invoked, should the patients not have even stronger SCRs when they would confront the conscious conflict between what they would like to do, and what their 'impulse' would force them to do?

In closing, Maia and McClelland state that their 'participants report knowledge of the advantageous strategy more reliably than they behave advantageously.' This important finding is in keeping with those of economists who have long recognized that decision-makers often deviate from rational choices, despite prior knowledge that could lead them in a different direction [25]. The SMH addresses the possible physiological processes, conscious or not, intervening between knowledge and behavior, between what one knows and what one does, and suggests that emotion plays a key role (see also Box 2).

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Research Focus Response

The somatic marker hypothesis: still many questions but no answers

Response to Bechara et al.

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In the short space we have to reply to Bechara, Damasio, Tranel and Damasio [1] we will focus on three issues. First, we review important problems with their interpretations of our study. Second, we address the deficits of ventromedial prefrontal cortex (VMPFC) patients, as work with these patients played a major role in the development of the somatic marker hypothesis (SMH). We end by discussing the current status of the SMH.

Problems with Bechara *et al.*'s interpretation of our study

In our study [2], we found that participants report knowledge of the advantageous strategy more reliably than they *behave* advantageously. Bechara *et al.* state that

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this 'demonstrates yet again that even in normal participants, adequate knowledge of a situation does not guarantee correct decisions', suggesting that somatic markers (SMs) are required to explain this finding. There is, however, a simpler explanation. Any agent that is uncertain about the expected values of the outcomes associated with the available actions has to achieve a balance between 'exploration' and 'exploitation' [3]. To guarantee optimal behavior, participants *must* incorporate some variability in their behavior, to garner information about the different decks.

Bechara *et al.* also attempt to dismiss our suggestion that skin conductance responses (SCRs) in their original study [4] could have been due to conscious knowledge, arguing that 'the anticipatory SCRs to the bad decks began to occur in our study at a point at which Maia and McClelland do not claim the presence of "adequate knowledge", only "minimal knowledge" '. We disagree with this statement. First, in our study, most participants showed knowledge of the advantageous strategy right from the first question period. Second, as noted in our paper, our definition of advantageous strategy is different from that of Bechara *et al.* [4]: in our paper we show participants' knowledge of the best strategy according to their experience up to the relevant point in the game, whereas Bechara *et al.* show the SCRs for the $100 versus the $50 decks. Thus, a direct comparison between these results is inappropriate.

To address this issue properly, it is necessary to determine when our participants started exhibiting knowledge that the $50 decks were the good decks. We performed this analysis, and found that by trial 50, in all measures of explicit knowledge that we used, at least 80% of our participants demonstrated such knowledge. Bechara *et al.* [4] claim that their hunch period was on average between the 50th and 80th cards, and there is no statistically significant evidence that participants had higher SCRs for the $100 decks before that. Now, by trial 50, most of our participants already exhibited knowledge that the $50 decks were the good decks. They would therefore be expected to show higher SCRs for the $100 decks, even if such SCRs depended on conscious knowledge.

The deficits of VMPFC patients

In our paper [2], we suggested that the difficulties of VMPFC patients might be due to a deficit in reversal learning (RL) – the ability to adjust their responses when the reinforcement values of stimuli are reversed (for similar suggestions, see [5–7]). A deficit in RL would explain these patients' problems in the Iowa Gambling Task (IGT): at the beginning of the game the $100 decks seem better, and as the game unfolds participants have to overcome the tendency to select from those decks and switch to the $50 decks [2,6,7]. A study by Fellows and Farah [7] strongly supports this view. They used a 'shuffled' version of the IGT in which the order of card presentations was changed to avoid the initial advantage for the $100 decks; in this version the performance of VMPFC patients was similar to that of controls. These results seem problematic for the SMH: according to the proposals of Damasio, Bechara, and colleagues [4,8], VMPFC patients should have exhibited difficulties in the shuffled IGT too, because they would lack SMs.

