Cognitive and neural mechanisms of emotional memory

Stephan Hamann

A highly adaptive aspect of human memory is the enhancement of explicit, consciously accessible memory for emotional stimuli. Recent findings from neuroimaging, neuropsychological, drug and neural stimulation studies indicate that emotional stimuli engage specific cognitive and neural mechanisms that enhance explicit memory. Emotional arousal influences memory via factors that act during memory encoding (attention and elaboration) and factors that modulate memory consolidation. Across studies, the amygdala has been consistently implicated as playing a key role in enhancing explicit memory for both pleasant and unpleasant emotional stimuli through modulation of encoding and consolidation processes.

> An impression may be so exciting emotionally as almost to leave a *scar* upon the cerebral tissues... The primitive impression has been accompanied by an *extraordinary degree of attention*, either as being horrible or delightful. William James, 1890 (Ref. 1)

It has long been known that emotionally arousing events are more likely to be later recollected than similar, neutral events[2]. An extreme example of this enhancement of memory by emotion is the so-called flashbulb memory: a highly vivid memory for an intensely emotionally engaging event such as hearing the news of the death of a relative or cherished celebrity[3]. From an evolutionary perspective, emotional arousal, whether of an appetitive or aversive nature, signals an event or stimulus that is likely to have both immediate and future relevance to survival and reproductive success. Accordingly, it is adaptive to enhance memory for stimuli that elicit emotional arousal, thus ensuring that important information is available on future occasions[4].

Are there special mechanisms for emotional memory that are not engaged in the encoding and retrieval of non-emotional memory? Several psychological studies have suggested that the effects of emotional arousal on memory can be explained without needing to postulate special mechanisms for emotional memory[3,5]. By this view, ordinary cognitive factors such as increased rehearsal, enhanced attention and increased elaboration might be sufficient to account for the memory advantage observed for emotional stimuli. Although such factors clearly play some role in the enhancement of memory for emotional stimuli, specific neural and hormonal mechanisms do exist that enhance memory for emotional stimuli and that are not normally engaged by non-emotional stimuli[4,6]. Interestingly, these mechanisms appear to operate in part by recruiting the general cognitive mechanisms such as attention and elaborative encoding, the role of which has been emphasized in earlier behavioral studies[5,7].

This article examines recent findings regarding the cognitive and neural mechanisms involved in the encoding, consolidation and retrieval of emotional explicit or consciously accessible memory. Relevant data from neuroimaging studies of normal individuals using positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) that concern the key brain structures and mechanisms of emotional memory will be considered, as will data from neuropsychological studies, and from studies of the effects of drugs and of electrical neural stimulation. These findings will be related to a model that accounts for effects of emotion on explicit memory in terms of two classes of effects, both mediated by the amygdala: effects at time of encoding, including increased attention and elaboration, and post-encoding effects, including stress hormone release and enhanced consolidation of memory traces.

Only data from studies in humans will be reviewed. However, because many such studies have been guided by concepts arising from previous animal studies, correspondences between the findings in animals and humans will be briefly considered. Implicit or non-conscious forms of emotional memory have also been the subject of considerable recent interest[7,8]; however, the focus of this article is explicit emotional memory. It should be noted that, although this article focuses on the enhancing effects of emotional arousal on explicit memory, emotional arousal can also sometimes impair memory, particularly if high levels of stress and cortisol release are elicited[9,10].

Encoding versus post-encoding effects

Figure 1 illustrates the time course of processes that are associated with experiencing an emotionally arousing event. Encoding processes create the initial memory representation. After the event, post-encoding processes continue to influence the memory representation. The most important of these post-encoding processes is consolidation, a process by which new memories become more permanent and resistant to loss. The consolidation process is widely thought to require an extended period to complete[5]. Thus, the observed effects of emotion on memory should increase with time, as the gradual process of consolidation proceeds.

