



## Indirect exposure to trauma

### Introduction

For a person to be diagnosed with PTSD, at least one stressor is required. Stressors as determined by the latest version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) include being exposed to threatened death, actual or threatened serious injury, or actual or threatened sexual violence. Examples are direct exposure, witnessing the trauma, or learning that a relative or close friend was exposed to a trauma. Stressors can also be encountered in the course of professional duties.

This summary table presents the evidence for risk of PTSD in people indirectly exposed to trauma.

### Method

We have included only systematic reviews (systematic literature search, detailed methodology with inclusion/exclusion criteria) published in full text, in English, from the year 2010 that report results separately for people with PTSD. Reviews were identified by searching the databases MEDLINE, EMBASE, and PsycINFO. When multiple copies of reviews were found, only the most recent version was included. We prioritised reviews with pooled data for inclusion.

Review reporting assessment was guided by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist that describes a preferred way to present a meta-analysis<sup>1</sup>. Reviews with less than 50% of items checked have been excluded from the library. Note that early reviews may have been guided by less stringent reporting checklists than the PRISMA, and that some reviews may have been limited by journal guidelines.

Evidence was graded using the Grading of Recommendations Assessment, Development and Evaluation ([GRADE](#)) Working Group approach where high quality evidence such as that gained from randomised controlled trials

(RCTs) may be downgraded to moderate or low if review and study quality is limited, if there is inconsistency in results, indirect comparisons, imprecise or sparse data and high probability of reporting bias. It may also be downgraded if risks associated with the intervention or other matter under review are high. Conversely, low quality evidence such as that gained from observational studies may be upgraded if effect sizes are large or if there is a dose dependent response. We have also taken into account sample size and whether results are consistent, precise and direct with low associated risks (see end of table for an explanation of these terms)<sup>2</sup>. The resulting table represents an objective summary of the available evidence, although the conclusions are solely the opinion of staff of NeuRA (Neuroscience Research Australia).

### Results

We found six systematic reviews that met our inclusion criteria<sup>3-8</sup>.

- Moderate quality evidence finds the prevalence of PTSD in direct victims of terrorist attacks after one year is between 33% and 39%, while indirect victims showed lower prevalence rates (community = 4%, rescue teams = 5-6%, family and friends = 3-13.8%).
- Moderate to high quality evidence found small associations between increased PTSD symptoms and more caseload volume and frequency, and more personal trauma history in professionals exposed to secondary workplace trauma. Lower PTSD symptoms were associated with more social support, work support, trauma training, experience, and older age.
- Moderate to high quality evidence found a medium-sized effect of increased PTSD symptoms in health workers exposed to critical incidents (secondary trauma) compared to health workers not exposed to critical incidents. The effect was larger after



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4 weeks post-incident than within 4 weeks post-incident.

- Moderate to high quality evidence found a small association between increased exposure to televised mass trauma and increased PTSD symptoms.
- Moderate quality evidence found a small effect of increased rates of PTSD in people exposed to longer vs. shorter COVID-19 media reporting.
- Moderate to high quality evidence found a large effect of more PTSD symptoms in parents of chronically ill children than in parents of healthy children. Rates were highest in parents of children with epilepsy or diabetes, in mothers, in parents of children with more illness severity, longer treatment duration and intensity and in parents of children with PTSD symptoms. Rates were lowest in parents of children with longer illness duration, longer time since active treatment and in those with more social support.



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de Boer J, Lok A, Van't Verlaat E, Duivenvoorden HJ, Bakker AB, Smit BJ

**Work-related critical incidents in hospital-based health care providers and the risk of post-traumatic stress symptoms, anxiety, and depression: a meta-analysis**

Social Science and Medicine 2011; 73: 316-26

[View review abstract online](#)

<b>Comparison</b>	PTSD after exposure to work-related hospital critical incidents. Most critical incidents comprised treating SARS patients. Others concerned treating victims of terror or treating patients in critical care units.
<b>Summary of evidence</b>	Moderate to high quality evidence (large sample, inconsistent, precise, direct) found a medium-sized effect of increased PTSD symptoms in health workers exposed to critical incidents compared to health workers not exposed to critical incidents. The effect was larger after 4 weeks post-incident than before 4 weeks post-incident.
<b>Secondary workplace trauma</b>	
<p><i>A medium-sized effect showed increased PTSD symptoms in people exposed to critical incidents;</i>                      11 studies, N = 3,866, SMD = 0.32, 95%CI 0.12 to 0.50, <math>p &lt; 0.05</math>, <math>I^2 = 82\%</math></p> <p>The pooled effect size was smaller in the subgroup analysis of studies measuring PTSD during the first four weeks of the incident compared to studies measuring PTSD between four and 26 months after the incident (SMD = 0.20 vs. 0.52).</p>	
<b>Consistency in results<sup>†</sup></b>	Inconsistent
<b>Precision in results<sup>§</sup></b>	Precise
<b>Directness of results<sup>  </sup></b>	Direct