The results of Fellows and Farah do not, by themselves, rule out the possibility that SMs originating from VMPFC might play the more limited role of supporting RL. In fact, confronted with the overwhelming evidence that VMPFC plays a crucial role in RL [5,6,9,10], Bechara *et al.* attempt to explain the deficit in RL in terms of the SMH. However, there is data suggesting that RL does *not* rely on SMs. In addition to projections to autonomic centers in the brain stem and hypothalamus through which it can orchestrate autonomic reactions, the VMPFC has direct projections to the striatum. Rolls notes that the latter projections could directly guide action selection, and argues that it would be noisy and inefficient for action selection to rely on markers that are generated in VMPFC, go through the body, and are then read back by the brain [5]. Importantly, lesions to the regions of the striatum that receive projections from VMPFC cause deficits in RL [5,6], just like lesions to VMPFC. Furthermore, the responses of neurons in those regions of the striatum reflect the output of neurons in VMPFC [11]. This suggests that RL is probably mediated via direct projections from VMPFC to the striatum – not via the generation of SMs by VMPFC, as suggested by Bechara *et al.*

It thus appears that the VMPFC plays a role in generating *both* autonomic responses *and* signals that can guide behavior, using different output routes. It therefore seems likely that the deficits in RL and in the generation of autonomic responses may be doubly dissociable within VMPFC. This should not be surprising, as VMPFC lesions in humans span a region that can be divided into nearly a dozen areas [12]. These deficits may tend to co-occur simply because naturally occurring lesions typically implicate several of these areas. The relevant investigations in VMPFC have not yet been performed. However, work on the amygdala has found just such a double dissociation between regions involved in the generation of autonomic and reflexive responses (the central nucleus) and those involved in the control of instrumental behavior (the basolateral amygdala) [13]. These findings pose a problem for the SMH: if bodily states were important in guiding instrumental behavior, interfering with the generation of those states should affect instrumental behavior.

Despite first arguing that a deficit in RL is due to problems with SMs, Bechara *et al.* also suggest that the fact that VMPFC patients typically perform well in the Wisconsin Card Sorting Task (WCST) shows that some VMPFC patients could do well on RL tasks and that their difficulties cannot therefore be explained by a RL deficit. This is, however, incompatible with much evidence that these patients fail RL tasks [9,10,14]. Furthermore, the kind of shifting required to perform well in the WCST, in which it is necessary to change from responding on the basis of one stimulus dimension (e.g. color) to another (e.g. shape), has been doubly dissociated from RL – the former involving lateral prefrontal cortex and the latter VMPFC [15].

The current status of the SMH

The main thrust of Bechara *et al.*'s arguments is that the SMH is compatible with our findings and other findings discussed in our paper. We do not entirely disagree. Indeed, we emphasized that 'our findings, together with these other findings in the literature, do not prove that the somatic marker hypothesis is wrong' [2]. Some of the results reviewed above are at odds with apparent predictions of the SMH; however, they are not conclusive. Nevertheless, what we have claimed, and what we think remains clear after an analysis of Bechara *et al.*'s arguments, is that there is currently no evidence calling *for* SMs. The key contribution of our findings was to show that SMs are not necessary to explain the results regarding normal participants in the IGT. For all other evidence that Bechara, Damasio, and colleagues have proposed to support the SMH, there are also alternative (and arguably more parsimonious) explanations.

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Research Focus

Developmental changes in the linguistic brain after puberty

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The development of reading skills is a complex and very long-lasting process. In an influential study Booth *et al.* demonstrated age-related changes in the activation of a network of left hemisphere regions, including the inferior frontal area, the superior temporal gyrus, and the angular gyrus. Interestingly, they found that the angular gyrus, which is involved in the mapping between phonological and orthographic representation, is automatically activated in adults during visual orthographic tasks not requiring this operation.

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Reading relies on the fast and accurate conversion of orthographic characters into their phonological representations. As a result of years of training and exposure to written and spoken language, skilled readers have faster and automatic access to phonology when reading and to orthography when listening [1,2]. Although this might seem obvious, the mechanisms involved in the development of reading skills have still not been thoroughly investigated. Neuroimaging research has provided useful information about which brain regions are active during reading in adults (see Figure 1). These same areas seem to be abnormally activated in dyslexic children [3]. However, the effects of post-pubertal brain maturation and extensive education remain largely