obtained by simulating another 100 participants can be seen in Fig. 2a. As is clear from the figure, the model made similar predictions for n−2 repetitions and switches; thus, activation and inhibition of chunks are essentially balanced to produce an n−2 repetition cost of close to zero (−0.12 ms in the model).

From this simulation it is clear that some inhibition is required to produce a null n−2 repetition cost. Additionally, overall RT increases slightly with the introduction of reduced inhibition ($M = 717$ ms), albeit still being faster than the inhibition model ($M = 783$ ms) and human data ($M = 784$ ms). This finding is also congruent with reports of null n−2 repetition costs being accompanied by faster RTs than conditions which produce such costs (e.g. Houghton et al., 2009; see also Grange & Houghton, 2009, Experiment 1; Grange & Houghton, 2010, Experiment 2).

General discussion

In this article, we were interested in addressing whether an absence of n−2 repetition cost in task switching automatically implies an absence of inhibition. We have provided evidence from a family of computational models built within the ACT–R architecture that this is not the case. We successfully modelled an absence of an n−2 repetition cost with some inhibition present. Without inhibition, an n−2 repetition benefit arises, and it is only with some degree of reduced inhibition that an n−2 repetition cost of zero arises.

We first present a summary of implications of the modelling for the researcher interested in using the n−2 repetition cost as a tool to investigate inhibitory functioning; we then discuss possible limitations and extensions to the theoretical underpinnings of the modelling presented here. Specifically, we address whether a lateral-inhibition locus of the n−2 repetition cost explains the human data better than the self-inhibition mechanism reported in the present paper.

Implications for the researcher

The implications of this modelling effort are manifold and far reaching. Specifically, an n−2 repetition cost not significantly different from zero cannot automatically be considered evidence of a lack of inhibition, either due to clinical/neurological or theoretical reasons ("Appendix A"). An n−2 repetition cost of zero can imply that some inhibition being present—albeit a reduced amount—cannot be ruled out, and therefore provides a note to researchers to be cautious about interpreting a null n−2 repetition cost as reflecting no inhibition.

This finding has particular significance to those wishing to use the n−2 repetition cost as a behavioural marker of cognitive inhibition in clinical/neurological populations. For example, the conclusion of Mayr et al. (2006) that the observed absence of n−2 repetition cost for those patients with right-prefrontal lesions reflects an inhibitory deficit might need careful re-evaluation in light of the current simulations; it might be that these patients are deploying inhibition, but at a reduced level than control subjects. Of course, this raises a new question as to why these patients may be deploying less inhibition—it may well be that this is caused by some inhibitory deficit. Such questions are open for future research.

The current simulations also have implications for theoretical accounts of task switching performance. For example, some authors have taken a null n−2 repetition cost to reflect an absence of inhibition for theoretical

reasons (see "Appendix A"). For example, Houghton et al. (2009) reported a null n−2 repetition cost when task cues are transparent as to the cue–target relationship (i.e. presenting a cue that provides the target representation directly). They suggested that in such situations, as the cue provides the necessary WM representation directly, there is no conflicting information in WM when a task switches, thus requiring no inhibition. This is important theoretically as it provides an insight into what aspects of the task performance can trigger inhibition. However, in light of the current simulations, it might be more prudent to suggest that some inhibition may be occurring for transparent cues, but at a reduced level. It would then be left open to explain what is triggering this inhibition if it is not the representation of the cued target. Additionally, Schuch and Koch (2003) reported a null n−2 repetition cost in an ABA sequence if task B did not require a response (i.e. a no-go trial). The authors suggested that in this scenario, the conflicting response mappings for task B will not be implemented, and as such no inhibition of task A is required, thus providing evidence that inhibition targets response-related processes. Again, if a null n−2 repetition cost is caused by some degree of inhibition, what is the target of this inhibition? These questions remain open to future research.

