Can cognitive neuroscience illuminate the nature of traumatic childhood memories?

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Recent findings from cognitive neuroscience and cognitive psychology may help explain why recovered memories of trauma are sometimes illusory. In particular, the notion of defective source monitoring has been used to explain a wide range of recently established memory distortions and illusions. Conversely, the results of a number of studies may potentially be relevant to forgetting and recovery of accurate memories, including studies demonstrating reduced hippocampal volume in survivors of sexual abuse, and recovery from functional and organic retrograde amnesia. Other recent findings of interest include the possibility that state-dependent memory could be induced by stress-related hormones, new pharmacological models of dissociative states, and evidence for 'repression' in patients with right parietal brain damage.

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Abbreviation
NMDA   *N*-methyl-D-aspartate

Introduction
Cognitive neuroscience studies of memory have many important implications for everyday life. Such implications are nowhere more evident than in the recent explosion of cases in which adult women and men, usually in the context of psychotherapy, claim to have recovered long-forgotten memories of childhood abuse suffered at the hands of parents, friends, or other adults. The memories range from single incidents of inappropriate fondling to years of rape and even ritualistic abuse. People who recover such memories are often certain that they reflect actual past events. This conviction is shared by some psychotherapists, who have argued that memories of sexual abuse can be repressed and later recovered (e.g. [1–3]). Yet, those who are accused of perpetrating the abuse frequently deny that the incidents ever occurred. A variety of psychologists, psychiatrists, and others have argued that recovered memories are frequently illusory and are attributable to suggestive practices used in psychotherapy (cf. [4•,5–8]).

The recovered memories debate raises issues that are relevant to cognitive neuroscience. How accurate is memory and under what conditions is it subject to distortion? Can traumatic events be forgotten, and if so, can they be later recovered? We first consider evidence that pertains to claims of recovered memories of trauma. We then consider the relevant memory phenomena in the context of concepts and findings from the contemporary cognitive neuroscience of memory.

The recovered memories debate: what do we know?
The controversy over recovered memories is a complex affair that involves several intertwined psychological and social issues (for elaboration of this point, see [8–13]). Here, we consider four critical questions. First, can memories of abuse be forgotten? Second, does the evidence warrant the postulation of a special mechanism of repression? Third, can memories of childhood trauma, if forgotten, later be remembered accurately? And, finally, is there evidence that false memories of abuse can occur?

Can memories of abuse be forgotten?
In several studies, patients who reported that they were sexually abused as children also said there were periods of time in the past when they had forgotten about the abuse [14–17]. However, these studies provide only weak evidence that abuse can be forgotten, because, first, none of them contained corroborating evidence that the reported abuse had actually occurred, and second, all of them relied on retrospective estimates of forgetting, which are of questionable validity (for a discussion, see [5,8,18,19]). Stronger evidence for forgetting has been provided by Williams [20••], who found that 38% of women who had been brought to a hospital emergency room as children for treatment of abuse failed to report the incident when interviewed two decades later. Most remembered other episodes of abuse, but 12% reported no memory of any abuse. Likewise, several individual cases have been reported in which incidents of corroborated abuse were temporarily forgotten (e.g. [13,21]). Thus, although the evidence indicates that most adults who were abused during childhood always remember their abuse, it also shows that some abusive episodes can be forgotten.

Does the evidence warrant postulation of a special mechanism of repression?
Although it is apparent that forgetting of abusive events can occur, ordinary mechanisms of forgetting, such as decay, interference, or infantile and childhood amnesia, are probably sufficient to explain inaccessibility of some traumatic incidents [22,23]. For example, when people fail to remember single incidents of sexual abuse that occurred when they were children (e.g. [20••,21]), forgetting may

be caused by the same ordinary mechanisms that are responsible for forgetting of non-traumatic experiences.

In contrast, when people claim to have forgotten about extended periods of repeated and horrific abuse, something more than ordinary forgetting is probably involved. Although there is little firm evidence for such extraordinary forgetting, some researchers have invoked the concept of repression to account for it (e.g. [1,3]). The notion of repression has a long and controversial history, dating to Freud's early contributions (see [24]), and the strength of the evidence for it depends on how the concept is defined.