Hensel JM, Ruiz C, Finney C, Dewa CS

**Meta-analysis of risk factors for secondary traumatic stress in therapeutic work with trauma victims**

Journal of Traumatic Stress 2015; 28: 83-91

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<b>Comparison</b>	<b>Prevalence of PTSD in professionals indirectly exposed to trauma through their therapeutic work with trauma victims.</b>
<b>Summary of evidence</b>	<b>Moderate to high quality evidence (large samples, some inconsistency, precise, direct) found small associations between increased PTSD symptoms and more caseload volume and frequency, and more personal trauma history in people exposed to workplace secondary trauma. Lower PTSD symptoms were associated with more social support, work support, trauma training, experience, and older age.</b>
<b>Secondary workplace trauma</b>	
<p><i>Small associations were found between increased PTSD symptoms and more;</i></p> <p>Caseload volume: 5 studies, N = 779, <math>r = 0.16</math>, 95%CI 0.07 to 0.26, <math>p &lt; 0.001</math>, <math>I^2 = 40\%</math>  Caseload frequency: 15 studies, N = 1,462, <math>r = 0.12</math>, 95%CI 0.02 to 0.22, <math>p &lt; 0.05</math>, <math>I^2 = 70\%</math>  Caseload ratio: 4 studies, N = 1,138, <math>r = 0.19</math>, 95%CI 0.14 to 0.25, <math>p &lt; 0.001</math>, <math>I^2 = 0\%</math>  Personal trauma history: 11 studies, N = 1,972, <math>r = 0.19</math>, 95%CI 0.14 to 0.24, <math>p &lt; 0.001</math>, <math>I^2 = 29\%</math></p> <p>Volume = number of traumatised clients encountered  Frequency = frequency of contact with traumatised clients  Ratio = proportion of caseload comprising time spent with trauma clients</p> <p><i>Small associations were found between decreased PTSD symptoms and more;</i></p> <p>Social support: 5 studies, N = 1,135, <math>r = -0.26</math>, 95%CI -0.35 to -0.17, <math>p &lt; 0.001</math>, <math>I^2 = 61\%</math>  Work support: 5 studies, N = 1,145, <math>r = -0.17</math>, 95%CI -0.32 to -0.02, <math>p &lt; 0.05</math>, <math>I^2 = 86\%</math>  Trauma training: 6 studies, N = 2,395, <math>r = -0.05</math>, 95%CI -0.10 to -0.00, <math>p &lt; 0.05</math>, <math>I^2 = 27\%</math>  Experience: 16 studies, N = 2,429, <math>r = -0.07</math>, 95%CI -0.12 to -0.01, <math>p &lt; 0.01</math>, <math>I^2 = 41\%</math>  Older age: 15 studies, N = 2,491, <math>r = -0.05</math>, 95%CI -0.10 to -0.00, <math>p &lt; 0.05</math>, <math>I^2 = 27\%</math></p> <p>There were no associations with emotional involvement, ethnicity, sex, posttraumatic growth, or supervision.</p>	
<b>Consistency in results</b>	Some inconsistency
<b>Precision in results</b>	Precise
<b>Directness of results</b>	Direct

*Paz Garcia-Vera M, Sanz J, Gutierrez S*

**A Systematic Review of the Literature on Posttraumatic Stress Disorder in**



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**Victims of Terrorist Attacks**

Psychological Reports 2016; 119: 328-59

[View review abstract online](#)

<b>Comparison</b>	<b>Prevalence of PTSD in direct and indirect victims of terrorist attacks.</b>
<b>Summary of evidence</b>	<b>Moderate quality evidence (large sample, appears inconsistent and imprecise, direct) finds the prevalence of PTSD in direct victims of terrorist attacks after one year is between 33% and 39%. Indirect victims showed lower prevalence rates (community = 4%, rescue teams = 5-6%, family and friends = 3-13.8%).</b>
<b>Direct and indirect exposure to terrorist attacks</b>	
<p>35 studies, N &gt;20,000</p> <p>Direct victims: 1-year post-attack prevalence = 33% to 39%</p> <p>Indirect victims (community): 1-year post-attack prevalence = 4%</p> <p>Indirect victims (rescue teams): 1-year post-attack prevalence = 5% to 6%</p> <p>Indirect victims (family and friends): 1-year post-attack prevalence = 3% to 13.8%</p>	
<b>Consistency in results</b>	Appears inconsistent
<b>Precision in results</b>	Appears imprecise
<b>Directness of results</b>	Direct