At the other end of the argument, the present simulations may be used to argue that task switching does not require inhibition at all (Altmann & Gray, 2008) as the activation-only instance of the model was more than capable at switching tasks—indeed, due to the faster responding it could be argued that it was better at switching tasks than those models that required inhibition. However, despite the appeal of this minimalist approach, there is considerable evidence for inhibition in task switching (Koch et al., 2010) which a purely activation-based model could not fit without some further—new—assumption added to it.[1]

Although not intended to be a complete account of inhibition in task switching, the simulations reported here provide a framework from which to build a more complete model of inhibitory control. Such a model in future work would have to take into account established backward inhibition effects (Gade & Koch, 2005; Grange & Houghton, 2010; Houghton et al., 2009; Schuch & Koch, 2003), and other behavioural effects in task switching thought to be caused by inhibition, such as the switch cost (Hübner & Druey, 2006), the asymmetric switch cost (Arbuthnott, 2008a), and the mixing cost (Mari-Beffa et al., in press). Such work would also have to justify more the need for inhibition. For example, the compound-cue model of Schneider and Logan (2005) assumes no inhibition at all, yet is able to perform analogous to humans in task switching situations. In their model, task cues are encoded together with targets, and both are used as a compound-cue which probes long-term memory for the correct response; task performance is achieved purely by memory retrieval, with no recourse to inhibition. Transition effects—such as slower RTs for switches compared to repetitions—are explained by priming benefits of encoding repeated or related cues. This model, however, cannot account for n−2 repetition costs in its current form (Altmann, 2007; Gade & Koch, 2008), and as such, the n−2 repetition cost remains an important constraint on models of task switching, suggestive of the presence of an inhibitory mechanism.

On different mechanisms of inhibition

Self-inhibition

It is likely that a complete model of inhibition in task switching may have to alter some of the assumptions we have built into the present model. For example, the current model assumes inhibition in task switching is a self-inhibitory mechanism, suppressing a chunk's activation immediately after its use (see Mayr & Keele, 2000). Self-inhibition has successfully been used to explain a variety of sequential effects in cognitive psychology (Arbuthnott, 1995; Arbuthnott & Campbell, 2003; Campbell & Arbuthnott, 1996; Houghton & Tipper, 1994). However, some authors have suggested against a self-inhibition locus of the n−2 repetition cost in task switching. In particular, Koch et al. (2010) provide three arguments against self-inhibition: (1) n−2 repetition costs are still evident even when immediate repetitions are allowed within an experimental block (e.g. Arbuthnott, 2008b); (2) self-inhibition should produce costs for task repetitions compared to task switches, the opposite of which is found in the literature; and (3) if self-inhibition is occurring, the n−2 repetition cost should be insensitive to the characteristics of the task at n−1, despite evidence to the contrary. We deal with (1) and (2) together and (3) separately below.

N−2 repetition costs in the presence of immediate repetitions

If n−2 repetition costs in task switching reflect some form of self-inhibition, then one would expect immediate repetitions of a task to be accompanied by a cost (as the task has just been inhibited), quite the opposite of the "switch cost" reported in the literature (Kiesel et al., 2010). Indeed,

[1] There are some non-inhibitory accounts of n–2 repetition costs in the literature, but to date these have been unable to explain extant data (Mayr, 2007).

self-inhibition in a scenario with immediate repetitions would be counter-productive, as self-inhibition is best suited to situations requiring frequent and rapid switching of behaviour (Arbuthnott, 1995), as is usually the case in backward inhibition paradigms. However, we would note that perhaps the system can alter its strategy in situations where task repetitions are possible. Indeed, in a situation with just two tasks and with the possibility of immediate repetitions, task switching behaviour has been rigorously and successfully modelled using purely activation-based strategies (Altmann & Gray, 2008). We have demonstrated clearly in the present paper that pure activation cannot explain n−2 repetition costs in task switching, even when modelled within the same cognitive architecture (ACT–R). This provides some evidence that different processing strategies are perhaps in operation in situations where immediate repetitions are possible.

However, this observation is difficult to reconcile with the finding that n−2 repetition costs are still found in paradigms where immediate repetitions are possible (e.g. Arbuthnott, 2005): if inhibition does not occur when immediate repetitions are possible, what is driving the n−2 repetition cost? We would like to point out that the literature is not so clear on this issue. Specifically, Philipp and Koch (2006) directly compared n−2 repetition costs in a condition with immediate repetitions to a condition with no immediate repetitions, and found the n−2 repetition cost was significantly reduced when immediate repetitions were possible. Further work is needed to address the discrepancy between Arbuthnott's finding and that of Philipp and Koch before any firm evidence against self-inhibition can be ruled out.

Influence of n−1 on the n−2 repetition cost

If self-inhibition is the source of the n−2 repetition cost, then the characteristics of the task at n−1 should not influence the observed cost, contrary to what is reported in the literature. For example, the finding from Schuch and Koch (2003) of no n−2 repetition cost if n−1 was a no-go trial provides strong evidence against a self-inhibition account. Specifically, in an ABA sequence, self-inhibition predicts there should still emerge an n−2 repetition cost when B required no response as A was self-inhibited at n−2. The present model does not accommodate this finding, but again we note that the cognitive system may adopt a different strategy when anticipating the potential occurrence of a no-go trial compared to a condition where no such anticipation exists. Self-inhibition is not an adaptive strategy in situations where a task may be required immediately (as is the case in an n−2 repetition when n−1 required no response). Therefore, adoption of self-inhibition may be paradigm specific.

