Trauma and thinking, *what recovery does and does not restore*

Twin, birth-cohort and treatment studies on whether trauma causes the cognitive difficulties seen in PTSD, and whether they lift when symptoms do.

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IN PLAIN WORDS

People with post-traumatic stress disorder, or PTSD, often score lower on tests of memory, attention and mental speed. A common assumption is that the trauma caused this. In identical twins, though, the brothers of veterans with PTSD who never went to war scored much like the veterans, and children followed from birth show that much of the gap after childhood victimisation was there before it, so the trauma cannot have caused that part. Some may still come from the disorder, because in soldiers tested before and after war, rising symptoms went with worsening recall. On whether recovery brings the thinking back, the evidence is thin. A few small studies find memory scores improve over a course of psychological treatment while other kinds of thinking mostly do not change, and no study we found tested reasoning or decisions after recovery.

A claim with two halves

A common assumption, which Gilbertson et al. (2006) set out to test, holds that a terrible experience harms the brain and that the harm shows up as poorer memory and concentration. A natural follow-on, and the second question this piece asks, is whether the thinking comes back once the distress is resolved. One question is about cause and the other about reversal, and the evidence on each has a different record.

The association behind both is consistent across the two meta-analyses opened here, though both found signs that publication bias may have inflated it. A meta-analysis is a study that pools the results of many earlier studies, and the one for adults by Scott et al. (2015), the first to cover a broad range of cognitive domains, pooled 60 studies that together included 1,779 people with PTSD, 1,446 people who had been through trauma without developing it, and 895 people with no trauma. Its results are expressed as standardised gaps, the difference between group averages divided by the spread of scores, so that 0.5 means half a standard deviation. The gaps varied widely from one domain to another. The largest were in verbal learning (0.62), in processing speed, meaning how fast simple mental operations are done (0.59), in attention and working memory, the ability to hold and use information for a few seconds (0.50), and in verbal memory (0.46). The picture in young people is similar. A meta-analysis of 55 studies with 8,070 participants aged 2 to 25 found that those exposed to trauma scored lower on working memory, on inhibition and on mental flexibility, with gaps between 0.44 and 0.49 (Op den Kelder et al., 2018). These executive functions are the control processes that hold goals in mind and suppress what is irrelevant.

In the Scott et al. (2015) data, smaller studies reported larger gaps, a pattern that selective publication can produce, and the authors estimated that a standard correction would shrink the overall gap by roughly 29%. The gap was also about twice as large in samples of people seeking treatment (0.65) as in community samples (0.30). Op den Kelder et al. (2018) likewise found signs of publication bias and say the true effects may be smaller. In their data, youth exposed to a single traumatic event did not differ from comparison youth on inhibition; the gaps

sat in groups exposed to violence or abuse and in adopted or fostered children.

Neither review can say which way cause runs. Much of the research has compared groups at a single point after the trauma, a limit that Vasterling et al. (2018) give as the reason causal inference has been hard, and Scott et al. (2015) state plainly that their data cannot separate a consequence of the disorder from a vulnerability that came first.

Where the damage story came from

The causal reading drew much of its force from animal work on stress hormones and the hippocampus, a structure in the temporal lobe needed for forming new memories of events. Sapolsky et al. (1990) implanted pellets of cortisol, the main stress hormone in primates, into one hippocampus in each of four vervet monkeys and an inert control pellet on the other side. A year later the cortisol side showed damage in every animal, sometimes severe, and the authors suggested that prolonged exposure might do the same in humans. We read this study as an abstract only. Its finding did not settle the question, since in a randomised study of 16 older pigtailed macaques, a year of high-dose oral cortisol raised cortisol in the fluid around the brain about threefold and produced no difference in hippocampal volume or neuron numbers compared with placebo (Leverenz et al., 1999). The authors left open subtler effects on how neurons work.