*Pfefferbaum B, Nitiema P, Newman E*

**Is Viewing Mass Trauma Television Coverage Associated With Trauma Reactions in Adults and Youth? A Meta-Analytic Review**

Journal of Traumatic Stress 2019; 32: 175-85

[View review abstract online](#)

<b>Comparison</b>	<b>PTSD after viewing mass trauma on television.</b>
<b>Summary of evidence</b>	<b>Moderate to high quality evidence (large sample, inconsistent, precise, direct) found a small association between increased exposure to televised mass trauma and increased PTSD symptoms.</b>



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<b>Exposure to mass trauma on television</b>	
<p><i>Small association between increased exposure to televised mass trauma and increased PTSD symptoms;</i></p> <p>43 studies, N = 31,162, <math>r = 0.17</math>, 95%CI 0.13 to 0.22, <math>p &lt; 0.001</math>, <math>I^2 = 91\%</math></p>	
<b>Consistency in results</b>	Inconsistent
<b>Precision in results</b>	Precise
<b>Directness of results</b>	Direct

<p><i>Pinquart M</i></p> <p><b>Posttraumatic Stress Symptoms and Disorders in Parents of Children and Adolescents With Chronic Physical Illnesses: A Meta-Analysis</b></p> <p><b>Journal of Traumatic Stress 2019; 32: 88-96</b></p> <p><a href="#">View review abstract online</a></p>	
<b>Comparison</b>	<b>PTSD in parents of children with a chronic physical illness (cancer, burns, heart disease, diabetes, epilepsy, and asthma) vs. community norms or parents of healthy children.</b>
<b>Summary of evidence</b>	<b>Moderate to high quality evidence (large sample, consistent, imprecise, direct) found a large effect of more PTSD symptoms in parents of chronically ill children than in parents of healthy children. Rates were highest in parents of children with epilepsy or diabetes, in mothers, in parents of children with more illness severity, longer treatment duration and intensity and in parents of children with PTSD symptoms. Rates were lowest in parents of children with longer illness duration, longer time since active treatment and in those with more social support.</b>
<b>Parents of children with chronic physical illness</b>	
<p>184 studies, N = 30,068</p> <p><i>A large effect of more PTSD symptoms in parents of chronically ill children than in parents of healthy children;</i></p> <p>OR = 7.12, 95%CI 6.01 to 8.44, <math>p &lt; 0.001</math>, <math>Qp = 0.151</math></p> <p>Parental PTSD symptoms were most prevalent among parents of children with epilepsy (<math>g = 1.25</math>), and diabetes (<math>g = 1.16</math>), and were positively associated with being the mother (<math>r = 0.19</math>), increased illness severity (<math>r = 0.18</math>), more treatment duration/intensity (<math>r = 0.21</math>), and PTSS in the child (<math>r =</math></p>	





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0.34).	
Longer illness duration ( $r = -0.19$ ), longer time since active treatment ( $r = -0.10$ ), and better social resources ( $r = -0.17$ to $-0.07$ ) were associated with lower parental PTSD symptoms.	
<b>Consistency in results</b>	Consistent
<b>Precision in results</b>	Imprecise
<b>Directness of results</b>	Direct

<p><i>Wang Y, Kala MP, Jafar TH</i></p> <p><b>Factors associated with psychological distress during the coronavirus disease 2019 (COVID-19) pandemic on the predominantly general population: A systematic review and meta-analysis</b></p> <p>PLOS ONE 2021; 15: e0244630</p> <p><a href="#">View review abstract online</a></p>	
<b>Comparison</b>	<b>PTSD in people exposed to media reporting of COVID-19.</b>
<b>Summary of evidence</b>	<b>Moderate quality evidence (large samples, consistent, some imprecision, direct) found a small effect of increased rates of PTSD in people exposed to longer vs. shorter COVID-19 media reporting.</b>
<b>COVID-19</b>	
<p><i>Small effects showed higher risk of PTSD during the COVID-19 pandemic was associated with;</i></p> <p>Longer vs. shorter media exposure: 3 studies, N = 5,267, OR = 1.48, 95%CI 1.23 to 1.78, I<sup>2</sup> = 0%</p>	
<b>Consistency in results</b>	Consistent
<b>Precision in results</b>	Some imprecision
<b>Directness of results</b>	Direct

**Explanation of acronyms**

CI = confidence interval, I<sup>2</sup> = the percentage of the variability in effect estimates that is due to heterogeneity rather than sampling error (chance), N = number of participants, OR = odds ratio,  $p$  = statistical probability of obtaining that result, Q = test of heterogeneity,  $r$  = correlation coefficient, SMD = standardised mean difference, vs. = versus



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### Explanation of technical terms

\* Bias has the potential to affect reviews of both RCT and observational studies. Forms of bias include; reporting bias – selective reporting of results; publication bias - trials that are not formally published tend to show less effect than published trials, further if there are statistically significant differences between groups in a trial, these trial results tend to get published before those of trials without significant differences; language bias – only including English language reports; funding bias - source of funding for the primary research with selective reporting of results within primary studies; outcome variable selection bias; database bias - including reports from some databases and not others; citation bias - preferential citation of authors. Trials can also be subject to bias when evaluators are not blind to treatment condition and selection bias of participants if trial samples are small<sup>9</sup>.