On the one hand, repression may be defined as a process of conscious avoidance, in which a person fails to think about, talk about, or otherwise rehearse an unpleasant experience. Cognitive research has shown that such motivated or 'directed' forgetting can lead to a reduced likelihood of recalling an event (see [25] for a review). On the other hand, repression may be defined as an automatic, defensive process that functions to exclude threatening material from awareness. However, little or no experimental evidence for this latter kind of repression exists [18,26]. With respect to the recovered memories debate, the consciously motivated form of repression that results in failure to rehearse or think about traumatic events could account for gradual forgetting of an abusive episode or episodes over time (see, for example, [27]). However, this form of repression does not seem powerful enough to produce severe amnesia for repeated, horrific events soon after they occur.

Can memories of childhood trauma, if forgotten, later be remembered accurately?

The fact that some episodes of abuse can be forgotten need not mean that they can be recalled again years later. However, several cases have been reported in which people who have recovered previously forgotten memories of abuse have obtained corroboration that the abuse occurred (e.g. [13,21,27]; for discussion of corroborated cases, see [8,12]). Recovery of such memories may simply reflect the well established fact that appropriate retrieval cues can produce recall of aspects of seemingly forgotten experiences (e.g. [28,29]).

Is there evidence that false memories of abuse can occur?

There is no direct experimental evidence that illusory memories of sexual abuse can be created, and such a demonstration is precluded for ethical reasons. Nevertheless, several lines of evidence support the conclusion that illusory memories of abuse do indeed occur. First, some techniques used by therapists to recover forgotten memories, such as hypnosis (see [30]), are known to produce subjectively compelling pseudo-memories in suggestible subjects [31,32], including memories for having been abused in a 'past life' [33]. Second, some people claim to have recovered memories of previously forgotten ritualistic abuse in satanic cults, yet no evidence for any such abuse

has ever been uncovered despite extensive investigations by law enforcement agencies [7,34,35]. Finally, a growing number of people have disavowed or 'retracted' their recovered memories, and recent evidence indicates that many of these individuals were treated by therapists who used suggestive techniques to recover memories [36].

Our brief overview of recovered memories reveals, therefore, that some may be accurate and others illusory. We now examine insights and evidence from cognitive neuroscience that are relevant to both sides of the issue.

Illusory memories: cognitive and neurobiological perspectives

Memory usually preserves a reasonably accurate representation of the past. Nonetheless, most researchers acknowledge that memory does not preserve an exact or 'photographic' representation of all aspects of past experiences. Instead, memory is a fundamentally constructive process. Cognitive psychologists have argued that memories of past experiences are constructed from several sources: stored fragments of an event; pre-existing knowledge, beliefs, and expectations that the rememberer brings to an experience; and properties of the environment in which the experience is retrieved (cf. [12,37–42]). Likewise, neuroscientists have argued that memories are constructed on the basis of stored fragments of experiences that are distributed throughout a variety of cortical regions and are bound together by systems that work co-operatively with cortical storage areas during encoding and retrieval (cf. [43–46]). From both the cognitive and neurobiological perspectives, memory distortions are a natural by-product of the fundamentally constructive nature of the memory process [12].

Recent research has begun to illuminate the cognitive and neurobiological factors that contribute to illusory memories. One important phenomenon is known as source memory or source monitoring—remembering when, where and how a memory was acquired [39]. Numerous studies with college students have shown that recollections of external events and internal imaginings can be confused, thereby producing distorted memories (e.g. [47]). Failures of source memory also play a key role in memory distortions that occur when people are exposed to misleading post-event suggestions, as observed initially in studies by Loftus and colleagues (e.g. [48]). When people witness a particular event (e.g. a car stopped at a stop sign), and are later given misleading information about it (e.g. the car stopped at a yield sign), they often fail to remember whether the critical information was part of the original event or was only suggested to them later (e.g. [49–51]).