Imaging studies often found smaller hippocampi in PTSD, though they did not always agree. When a consortium analysed scans from 16 cohorts with one shared processing method, 1,868 people in all, 794 of them with PTSD, the hippocampus was smaller in current PTSD than in trauma-exposed people without it, with a standardised gap of 0.17 (Logue et al., 2018). That is a small difference, and the authors write that the cause remains an open question.

On the second half of the claim, that the change reverses, the closest human evidence among the studies opened here concerns stress rather than trauma. Liston et al. (2009) scanned 20 healthy young adults after a month of preparing for a major examination and compared them with 20 matched controls. The stressed group was worse at shifting attention between rules, and coupling was weaker within a network linking frontal and parietal regions that supports such shifts. A month after the examination the differences were no longer significant. Its participants were healthy medical students under a stress with a known end date. The authors considered that people with weaker prefrontal function to begin with might simply report more stress, and used the retest after the examination to address that. The study shows that ordinary stress can impair attention and then lift, which says little about how the cognitive profile of PTSD behaves.

What twins and birth cohorts did to the first half

Comparing identical twins when only one of them was exposed can separate a cause from a prior vulnerability, and so can following children from birth, which lets cognition be measured before any trauma happens.

Among Vietnam-era veterans, Gilbertson et al. (2002) scanned 17 pairs of identical twins in which the brother who saw combat developed PTSD and 23 pairs in which he did not; no unexposed brother had PTSD. In the pairs where the veteran had severe PTSD, both twins had smaller hippocampi than the members of pairs without PTSD, and the brothers in each pair did not differ from each other. The hippocampal volume of the brother who never went to war tracked the symptom severity of the brother who did. The authors read this as a familial risk factor and concluded that calling the smaller volume atrophy may be a misnomer. A companion study tested cognition in the same design and found that the unexposed brothers of veterans with PTSD largely performed like their brothers, and below the other pairs (Gilbertson et al., 2006; abstract only). In the 2002 study the sample was small and all male, and the PTSD pairs were recruited through a different route from the others (the authors argue this is unlikely to explain the result); with no non-identical twins it could not tell inherited factors from a shared upbringing.

The birth cohorts have larger and more general samples, and they ask related questions that are not quite the same one. In the Dunedin cohort of 1,037 people born in New Zealand in 1972 and 1973, among those who had experienced trauma, each standard deviation higher on an IQ test at age five went with 29% lower odds of developing PTSD (95% confidence interval 5 to 47%), and low IQ at five still predicted PTSD from events that happened between ages 26 and 32 (Koenen et al., 2007). Danese et al. (2017) asked whether victimisation in childhood, rather than PTSD itself, lowers later cognition, using 2,232 British children followed to age 18 and the same Dunedin cohort followed to 38. Victimised children did score lower, across intelligence, executive function, processing speed and memory.

Most of that gap disappeared once the analysis allowed for each child's IQ before the victimisation period and for the family's socioeconomic status. In the British sample the association with IQ at 18 went from a standardised coefficient of 0.12 to zero, and in Dunedin the association with IQ at 38 went from 0.14 to 0.04, no longer significant. Within twin pairs raised together, the twin who was victimised generally did not score lower than the one who was not, which points to family-level genetic and environmental factors. Danese et al. (2017) note that one residual association remained, so a small causal effect cannot be ruled out, and that their data cover victimisation between ages 3 and 12 and leave out more extreme circumstances such as institutional care.

The adult literature has the same weakness from the other side. In the Scott et al. (2015) meta-analysis, 35 of the 60 studies did not match the PTSD and healthy groups on estimated IQ before the trauma, and the larger the IQ difference between groups, the larger the reported cognitive gap.

These results describe group averages and probabilities. They do not mean that the difficulties people report are imagined, since the deficits were measured, and they do not assign any responsibility for what happened to anyone. What they say is that in the studies able to check, much of the gap seen after trauma was already there before it.