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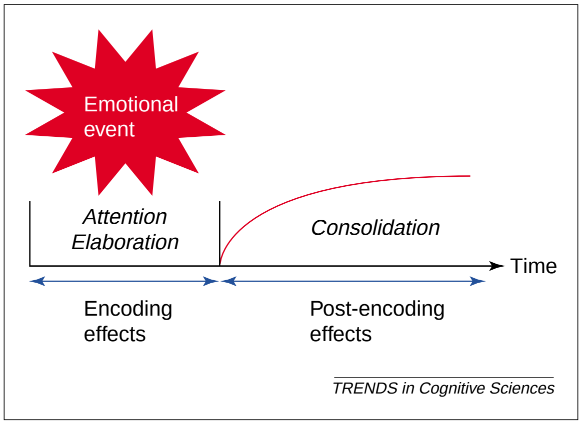

Fig. 1. Encoding and post-encoding effects of emotion. Encoding processes create the initial memory representation. After the event, post-encoding processes, primarily consolidation, continue to influence the memory representation. Consolidation is thought to continue for an extended period; therefore, the observed effects of emotion on memory should increase with time until consolidation is complete.

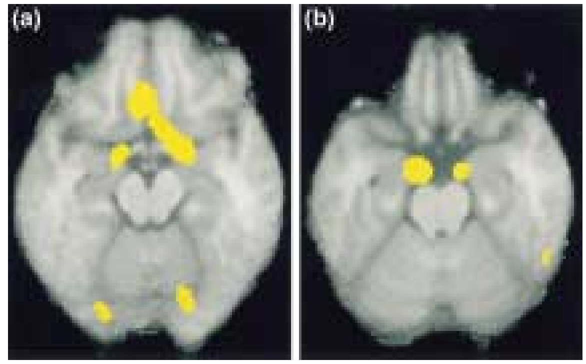

Fig. 2. Amygdala activity during encoding correlated with subsequent memory for positive and negative picture stimuli. (a) Correlation map for positive stimuli; (b) correlation map for negative stimuli. Correlated activity is shown in yellow, superimposed over averaged horizontal structural magnetic resonance image at $z = -10.5$ (a) and $z = -16.5$ (b) in the Talairach brain atlas.

Studies of emotional memory in humans have been strongly influenced by earlier animal studies by McGaugh and others that served to characterize basic structures and mechanisms[4,6]. Four key concepts have emerged:

(1) The amygdala is the primary orchestrator of processes of emotional memory, without which emotional effects on memory cannot occur.
(2) The amygdala can affect explicit memory by modulating or enhancing the activity of other brain regions involved in memory.
(3) Emotional arousal can affect explicit memory through the release of stress hormones that interact with the amygdala.
(4) The modulatory influence of emotional arousal via the amygdala acts specifically on consolidation processes in memory regions such as the hippocampus.

As will be evident in the following survey, each of these key concepts has now received considerable support from studies in humans.

Neuroimaging studies

Neuroimaging studies of explicit emotional memory have attempted to identify key regions that are preferentially involved in memory for emotional stimuli. Encoding studies have characterized brain regions in which increased activity during encoding successfully predicts later memory for emotional stimuli. Retrieval studies have characterized areas that are preferentially active during the retrieval of emotional memories. The role of the amygdala has been of particular interest in these studies.

In the first neuroimaging study of explicit emotional memory, Cahill *et al.* examined the relationship between brain activity at encoding and subsequent long-term memory for negative and neutral films in male subjects[11]. Brain activity was assessed using PET while subjects viewed the films. Subjects who showed increased brain activity in the right amygdala remembered more of the emotional films on an explicit memory test administered three weeks later than did subjects with lower activity. This correlation was observed only for the emotional films. Interestingly, a follow-up study with females found the same correlation but in the contralateral hemisphere (left amygdala). This preliminary finding suggests that sex differences might exist in the lateralization of emotional memory[12].