Source memory has also been implicated in recent studies showing that some young adults can be induced to create false memories of childhood experiences in response to repeated questioning [52,53]. For instance, Hyman *et al.*

[52] asked college students about unusual events from their past that, according to their parents, had never occurred (e.g. an overnight stay at a hospital for an ear infection at age five, or a birthday party with pizza and a clown). In two separate experiments, no students initially remembered any such event. However, after being repeatedly questioned about the event, 4 of 20 subjects (20%) in experiment 1, and 13 of 51 subjects (26%) in experiment 2 developed an elaborate illusory memory. Hyman et al. [52] suggest that with repeated questioning, subjects may recall isolated fragments of actual childhood events and then misattribute them to the fabricated target event.

Source memory also plays a role in another recently described form of memory distortion. Roediger and McDermott [54•], using a procedure originally introduced by Deese [55], showed that people who study a list of words, each associated to a non-presented theme word, subsequently often incorrectly 'remember' having encountered the non-presented theme word (e.g. subjects who study 'drowsy', 'bed', 'tired', 'pillow', 'rest', 'pajamas', and other related words later claim with high confidence to remember having been exposed to the theme word 'sleep', even though it was not presented). Schacter, Verfaellie, and Pradere [56] showed that amnesic patients are less susceptible than normal subjects to this memory distortion, and argued that the illusion is based on recollection of the semantic gist of the studied lists, together with inadequate monitoring of the source of the memory.

Source memory appears to depend critically on the functioning of frontal lobe systems that are involved in strategic retrieval and monitoring of past experiences [57–61]. Indeed, patients with frontal lobe damage exhibit a variety of memory illusions and distortions [62–66].

It is not yet known whether source memory failures play a role in illusory memories of abuse. However, some false memories may be created when elements of actual experiences are recalled and their source is forgotten, with the result that something that was said, suggested, or imagined is mistaken as an actual event from one's past. Recent neuroimaging research indicates that some of the same posterior brain regions involved in perceiving are also involved in imagining [67,68], which may be one reason why experiences that are only imagined can nonetheless be experienced as real.

Forgetting and recovery of memories of abuse: what can cognitive neuroscience offer?

In this section, we consider several lines of evidence that are potentially relevant to forgetting and recovery of traumatic experience. As stated earlier, however, it remains unclear whether explanations above and beyond ordinary forgetting and conscious avoidance are required to account

for documented cases of forgetting and recovery (e.g. [13,21,27]).

Brain systems in victims of sexual abuse

A recent study of abused women (MB Stein et al., personal communication) revealed abnormalities in the hippocampal region, which is known to be critically important for explicit or declarative memory [69–71,72••,73]. Stein et al. (MB Stein et al., personal communication) used structural magnetic resonance imaging (MRI) to examine the brains of 22 women with a history of prolonged and severe sexual abuse. They found a significant reduction (5%) of left hippocampal volume in abused women compared to non-abused women. Although alternative interpretations of the observed hippocampal volume reductions are possible (cf. [74•]), decreased hippocampal volume might be related to toxic effects of glucocorticoids that are released in response to prolonged stress [75•,76,77], and can produce memory deficits [78•,79•,80]. However, none of the women in Stein et al.'s study (MB Stein et al., personal communication) were amnesic for their abuse, and, as a group, they showed normal performance on laboratory tests of explicit memory for recently studied information.

Studies of women with reported histories of sexual abuse have revealed some deficits in recalling autobiographical incidents [81•,82•]. However, these deficits — involving the failure to retrieve specific episodic childhood memories in response to single word cues or the tendency to retrieve 'overgeneral' memories that do not refer to a single episode — may not be specifically linked to hippocampal function, and may reflect a combination of deficient encoding and retrieval strategies [83]. Moreover, even if hippocampal volume reductions in abuse survivors do produce memory deficits, they would not readily explain recovery of forgotten traumas. Thus, there is currently no evidence that hippocampal volume reductions in survivors of sexual abuse are related to forgetting of traumatic experiences.

Retrograde amnesia: a model for forgotten trauma?