In a related vein, the self-inhibition assumption also states inhibition occurs at n−2; it might be more parsimonious to assume inhibition is triggered at n−1 when a different task is being activated, and there is conflict in the system. Indeed, the need to deal with conflict in this manner is a strong theoretical justification for the presence of an inhibitory mechanism in task switching (Houghton et al., 2009; Koch et al., 2010). However, although we do not discount the conflict-triggered inhibition account (Grange & Houghton, 2010; Houghton et al., 2009), we note that there is reason to suggest a self-inhibition mechanism may ideally be suited to situations in which task switches occur on every trial, as is the case in backward inhibition research. In such situations, the system does not need to wait to detect conflict in order to deploy inhibition in a situation where conflict is guaranteed on the next trial (as the next trial is guaranteed to be a task switch). A more flexible strategy may be to inhibit a task immediately after its use. This issue would have to be explored thoroughly in a more complete model of inhibition in task switching, and is an important step for future work.

Lateral inhibition

Lateral inhibition (LI) is a well-established neural mechanism whereby neurons responding to similar or related representations undergo mutual inhibition, implementing a form of competition which de-correlates their temporal activity profiles. This leads for instance to the sharpening of the tuning curves of sensory neurons when competing stimuli are present (Blakemore & Tobin, 1972; Sillito, 1975; Isaacson & Scanziani, 2011). In output systems, the same mechanism appears to play a crucial role in response selection, and is found in models of action selection and sequencing too numerous to cite (e.g. the contention scheduling mechanism of Norman & Shallice, 1980, and all related work). Given an initially broad set of activations, over competing representations, LI narrows the activation profile in favour of the strongest representation(s). Since the costs associated with task switching are generally seen to arise from the need to reduce heightened "uncertainty" in task-set activation, LI is a natural mechanism to incorporate in any activation-based model of task-set selection (e.g. Gilbert & Shallice, 2001). On this basis, it would appear most parsimonious to explain the n−2 repetition cost as due to task A (in an ABA vs. CBA sequence) having been "laterally inhibited" during the rise to dominance of task B at n−1, thus hampering its reactivation on the following trial. As noted earlier, this idea has been stated by a number of authors (Arbuthnott & Frank, 2000; Koch et al., 2010; Philipp & Koch, 2006). However, the

proposal does not work, because it ignores the fact that with LI (as typically understood) task A will also be inhibited during the (control) CBA sequence. Thus, while it is true that in a CBA sequence, activation of task B will inhibit task C, it will also send exactly the same inhibitory signal to task A (as a member of the set of competing alternatives), whether task A happens to have just been performed or not. In the control sequence CBA, task A is thus inhibited twice in succession, at n−2 when task C was performed, and again at n−1 when task B was performed. Thus, a lateral-inhibition account may actually predict a *cost* for n−2 switches compared to n−2 repetitions; the net effect would be an n−2 repetition benefit.

To formalise this argument, we produced a further, simplified, model which abstracts away from the details of the task being performed, which the ACT–R model simulates. This enables us to express lateral inhibition in its simplest and most widely implemented form. We refer the reader to "Appendix C" for the details of this simulation, which produces the output shown in Fig. 4.

Figure 4 shows the activation dynamics of task units through a CBA sequence. Tracing the pattern of activation of task A demonstrates clearly the "double-dip" inhibition when the tasks at n−2 and n−1 are performed. When the cue for task A onsets (as indicated by the arrow in Fig. 4), contrary to expectancy, task A is actually the least active among all task units, and would thus be the slowest to perform. The result is slower n−2 switches than n−2 repetitions, producing a net effect of an n−2 repetition benefit. Thus lateral inhibition, at least in its most widely implemented form, cannot account for n−2 repetition costs