Hamann *et al.* examined the relationship between brain activity at encoding and later explicit memory for emotional and non-emotional picture stimuli[13]. For both pleasant and unpleasant stimuli, bilateral amygdala activity during encoding was correlated with later enhancement of recognition memory for these stimuli assessed a month later (Fig. 2) but not with recall memory assessed immediately after scanning. This correlation was not observed for non-emotional stimuli. These findings extend the results of Cahill *et al.* by showing that the amygdala can enhance explicit memory for both positive and negative stimuli (see Box 1). Hippocampal activity in this study was correlated with amygdala activity, supporting the view that the amygdala enhances explicit memory by modulating activity in the hippocampus.

Further support for the relationship between amygdala activity at encoding and subsequent memory was found in an fMRI study[14]. Brain activity was assessed during presentation of alternating blocks of positive and negative emotional photographs. Greater activity during viewing of negative versus positive photographs was associated with better recognition memory for the negative stimuli in regions including the amygdala (bilaterally), the insula (bilaterally) and several frontal lobe regions. In a subsequent event-related fMRI study, Canli *et al.* examined whether the neural response to individual negative pictures was related to subsequent memory[15]. Greater activity in the left amygdala was associated with better memory for individual negative pictures (Fig. 3). Notably, memory was enhanced only for the stimuli that produced the highest arousal, leading the authors to speculate that some minimum threshold of emotional arousal might exist below which amygdala activation does not result in memory enhancement.

Box 1. Positive and negative valence and emotional memory

Memory can be enhanced for both positive and negative emotional stimuli relative to neutral stimuli. Do similar or different systems mediate enhanced memory for positive versus negative stimuli? Most attention to date has focused on the amygdala. The amygdala has been much more consistently linked with negative emotion and emotional memory for negative stimuli. However, there are a small but growing number of studies that have implicated the amygdala in a corresponding role for positive stimuli. These studies complement an extensive literature from animal work that has demonstrated the role of the amygdala in learning to associate stimuli with affectively positive rewards.

Using functional neuroimaging, Hamann *et al.* and Dolan *et al.* have demonstrated a key role for the amygdala in both encoding[a] and retrieval[b] for positive and negative picture stimuli. Although few corresponding neuropsychological studies have examined memory for both positive and negative stimuli, one report has found impairment in emotional memory for both positive and negative pictures in an individual with bilateral amygdala lesions[c]. Johnsrude *et al.* have found impaired stimulus-reward association learning in individuals with unilateral amygdala lesions[d], extending the previous findings in animal studies to humans. In general, emotional arousal and associated amygdala activation appears to be the primary factor modulating memory for emotional stimuli, regardless of positive or negative stimulus valence[a,e]. However, there is also evidence that positive and negative stimuli can recruit additional, valence-specific processes[a,f–h]. For example, memory for positive stimuli has been linked to activation of reward systems[a].

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Tabert *et al.* examined the relationship between activity at encoding for negative emotional words and later recognition memory using fMRI (Ref. 16). The right amygdala was more active during encoding of negative words, but no correlation was found between this activity and immediately tested recognition memory for the negative words. As in the earlier study by Hamann *et al.*, the lack of a correlation between encoding activity and memory tested at a short delay was interpreted as supporting the consolidation model[13]. However, a recent report did find that increased amygdala activity at encoding for negative words versus neutral words was significantly correlated with enhanced recognition memory for negative versus neutral words tested after a short delay (<15 min), suggesting that the amygdala can modulate encoding processes as well as consolidation[17]. Low statistical power might account for the variable findings in these three correlational studies.