The strongest case for a real effect

Among the studies opened here, the clearest evidence that the disorder itself may cost something comes from soldiers tested before they were exposed. Vasterling et al. (2018) assessed 315 United States Army soldiers before deployment to Iraq, again after it, and again about 7.6 years later, with models that adjusted for each person's earlier score on the same measure, for combat severity and for several background characteristics. Better visual learning before deployment predicted milder PTSD symptoms afterwards, which is the vulnerability direction, while rising symptoms over each interval predicted poorer verbal or visual recall at the end of it, which is the consequence direction. The authors describe the relationship as possibly bidirectional, with a modest protective role for memory and a gradual cost of worsening symptoms. The study had few women and no non-deployed comparison group at the long-term assessment, and conditions such as depression or chronic pain were not measured.

Scott et al. (2015) reach a similar synthesis from the studies available to them, in which weaknesses that exist before trauma may raise the risk of PTSD and may then worsen as the disorder develops. This is the strongest version of the opposing view, and it fits the twin and cohort results. Our reading of the evidence is that a large part of the gap is prior vulnerability and a smaller part, seen mainly in memory, may follow from the disorder.

What the treatment studies say about the second half

If some of the deficit follows from the disorder, that part might lift when symptoms ease, and few studies have looked. Nijdam et al. (2018) gave cognitive tests before and after a 17-week randomised trial comparing two trauma-focused psychotherapies for PTSD. Of 140 patients randomised, 88 completed both assessments. Verbal memory, processing speed and executive function improved, with effects ranging from small to medium (0.16 to 0.68); larger drops in symptoms went with better scores at the end, though not with larger gains. The authors note that the gains were larger than practice effects reported elsewhere for repeated testing on the same instruments. They also state the main limit, which is that for ethical reasons the trial had no untreated group, so no causal claim about the therapy can be made, and people who finished treatment were overrepresented. Whether the gains carry into daily life was left for future work.

The one systematic review we found included nine studies, the Nijdam trial among them, five randomised and four not, with 285 participants in the randomised trials and 180 in the others (Susanty et al., 2022). The data could not be pooled, because no outcome was measured the same way against an inactive control in at least three studies. Effects on memory were medium to large, though some were not statistically significant, and effects on attention, executive function and processing speed were mostly not significant. Six studies were rated at high risk of bias for blinding of participants, outcome measures varied widely, and some trials compared against another active treatment. The reviewers conclude that psychological treatment for PTSD leaves most cognitive functions unchanged, memory excepted.

So the second half of the claim is partly supported for memory, in small studies with weak controls, and not established for attention, speed or executive function. None of the studies we opened tested reasoning, judgement or decision-making after symptoms resolved.

A study that could settle it

This section is our argument about design, and no study in the sources above meets it. A decisive test would measure cognition before trauma, as the deployment and birth-cohort studies did, so that each person serves as their own baseline. It would include a comparison group exposed to the same trauma who did not develop PTSD, which separates the effect of the event from the effect of the disorder. And it would assign people at random to treatment now or after a delay, with cognitive tests as a planned outcome, alternate test forms, and an untreated period long enough to estimate practice effects and natural change. Outcomes would include everyday functioning and reasoning tasks alongside memory tests, and follow-up would continue after symptoms remit. Such a study would also need enough women and enough non-military trauma to say whom the results apply to.

What is not yet known

- Whether the memory gains reported after treatment for PTSD exceed what an untreated group with the same symptoms would show over the same period. The review we found had only one study with a waiting-list group for memory, too few to pool.
- Whether reasoning, planning and decision-making change as PTSD symptoms resolve. The review we found grouped its outcomes as memory, attention, information processing and executive function, and only one included trial reported a problem-solving test, against another active treatment.
- How much of the familial vulnerability in the twin studies is genetic and how much comes from a shared upbringing, which needs non-identical twins in the same design.
- Whether the consequence direction found in soldiers by Vasterling et al. (2018) holds for civilians, for women, and for trauma in childhood.
- Whether victimisation before age three, or more extreme early adversity than the cohorts captured, has the causal effect on cognition that Danese et al. (2017) did not find in children aged 3 to 12.

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