† Different effect measures are reported by different reviews.

Prevalence refers to how many existing cases there are at a particular point in time. Incidence refers to how many new cases there are per population in a specified time period. Incidence is usually reported as the number of new cases per 100,000 people per year. Alternatively some studies present the number of new cases that have accumulated over several years against a person-years denominator. This denominator is the sum of individual units of time that the persons in the population are at risk of becoming a case. It takes into account the size of the underlying population sample and its age structure over the duration of observation.

Reliability and validity refers to how accurate the instrument is. Sensitivity is the proportion of actual positives that are correctly identified

(100% sensitivity = correct identification of all actual positives) and specificity is the proportion of negatives that are correctly identified (100% specificity = not identifying anyone as positive if they are truly not).

Weighted mean difference scores refer to mean differences between treatment and comparison groups after treatment (or occasionally pre to post treatment) and in a randomised trial there is an assumption that both groups are comparable on this measure prior to treatment. Standardised mean differences are divided by the pooled standard deviation (or the standard deviation of one group when groups are homogenous) that allows results from different scales to be combined and compared. Each study's mean difference is then given a weighting depending on the size of the sample and the variability in the data. Less than 0.4 represents a small effect, around 0.5 a medium effect, and over 0.8 represents a large effect<sup>9</sup>.

Odds ratio (OR) or relative risk (RR) refers to the probability of a reduction ( $< 1$ ) or an increase ( $> 1$ ) in a particular outcome in a treatment group, or a group exposed to a risk factor, relative to the comparison group. For example, a RR of 0.75 translates to a reduction in risk of an outcome of 25% relative to those not receiving the treatment or not exposed to the risk factor. Conversely, a RR of 1.25 translates to an increased risk of 25% relative to those not receiving treatment or not having been exposed to a risk factor. A RR or OR of 1.00 means there is no difference between groups. A medium effect is considered if  $RR > 2$  or  $< 0.5$  and a large effect if  $RR > 5$  or  $< 0.2$ <sup>10</sup>. InOR stands for logarithmic OR where a InOR of 0 shows no difference between groups. Hazard ratios measure the effect of an explanatory variable on the hazard or risk of an event.

Correlation coefficients (eg,  $r$ ) indicate the strength of association or relationship





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between variables. They can provide an indirect indication of prediction, but do not confirm causality due to possible and often unforeseen confounding variables. An  $r$  of 0.10 represents a weak association, 0.25 a medium association and 0.40 and over represents a strong association. Unstandardised ( $b$ ) regression coefficients indicate the average change in the dependent variable associated with a 1 unit change in the independent variable, statistically controlling for the other independent variables. Standardised regression coefficients represent the change being in units of standard deviations to allow comparison across different scales.

‡ Inconsistency refers to differing estimates of effect across studies (i.e. heterogeneity or variability in results) that is not explained by subgroup analyses and therefore reduces confidence in the effect estimate.  $I^2$  is the percentage of the variability in effect estimates that is due to heterogeneity rather than sampling error (chance) - 0% to 40%: heterogeneity might not be important, 30% to 60%: may represent moderate heterogeneity, 50% to 90%: may represent considerable heterogeneity and over this is considerable heterogeneity.  $I^2$  can be calculated from  $Q$  (chi-square) for the test of heterogeneity with the following formula<sup>9</sup>;

$$I^2 = \left( \frac{Q - df}{Q} \right) \times 100\%$$

§ Imprecision refers to wide confidence intervals indicating a lack of confidence in the effect estimate. Based on GRADE recommendations, a result for continuous data (standardised mean differences, not weighted mean differences) is considered imprecise if the upper or lower confidence

limit crosses an effect size of 0.5 in either direction, and for binary and correlation data, an effect size of 0.25. GRADE also recommends downgrading the evidence when sample size is smaller than 300 (for binary data) and 400 (for continuous data), although for some topics, these criteria should be relaxed<sup>11</sup>.

|| Indirectness of comparison occurs when a comparison of intervention A versus B is not available but A was compared with C and B was compared with C that allows indirect comparisons of the magnitude of effect of A versus B. Indirectness of population, comparator and/or outcome can also occur when the available evidence regarding a particular population, intervention, comparator, or outcome is not available and is therefore inferred from available evidence. These inferred treatment effect sizes are of lower quality than those gained from head-to-head comparisons of A and B.



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### References

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