Retrieval of emotional memories

Turning to retrieval, emotional memory retrieval has been examined by assessing brain activity with PET during retrieval of emotional personal autobiographical memories and during the retrieval of experimenter-created memories for emotional picture stimuli. Retrieval of traumatic versus neutral personal memories by individuals with post-traumatic stress disorder activates limbic areas primarily in the right hemisphere, including the amygdala, anterior cingulate, and insular and temporal cortices[18]. Similar results were found in a study that examined the retrieval of personal autobiographical memories[19]. Increased activity in right prefrontal cortex and in right anterior limbic areas including the amygdala, hippocampus, temporal pole and insula were associated with retrieval of personal autobiographical memories, some of which were affectively laden.

In the first of two studies that examined experimenter-created emotional memories, brain activity was scanned during both the encoding and retrieval of emotionally arousing and neutral pictures[20]. The findings of this study were largely negative, however, with no significant activations in any of the regions of interest. By contrast, the results of the other retrieval study were highly informative[21]. Dolan *et al.* examined retrieval using an elegant factorial design that varied stimulus valence (negative, positive or neutral), retrieval intentionality (intentional versus incidental retrieval) and retrieval success. This study identified two distinct regions of brain activity specifically related to emotional memory retrieval. The temporal pole was related to the psychological set associated with engaging in emotional memory retrieval. By contrast, the left amygdala (Fig. 4) was activated only during the successful intentional retrieval of emotional items, indexing the actual retrieval of emotional memories. This left amygdala activation was observed both during retrieval of positive and negative emotional memories, mirroring the encoding results of Hamann *et al.* that implicated the amygdala in encoding for both positive and negative emotional stimuli[13]. These results also illustrate that the response of the

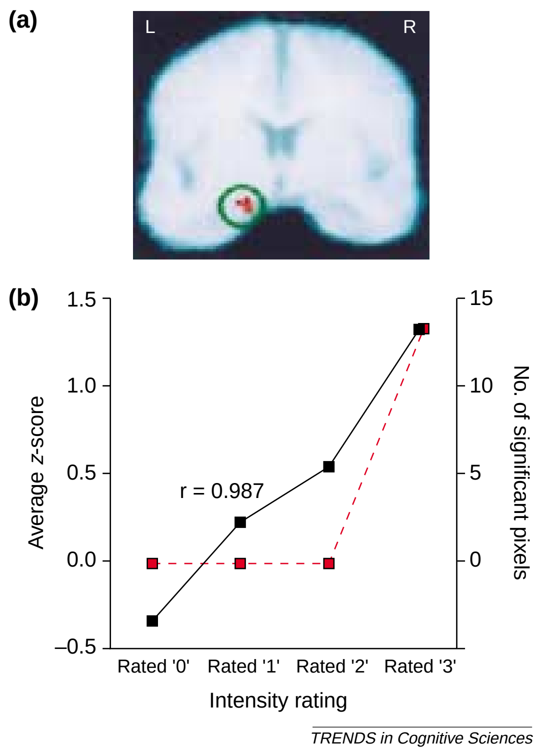

Fig. 3. Relationship between stimulus arousal and correlations between amygdala activity at encoding and later memory. (a) Region in which encoding activity for the highest-arousal negative items was correlated with subsequent memory; (b) graph indicating that the correlation between left amygdala activation and later memory increases with subject-rated arousal. Black data plot: *Z*-scores represent the strength of correlation. Red data plot (dotted): number of significantly correlated pixels within the amygdala. The x-axis shows the arousal rating ('0' lowest, '3' highest) given to each stimulus.

amygdala to negative emotional stimuli is not obligatory, but depends on the psychological context.

In summary, these neuroimaging studies have consistently found the amygdala and related limbic areas to be involved in both the encoding and retrieval of emotional explicit memory for negative emotional stimuli, and (less frequently) for positive stimuli. An issue for further study is determining what differential roles, if any, the left versus right amygdala have in emotional explicit memory encoding and retrieval.

Neuropsychological studies

Neuropsychological studies provide important converging evidence to support the findings of the neuroimaging studies, by showing that the areas identified by imaging are crucial for emotional memory, rather than simply showing correlated activity. Based on animal studies, bilateral amygdala lesions should abolish the enhancing effect of emotion on explicit memory.