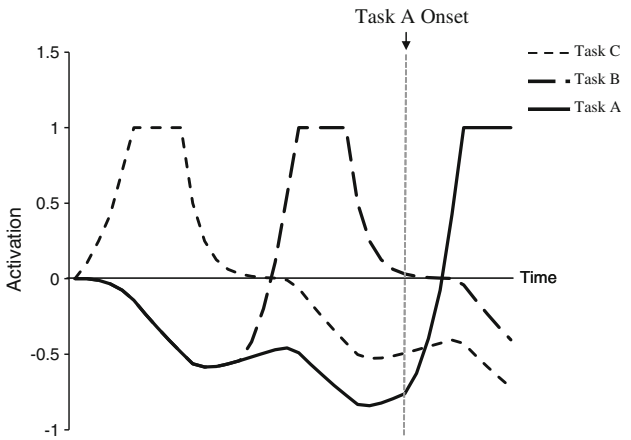

Fig. 4 Simulation of lateral-inhibition dynamics across a CBA task switching sequence (from the model outlined in "Appendix C"). Note at time of task A onset (*vertical grey dotted line*), task A is the least active due to it being laterally inhibited at n−2 and n−1. For details of the algorithm driving activation and lateral inhibition, see "Appendix C"

in task switching. We do not exclude the possibility that lateral inhibition might be relevant to n−2 repetition costs in some task switching situations (e.g. Arbuthnott, 2008a), but this would require further investigation, supported by computational modelling. We believe this to be a promising avenue for future work.

Conclusion

To summarise the contribution of this paper, we have provided a note of caution to researchers that an absence of an n−2 repetition cost is not automatically evidence for an absence of inhibition. It is proposed that the best evidence for a lack of inhibition during task switching consists of reporting a significant n−2 repetition *benefit*; only in this scenario can it be assured that no inhibition occurred.[2] Formal modelling of the assumptions lends strong support to the idea that researchers must go beyond an absence of n−2 repetition costs to implicate a lack of task inhibition. In addition, formal modelling of empirical data in task switching affords a rigorous quantification of inhibition in various populations. For example, one can assess the magnitude of a presumed inhibition deficit by comparing the repetition cost/benefit of a clinical population and a normative model. Future work must bring together experimental and theoretical modelling techniques to understand the nature of the n−2 repetition cost, and its use as a behavioural marker of cognitive inhibition.

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Appendix A

See Table 2

Appendix B

See Table 3

[2] Of course, one cannot be certain of *no* inhibition even in the presence of an n–2 repetition benefit without explicit modelling of the latent processes.

Table 2 A sample of studies that report n−2 repetition effects that are either not significantly different from zero, or are negative, together with a broad overview of how the authors interpreted the finding

Study	Was there a significant n−2 repetition benefit?	Interpretation
Arbuthnott and Woodward (2002)		
Spatial Cues	No	No/less inhibition
Arbuthnott (2005)		
Experiment 1 (spatial cues)	Marginal ($p = 0.092$)	Less inhibition
Experiment 2 (spatial cues)	Yes	Less inhibition
Experiment 3 (spatial cues)	Marginal ($p = 0.062$)	Less inhibition
Arbuthnott (2005)		
Experiment 2 (location switches)	Yes	No inhibition
Arbuthnott (2009)		
Distinct cue location	No	No inhibition
Druey and Hübner (2007)		
Experiment 1 (no temporal overlap of cues and targets)	No	Possibly no inhibition
Experiment 1 (spatially non-integrated cues and targets)	No	Possibly no inhibition
Experiment 2 (no temporal overlap of cues and targets at n−2	No	Both irrelevant tasks inhibited at n−1
Gade and Koch (2007)		
Experiment 1 (TUT trials)	No	No inhibition
Experiment 2 (TUT trials)	No	No inhibition
Grange and Houghton (2009)		
Experiment 1 (iconic cues)	No	No/less inhibition
Grange and Houghton (2010)		
Experiment 2 (matching cues)	No	No/less inhibition
Houghton et al. (2009)		
Experiment 1 (iconic cues)	No	No/less inhibition
Mayr et al. (2006)		
Right-prefrontal patients	Unclear	No inhibition
Mayr and Keele (2000)		
Experiment 3 (bottom-up condition)	No	No inhibition
Schneider and Verbruggen (2008)		
Experiment 1 (single-mapping)	Yes	No inhibition
Experiment 2	Yes	No inhibition
Schuch and Koch (2003)		
Experiment 2 (no-go at n−1)	No	No inhibition
Experiment 4 (double-press at n−1)	No	No inhibition

Appendix C

Implementation details of the lateral-inhibition model.