Retrograde amnesia refers to impaired memory for experiences that occurred before brain injury or psychological trauma. In psychogenic or functional retrograde amnesias, people can temporarily forget large portions of their pasts and/or identities after various kinds of disturbing events (for reviews, see [12,84,85]). Appropriate cueing and other factors often lead to recovery of memory in such cases, but it is unknown how such amnesias are related to forgetting of sexual abuse.

Patients with amnesic syndromes that result from damage to the hippocampus and related medial temporal lobe/diencephalic structures show a form of temporally graded retrograde loss, such that relatively recent memories are most affected and more remote experiences, particularly childhood memories, are less affected or

entirely unaffected [46]. This well known pattern, commonly referred to as Ribot's Law, is usually permanent and typically accompanied by significant anterograde amnesia for ongoing events; it therefore differs from the kind of amnesia purported to be operative in cases of recovered memories, which primarily involves childhood events. Other patients with damage extending into cortical association areas, the likely sites of long-term memory storage, show more extensive retrograde amnesias, sometimes covering virtually the entire personal past (see e.g. [86**,87–89]). Amnesia is usually permanent, although sudden recovery has been reported [86**]. Although the mechanisms of such recovery remain poorly understood, such extensive retrograde amnesias do not appear to provide a promising model for the kind of forgetting at issue in the recovered memories controversy.

Stress, encoding, and repression

In retrograde amnesia, experiences that were normally encoded and stored become inaccessible as a result of subsequent events. However, traumatic experiences may not be encoded and stored normally in the first place. Indeed, it has been suggested that traumatic experiences may be encoded differently from non-traumatic ones [90*,91].

The most common outcome of emotionally traumatic experiences is intrusive and repetitive recollection of the traumatic event (for recent reviews, see [12,91]). Laboratory studies of both rats and people suggest that enhanced memory for traumatic events is mediated by stress-related hormones such as epinephrine [92**]; other substances released by the brain in stressful situations, such as opioid peptides, have inhibitory effects on memory retention (for a review, see [93]; also see Cahill and McGaugh, in this issue, pp 237–242). Many of these influences on memory act via the 'final common pathway' of increasing or decreasing the release of the adrenergic neuromodulator norepinephrine in the amygdala. For example, epinephrine boosts release of norepinephrine, and opiates inhibit norepinephrine release [94].

In order for the effects of stress-related hormones to be relevant to the recovery of traumatic memories, these hormones must result in a trace that is available in memory, but rendered temporarily inaccessible by traumatic stress (as opposed to a very weak and permanently unavailable trace). This would be the case if neurochemicals that are released in response to extreme stress made memories state dependent. Kandel and Kandel [95] have speculated that the release of opioid peptides during a stressful experience might lead to a temporary inability to remember the trauma; later, another arousing experience could trigger the release of neurochemicals that (if accompanied by other appropriate retrieval cues) activate the formerly inaccessible memory. Evidence pertaining to state-dependent explanations of opiate effects on memory is equivocal (cf. [96,97]).

Another approach to explaining how memories for trauma might come to be 'available but inaccessible' focuses on the link between trauma and dissociation. Many clinicians and researchers have argued that traumatic experiences can produce a dissociative state in some individuals; in this state, mechanisms that normally lead to integrated perceptual experience and memory traces are disrupted, resulting in fragmentary engrams that are difficult to retrieve [2,90*,98–100]. Experimental analogues of dissociative states have been produced in human subjects using the NMDA receptor antagonist ketamine, which disrupts glutamatergic transmission and produces impairments in thinking, problem solving, and memory that resemble deficits observed after frontal lobe lesions [101,102*,103]. The frontal lobes play an important integrative role in memory, both by promoting elaborative encodings in which new experiences become integrated with pre-existing knowledge [104,105*], and by allowing effortful, strategic search of memory [64,70,72**,106*]. However, it is not known whether the effects of ketamine mimic the effects of stress during a traumatic experience, nor is there any evidence that disrupted frontal lobe functioning could produce dense amnesia for repeated traumatic experiences, as has been reported in some alleged cases of recovered memories.