An early study examined emotional memory in two individuals with bilateral calcification lesions of the amygdalae, which resulted from congenital Urbach–Wiethe disease. It was found that the subjects had some impairments in emotional memory for pictures and words[22]. Later, Cahill *et al.* tested emotional memory in an individual with Urbach–Wiethe disease using an emotional illustrated story paradigm[23]. The subject failed to show the normal enhancement of explicit memory by emotion, suggesting that bilateral amygdala damage blocks the normal enhancement of long-term explicit

Fig. 4. Left amygdala activity associated with successful intentional retrieval of emotional explicit memory for picture stimuli. The region of activation is shown in yellow, superimposed over an averaged coronal structural MRI section through the amygdala.

memory. This finding was later replicated by Adolphs *et al.* with another individual with Urbach–Wiethe disease[24]. Hamann *et al.* found that D.B.B., who had bilateral surgical amygdala lesions, failed to show normal emotional enhancement of memory for positive and negative emotional pictures[25]. Because memory was tested immediately in this study, it suggests that amygdala damage can impair the encoding of emotional explicit memory as well as its consolidation. Together, these results suggest that enhancement for emotional picture stimuli is highly dependent on amygdala-dependent mechanisms engaged by emotional arousal.

Turning to verbal stimuli, Phelps *et al.* examined an individual (S.P.) with bilateral amygdala lesions. S.P. showed normal emotional enhancement of memory for emotional verbal stimuli[26], as did a group of individuals with right- or left-sided amygdala lesions[27]. However, S.P. and individuals with unilateral amygdala lesion showed an abnormal forgetting rate for highly emotional 'taboo' words[28]. Enhancement of memory for emotional verbal stimuli appears to be less dependent on the amygdala than for picture stimuli[26,27]. A key factor determining whether amygdala damage affects emotional memory might be whether the verbal stimuli elicit sufficient emotional arousal: taboo words elicited physiological emotional responses but the non-taboo emotional verbal stimuli did not.

Damage to the hippocampus in the absence of amygdala damage should impair explicit memory generally, but should not affect the emotional enhancement of explicit memory. That is, the amygdala should continue to modulate whatever residual explicit memory function exists after hippocampal damage. This prediction has been confirmed: amnesic individuals with hippocampal damage show normal emotional enhancement of explicit memory despite their overall memory impairment[29,30].

Questions for future research

- What are the similarities and differences between the cognitive and neural mechanisms that mediate the effects of positive and negative emotion on explicit memory?
- What is the relationship between conscious (explicit) and non-conscious (implicit) aspects of emotional memory?
- What neural systems beyond the amygdala are engaged by explicit emotional memory and how does the amygdala interact with them?
- Is the amygdala necessary for the actual retrieval of emotional explicit memories, or is the amygdala activated post-retrieval by the emotional arousal associated with the retrieved memory?

Damage to the amygdala alone does not appear to affect markedly either the subjective experience of emotional arousal or physiological emotional responses[23–27]. That is, individuals with amygdala damage appear to be impaired specifically in the mechanisms that enhance explicit memory for emotionally arousing stimuli, but show normal reactivity to emotional stimuli. Thus, there appear to be dissociable neural bases for enhanced emotional explicit memory and emotional reactions.

Because the amygdala undergoes pathology[31] early in Alzheimer's disease (AD), emotional explicit memory should be impaired in individuals with AD. One report has found that individuals with AD do not show memory enhancement for negative pictures[32]; however, they did show normal emotional memory in two studies that used the emotional story used in the studies by Cahill[33,34]. Finally, a study of individuals with AD who survived a devastating earthquake found that memory for that highly emotional event was significantly correlated with the amygdala volume[35]. Together, these results suggest that impairments in emotional memory exist in individuals with AD, and that the degree of impairment is dependent on task and the degree of amygdala dysfunction.