In a competitive network using LI as part of a selection mechanism, each element in the set of possible responses has inhibitory connections to all the others. The strength of this inhibitory connection, w^-, is usually equal for all connections and unchanging during processing. Thus any unit u_i, with activation level a_i will send an inhibitory signal of magnitude $w^- \times a_i$ to each of the others. Conversely, that unit u_i will receive a combined inhibitory signal $I = w^- \sum_{j \neq i} a_j$ from the set of units u_j, $j \neq i$.

To show how lateral inhibition is predicting an n−2 repetition benefit, we simulated LI in a simplified model computed in Visual Basic within Excel. In this case, each task (of 3) is represented as a single unit, with an activation level in the range [−1, 1] and a resting level of 0. A negative activation level represents a suppressed (below baseline) state and does not propagate. All three units are connected to each other via inhibitory links of equal strength. The only excitatory input to the model is an

Table 3 List of production rules—and their plain-English interpretation—that govern model behaviour

Production rule	Plain-English explanation
P attend-cue-location	IF the goal is to find a target with the state attend and there is something in the visual field THEN update the visual-location buffer to code where the object is and update the goal module to state start
P attend-cue	IF the goal is to find a target with the state start and there is a location in the visual-location buffer THEN request for the visual module to move visual attention to the location in the visual-location buffer and update the goal module to state encode
P encode-angled-cue-in-goal	IF the goal is to find a target with the state encode and there is a cue with the text property "angled" in the visual buffer THEN update the goal module with "angled" in the cue slot, and change the state to retrieve-target
P retrieve-target	IF the goal is to find a target with the state retrieve-target and there is a cue in the cue slot but no target has yet been encoded THEN make a retrieval request to declarative memory to retrieve the target description associated with the cue in the goal buffer and update the goal module to state attend-target
P found-target	IF the goal is to find a target with the state attend-target and there is a target description in the retrieval buffer (retrieved from declarative memory) and there is a new object in the visual field (i.e. the targets have appeared) THEN clear the retrieval buffer, the visual-location buffer, and clear the cue slot from the goal
P failed-to-retrieve-target	IF the goal is to find a target with the state attend-target but there is a retrieval error from the retrieval buffer (i.e. nothing could be retrieved) THEN update the goal module to state respond-randomly
P attend-target-top-left[a]	IF the goal is to find a target with the state attend-target and the cue slot of the goal is clear but a target has not yet been attended and there is a target in the top-left of the screen[a] THEN request for the visual module to move visual attention to the location top-left update visual-location buffer to the top-right of the screen[b] and update the goal module to state respond-top-left
P respond-top-left[a]	IF the goal is to find a target with the state respond-top-left and there is a target attended in the top-left which matches the description previously retrieved from declarative memory THEN make a request to the motor module to respond with a "D" key press and clear the cue slot of the goal and clear the target slot of the goal and change the goal state to attend [i.e. it now allows the model to start looking for a cue for the next trial, as (P attend-cue-location can now be selected)]
P respond-randomly	IF the goal is to find a target with the state respond-randomly THEN make a request to the motor module to respond with a random key press (i.e. "D", "C", "J", or "N") and clear the cue slot of the goal and clear the target slot of the goal and change the goal state to attend [i.e. it now allows the model to start looking for a cue for the next trial, as (P attend-cue-location can now be selected)]

[a] Only one location is discussed here, but the model had productions pertaining to all possible locations (top-left, top-right, bottom-left, bottom-right), but are excluded here for brevity

[b] This allows visual attention to move on the next target if the first one selected is not the correct target

external input I_{ext} representing the effect of a current task cue. On any trial only one task unit receives any such input, and that input builds up gradually (over 5 discrete time slices) to reach a maximum strength of 0.8. At each time t each unit u_i updates its activation level a_i according to

$$a_i(t+1) = \delta a_i(t) + I_i^{\text{ext}}(t) - I_i^{\text{inh}}(t) \quad (3)$$

(The activations are hard-clipped to the range $[-1, 1]$). In Eq. 3, δ is a decay (or recovery) parameter, which differs depending on whether the activation a_i is above or below baseline. For $a_i > 0$, $\delta = 0.5$, otherwise $\delta = 0.95$. The terms I_i^{ext} and I_i^{inh}, respectively, represent the excitatory external input, and the (internal) lateral inhibition to unit u_i. The latter is defined by

$$I_i^{\text{inh}}(t) = w^- \sum\nolimits_{j \neq i}^{n} a_j(t-1) \quad (4)$$

where w^- (=0.1) is the inhibitory weight as discussed above, and the summation is over all units other than u_i (recall also that sub-baseline activations do not propagate, i.e. they are treated as 0).

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