Finally, we note recent observations by Ramachandran [107] that may bear on the issue of defensive repression. A patient who sustained a right parietal stroke and had lost the use of her left arm denied that it was paralyzed (the phenomenon of anosognosia; for reviews, see [108,109]). Both immediately and thirty minutes after irrigation of her left ear with cold water, the patient acknowledged that her left arm was paralyzed and had been for several days (similar effects have been reported in other patients, although the mechanism is poorly understood; see [110,111]). Eight hours later, when the effects of the cold water irrigation had worn off, she once again denied her paralysis. Asked about what the two doctors had done to her that morning, she remembered correctly that her ear had been irrigated. But the patient had apparently forgotten her earlier admission of paralysis, and she incorrectly recalled that she had stated earlier that her arm was fine. Ramachandran suggests that the patient had selectively 'repressed' the part of her memory that was inconsistent with her present beliefs. Although this is an intriguing idea, more data concerning what the patient did and did not remember from the irrigation episode are needed before it can be interpreted confidently. While observations from brain-damaged patients cannot speak directly to the question of whether non-brain damaged people are capable of a kind of repression that would create amnesia for overwhelming traumas, they can

provide clues concerning possibly relevant processes and mechanisms.

Concluding comments

The possible links we have considered between cognitive neuroscience research and recovered memories of childhood sexual abuse are no more than suggestive. One major limitation is that so few systematic studies have examined illusory memories of abuse or accurate recovered memories. Before we can confidently apply evidence and ideas from basic cognitive neuroscience, the phenomena that we are attempting to explain must be characterized more fully. Unless, and until, more reliable information becomes available, we urge caution when extrapolating from cognitive neuroscience to the complex and important issues at stake in debates about recovered memories.

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Analysis of positron emission tomography (PET) scan images was used to separate out two aspects of memory retrieval: the effort involved in attempting to retrieve a past event and the actual recollection of the event. Subjects studied some words several times with an elaborative encoding task (high recall condition), and studied other words just once with a perceptual encoding task (low recall condition). Subjects were later asked to remember high recall words and low recall words in response to three-letter word cues during separate PET scans. Hippocampal activation was observed in the high recall condition, in which subjects easily recollected many words, but not in the low recall condition. Conversely, prefrontal activations were observed in the low recall condition, where subjects made extensive efforts to remember words but recollected few of them, but not in the high recall condition.

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•   SM, Delaney RC, McCarthy G, Charney DS, Innis RB: **MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder.** *Am J Psychiatry* 1995, 152:973-981.
Used magnetic resonance imaging to measure the hippocampal volume of 26 Vietnam combat veterans with combat-related posttraumatic stress disorder (PTSD) and 22 comparison subjects without combat exposure. Veterans with PTSD had significantly reduced right hippocampal volume (reduction of 8%) and a non-significant reduction in left hippocampal volume (reduction of 3.8%). Veterans with PTSD also showed impaired immediate and delayed

verbal recall on the Wechsler Memory Scale (Revised). Developmental risk factors and/or trauma-related release of glucocorticoids, excitatory amino acids, serotonin, and other neurotransmitters and neuropeptides may have led to the reduced hippocampal volume.

75.   DeBellis MD, Chrousos GP, Dorn LD, Burke L, Helmers K, Kling
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      axis dysregulation in sexually abused girls.** J Clin Endocrinol
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Measured plasma adrenocorticotropic hormone (ACTH), and total and free cortisol responses to ovine corticotropin-releasing hormone (CRH) stimulation in a self-selected sample of sexually abused and control girls (aged 7–15 years). Relative to controls, sexually abused girls showed lower basal and net ovine CRH-stimulated plasma ACTH levels, and reduced total ACTH responses, but their total and free basal and CRH-stimulated plasma cortisol levels did not differ. It is hypothesized that chronic and/or intermittent endogenous CRH hypersecretion due to previous emotional and physical stress associated with the sexual trauma led to hypersensitivity of the adrenal cortices, which release cortisol in response to ACTH. Downregulation of ACTH release is viewed as a compensatory response aimed at maintaining normal levels of cortisol secretion. Insofar as excessive exposure to glucocorticoids can result in hippocampal damage, the hypothalamic–pituitary–adrenal axis dysfunction discussed here is relevant to explaining poor memory in sexual abuse victims.