Encoding versus consolidation effects

These findings agree well with the model developed by McGaugh[4,6], based on earlier animal studies in which the amygdala modulates long-term memory consolidation. However, this model cannot completely account for all the data obtained from studies in humans. For example, a crucial aspect of the concept of consolidation is that it takes place gradually over an extended period[6,36]. However, the proposed timescale for consolidation has varied widely across studies, from as short as 30 min to several months. Even if the shorter estimates of consolidation time are correct, it is clear from several studies that explicit memory for emotional stimuli can be substantially enhanced even at immediate delays[13,16]. These immediate effects cannot be accounted for if the sole mechanism for enhanced explicit emotional memory is consolidation.

Clearly, there must be effects operating during encoding that can enhance emotional memory independently of consolidation. As previously noted, a host of behavioral studies have demonstrated that emotional arousal enhances attention to stimuli and leads to more elaborated memory representations[5]. Most recent studies, however, have focused solely on memory consolidation. Importantly, most of the studies that have been interpreted as supporting the role of emotion on consolidation processes in fact provide only partial support for this role, because they have assessed memory at only one point in time, usually after a long delay. To provide compelling evidence for emotional consolidation effects, a study must assess memory both before and after consolidation has occurred. In addition, memory enhancement associated with emotion should increase over time, and variables proposed to affect or be related to consolidation should exhibit their effect or relationship only after consolidation has taken place. Only a handful of studies meet these more stringent criteria[13,27].

A further consideration is the timescale of emotional effects on explicit memory. The noradrenalin-mediated activation effects linked to emotional memory are quickly initiated but persist for several minutes[4,36]. Many experimental paradigms, however, alternate rapidly between emotional and neutral stimuli. For example, Canli *et al.* presented emotional and neutral pictures during encoding every 15 s in a random order[15]. Persisting adrenoceptor activation and memory enhancement would be expected to carry over from emotional to the neutral stimuli in this paradigm, yet memory was enhanced for only the emotional stimuli. The specificity of this effect suggests that the memory enhancement was due, in part, to encoding processes such as increased attention, which are necessarily time-locked to each stimulus. A related possibility is that encoding and consolidation mechanisms interact so that emotionally arousing items are processed differently during encoding, in such a manner that their long-term consolidation is enhanced[37]. A more complete account of emotional effects on consolidation will require further exploration of these issues.

Conclusions

Each of the key concepts from the earlier animal studies has received considerable support from these neuroimaging, neuropsychological, drug and neural stimulation studies in humans (see also Box 2). The crucial role of the amygdala as modulator of explicit emotional memory has been supported by the observed effects of bilateral amygdala lesions and correlations between amygdala activity at encoding and later emotional memory enhancement. An important issue for future research will be to relate activity in the amygdala to broader networks that mediate emotional memory. For example, enhancement of visual attention and visual explicit

Box 2. Drug and stimulation studies

Drug studies of emotional explicit memory in humans have attempted to either reduce or increase the enhancing effect of emotion on memory by either blocking or stimulating the noradrenergic system. Cahill *et al.* found that administering propranolol, a drug that blocks β-adrenoceptors, blocked emotional enhancement of memory in an emotional story task[a]. This initial study provided an important link to the previous animal studies, by suggesting that enhancement of long-term explicit memory in humans by emotional arousal depended on β-adrenoceptor activation. However, it was unclear whether the effects observed in this study were central (brain based) or peripheral. Later drug studies implicated central effects more than peripheral effects. Peripheral but not central β-adrenoceptor block[b,c] failed to impair emotional enhancement, but combined peripheral and central blockade impaired the emotional enhancement[d]. In addition, stimulating the noradrenergic system with the drug yohimbine increased the degree of emotional memory enhancement.

Other data more clearly implicate peripheral mechanisms. Electrical stimulation of the vagus nerve, which carries peripheral sensory information from the viscera to the brain, produces robust enhancement in verbal explicit memory for neutral words when administered after learning[e]. This study highlights a route by which information about physiological arousal in the periphery can reach the brain and enhance memory. Together, these drug and stimulation studies point to the crucial role of stress hormones and physiological arousal in mediating the effects of emotion on explicit memory.

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memory by the amygdala might involve its well-characterized back-projections to visual processing areas. Several findings support the idea that the amygdala can enhance consolidation of memory for emotional stimuli. In addition, the enhancement of encoding processes by emotional arousal has been shown to be an important counterpart to consolidation-based explanations of emotional memory effects.

The recent wave of cognitive neuroscience studies that have examined the cognitive and neural mechanisms of emotional explicit memory have both confirmed the major findings from the earlier studies with animals and humans, and have provided new insights into these mechanisms. The challenge for future studies will be to combine the converging data from diverse methodologies into integrated conceptual and computational models of emotional memory.

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Telling what we know: describing inner experience

Russell T. Hurlburt and Christopher L. Heavey

It is claimed that psychological science can obtain accurate reports about people's inner experience. We reconsider three criticisms of introspection: Nisbett and Wilson's critical review of introspection, the failure of introspectionists to agree about imageless thought, and Skinner's behavioral position. We show that rather than dismissing introspection, these criticisms point the way towards technical improvements in the methods used to produce accurate descriptions of inner experience. One such method, Descriptive Experience Sampling, is described and used as an example to illustrate our conclusion that, although exploring inner experience is not trivially easy, it can provide important knowledge for many areas in cognitive science.

There is a chasm in psychological science. On one side are those who think that describing inner experience is essentially impossible and unnecessary – methodological behaviorism, for example. On the other side are those who think that describing inner experience is trivially easy and vitally important – cognitive psychotherapy, for example (which rests on the question 'What were you thinking when…?'). Our opinion is that both sides contain an element of truth. We reconsider the arguments on both sides and show that for each it is possible to separate the kernel of truth from the chaff of overgeneralization and thereby to bridge the chasm. It is possible, but not trivially easy, to provide accurate descriptions of inner experience, and doing so will advance cognitive science.

Our opinion rests largely on our experience with a method designed to give descriptions of inner experience – Descriptive Experience Sampling (DES). It is not our intention in this brief discussion to convince the reader of the adequacy of DES or any other particular method. We have discussed the validity of DES reports elsewhere[1–3]. We have established the reliability between raters of one DES rating system[4] and have demonstrated the connection of inner experience to external behavior[1–3,5]. We will, however, use DES examples as illustrations, so we provide here a brief description of the method. DES uses a beeper to cue subjects, at random times, to pay immediate attention to their ongoing experience at the moment they heard the beep. They then jot down in a notebook the characteristics of that particular moment, and subsequently (within 24 hours) describe the characteristics of that sampled moment in an in-depth interview[1–3]. Those interviews ask only one question (although it is phrased in a wide variety of ways): '*What was occurring in your inner experience at the moment of the beep*?'

It should be said that, upon hearing of DES for the first time, many scientists (and most prospective subjects) simulate DES in thought by imagining wearing a beeper, hearing it beep, and responding to the question, 'What was I experiencing at the moment of the beep?' In such a simulation the only possible answer to that question is, 'I was wondering what my experience would have been had I been beeped right now', so DES seems impossible or at best trivially uninteresting. However, when they try the actual DES procedure, most subjects find that the beep easily 'catches experience in flight'. Why the discrepancy? In the actual DES procedure, a real beep comes from outside the subject and (with some training) serves as a practically immediate cue to pay attention to actual ongoing experience. In the simulation, the subject first pretends to create the cue and then pretends to respond to an experience that is pretended *not to include the creation of the cue*. The simulation simply does not mirror the actual DES procedure. Does the actual beep affect inner experience in some way? Of course. However, most subjects, including sophisticated subjects, report that the disruption is

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