76.   Sapolsky RM, Uno H, Rebert CS, Finch CE: **Hippocampal
      damage associated with prolonged glucocorticoid exposure
      in primates.** J Neurosci 1990, 10:2897–2902.

77.   Sapolsky RM: Stress, the Aging Brain, and the Mechanisms of
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78.   Keenan PA, Jacobson MW, Soleymani RM, Newcomer JW:
•   **Commonly used therapeutic doses of glucocorticoids impair
      explicit memory.** Ann NY Acad Sci 1995, 761:400–402.
Evaluated memory and cognitive performance of 25 patients receiving long-term prednisone treatment for systemic disease without central nervous system involvement, in comparison to 25 control medical patients matched for age, gender, IQ, and diagnosis. Patients receiving long-term prednisone treatment (duration of at least 1 yr) were significantly impaired at delayed paragraph recall and showed a trend toward impaired delayed recall of word lists (both tests of explicit memory), but were not impaired on an implicit task (word stem priming) or a vigilance task, thereby supporting the notion that glucocorticoid exposure may selectively impair hippocampal function.

79.   Newcomer JW, Craft S, Hershey T, Askins K, Bardgett ME:
•   **Glucocorticoid-induced impairment in declarative memory
      performance in adult humans.** J Neurosci 1994, 14:2047–2053.
Reports a double-blind, placebo-controlled study of the cognitive consequences of brief glucocorticoid exposure in normal adults. Subjects who received dexamethasone for four consecutive days (0.5 mg, 1.0 mg, 1.0 mg, 1.0 mg, respectively) showed impaired immediate and delayed recall of paragraphs at both 4 days and 11 days compared to placebo controls. Other cognitive measures were not affected by dexamethasone treatment, thereby arguing against a non-specific effect of glucocorticoids on arousal or attention.

80.   Wolkowitz OM, Reus VI, Weingartner H, Thompson K, Breier
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      corticosteroids.** Am J Psychiatry 1990, 147:1297–1303.

81.   Kuyken W, Brewin CR: **Autobiographical memory functioning in
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The authors examined autobiographical memory functioning in depressed women patients with, versus without, a reported history of childhood physical and/or sexual abuse. Patients were asked to retrieve a specific personal memory in response to cue words describing positive and negative emotions (e.g. happy and angry). Patients with a reported history of sexual abuse or a history of both physical and sexual abuse retrieved more 'overgeneral' (non-specific) autobiographical memories to both positive and negative cue words than did either patients without a history of abuse, or patients with a history of physical abuse alone. Overgeneral memories may arise from high levels of avoidance of abuse-related memories, or incorporation of events into general schematic representations of positive and negative experiences.

82.   Parks ED, Balon R: **Autobiographical memory for childhood
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Psychiatric outpatients with a reported history of childhood sexual and/or physical abuse, psychiatric outpatients without a history of abuse, and non-patient controls were tested using an autobiographical word cuing technique requiring the generation of a specific personal memory from before 16 years of age. Patients reporting childhood abuse more often failed to generate memories to affective cue words than did either control group, and retrieved fewer early memories than non-patient controls; the groups did not differ on non-autobiographical memory function as measured by the Wechsler Memory Scale (Revised).

83.   Williams JMG: **Autobiographical memory and emotional
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      Elsevier Science; 1989:209–231.

86.   Lucchelli F, Muggia S, Spinnler H: **The 'petites madeleines'
••   phenomenon in two amnesic patients: sudden recovery of
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Subjects watched a series of slides depicting a neutral story or an emotional story after receiving either placebo or the β-adrenergic receptor antagonist propranolol hydrochloride. Propranolol significantly impaired memory for the emotionally arousing story but did not impair memory for the neutral story. These results were not attributable to differences in emotional responsiveness or to non-specific attentional or sedative effects and suggest that enhanced memory for emotionally arousing events involves activation of the β-adrenergic system. This important study provides a bridge from the extensive animal literature on memory and adrenergic chemicals to human memory.

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