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WHO SUFFERS FROM POST-TRAUMATIC STRESS DISORDER?

Introduction

Generally it is felt that post-traumatic stress disorder (PTSD) develops following psychologically distressing events which lie outside common experience, and these events are distressing to most people (Brewer 2001).

One way of viewing PTSD is in terms of layers of sufferers:

- Primary victims - survivors of the traumatic event;
- Secondary victims - families and friends of the survivors or non-survivors (ie: bereaved);
- Tertiary victims - witnesses but not involved in the event (ie: not physically injured);
- Rescuers - including emergency services personnel;
- Carers - medical staff who see the injuries and may not have been at the scene of the event;
- Peripheral victims - individuals indirectly affected by the event; eg: those who should have gone on the plane that crashes but did not for some reason, or individuals whose workplace was affected by the event.

Kinchin (1994) felt that the risk of developing PTSD was actually higher for secondary victims than the other groups even primary victims.

Diagnosis of PTSD is based upon the presence of certain symptoms. This has varied between editions of DSM. DSM-IV (APA 1994) listed six criteria for diagnosis of PTSD (table 1).

There are three variations of PTSD:

i) Acute - if the symptoms persist less than three months;

ii) Chronic - if the symptoms persist for longer than three months;

iii) Delayed onset - if the symptoms do not appear until six months after the trauma.

Acute Stress Disorder is a different diagnosis for symptoms that occur within four weeks of the event, but last only a short period (between two days to four weeks) (Brewer 2001).

CRITERION A	The person experiences, witnesses, or "was confronted with" a traumatic event that threatened death or serious injury, and the response involved "intense fear, helplessness, or horror"
CRITERION B	The traumatic event is "persistently re-experienced" as: - "recurrent and intrinsic distressing recollections - "recurrent distressing dreams" - "acting or feeling as if the traumatic event were recurring" - psychological distress at cues to the event - physiological responses to the cues
CRITERION C	Persistent avoidance of stimuli associated with trauma as shown by at least three of the following: - efforts to avoid thinking or talking about trauma - efforts to avoid people or places related to trauma - inability to recall important aspects of trauma - "markedly diminished interest" in normal activities - feeling of detachment from others - restricted range of emotions - "sense of foreshortened future"
CRITERION D	Persistent symptoms of increased arousal as shown by at least two of the following: - difficulty sleeping - irritability - concentration problems - hyper-vigilance - increased startle reaction
CRITERION E	Symptoms last more than one month
CRITERION F	Symptoms cause "clinically significant distress or impairment" in the person's life

Table 1 - DSM-IV criteria for PTSD.

Controversies of PTSD

PTSD has had its controversies (and still does) in relation to the introduction formally in 1980 in DSM-III (APA 1980). Scott (1990) felt that the issue was not about war trauma and the negative consequences of leaving that untreated, it was about whether in fact a special condition called PTSD existed "that had yet to be discovered".

Bracken et al (1995) argued that PTSD endorses implicit Western value systems, and is not applicable to non-Western societies:

> Our argument is not that concepts of post traumatic stress disorder...be abandoned, but rather that their limitations be recognised, and their use in non-Western situations approached with caution (p1081).

Summerfield (2001) was even more critical of PTSD as the legacy of the Vietnam war and the blame for the war faced by returning US soldiers. PTSD "legitimised their 'victimhood', gave them moral exculpation and guaranteed them a disability pension because diagnosis could be attested to by a doctor" (p95). Furthermore, "the misery and horror of war is reduced to a technical issue tailored to Western approaches to mental health" (p96).

Interestingly, Banyard (2000) noted how the Western bias on the portrayal of the effect of the Vietnam war ignores the consequences for the Vietnamese (who experienced 95% of the causalities). It is portrayed as a tragedy for the USA.

Another of the controversies relates to the existence of the symptoms of PTSD before PTSD formally existed. In other words, it has always existed, but went unrecognised (Gersons and Carlier 1992). It is a timeless condition: a "relatively common human problem... known for many hundreds of years, although under different names" (Trimble 1985 p5)

But, for Bracken (2001), PTSD is the product of "trauma and culture acting together, and thus it is not timeless".

Jones et al (2002) made use of detailed medical and military reports to compare the experiences of UK soldiers since the 1850s. They concentrated upon the core symptom of flashbacks. There tended to be more reports of them in recent years (eg: 1991 Persian Gulf War), and few in the Boer and First World wars.

Jones et al concluded that:

> There is no single way for human beings to respond to the terrifying events of war, and the concept of a "universal trauma reaction" appears flawed (p163).

The Growth of PTSD

Two main patterns have appeared in recent years with the increasing general knowledge of PTSD in societies: (i) an increase of the application to PTSD to more common events, and (ii) the increase of the peripheral victims category.

1. An increase in the application of PTSD to events that are more common (eg: car crashes) rather than just the uncommon ones (eg: plane crashes).

As the events that produce PTSD increase, it moves away from the original key idea which was life-threatening events that lie outside common experience. It is possible that long-term less traumatic events, like severe illness, can lead to PTSD (eg: Kelly et al 1998 and HIV; Cordova et al 2000 and breast cancer).

The widening of events to include the possibility of PTSD as a consequence has arrived at "ordinary" life events (eg: divorce). In other words, PTSD without the trauma (Scott and Stradling 1994).

Mol et al (2005) were interested as to whether such events generated PTSD symptoms. They used a general population sample from the province of Limburg in Holland. Of the respondents, 299 were classified as having experienced a traumatic event, and 533 only life events in the last thirty years.

Mean scores on the Post-Traumatic Stress Symptom Scale - Self-Report Version (PSS-SR) (Foa et al 1993) were higher for the "life events only" group, which the authors admitted was "a rather unexpected finding". Mol et al were unsure whether these findings challenged the validity of the diagnosis of PTSD, or were genuine evidence of PTSD after "ordinary" life events.

While, on the one hand, PTSD is increasingly diagnosed for all kinds of events, there are those who are arguing that the prevalence is low, for example, in soldiers. Mark Turner, of the army's community mental health department at the Duchess of Kent's Barracks, and colleagues (2005) argued that UK troops in Irag were suffering from "less dramatic symptoms" than PTSD. In fact, PTSD is never used, but "combat stress reaction" instead.

Turner et al (2005) analysed the records of 117

patients arriving at the military psychiatric hospital at Catterick Garrison, West Yorkshire, from Iraq during three periods in 2003: January-March (beginning of the war) (26% of evacuations), March-May ("formal" end of war) (44%), and May-October (post-war period) (31%). Most of the patients were support troops including reservists.

The authors clarified their position:

> Combat troops fighting in the low-intensity conflicts that characterise modern warfare, and support troops in any conflict, are not often exposed to the kind of acute, overwhelming stress that is necessary for the symptoms of combat stress reaction to develop. Instead, these groups typically present with a range of less dramatic symptoms that are characteristic of adjustment disorders and almost invariably include low mood. They are, incidentally, not dissimilar to those seen in military personnel during peacetime (p447).

I am not sure what this says about soldiers in peacetime.

Turner et al were also concerned that "pre-existing neurotic difficulties become misinterpreted as post traumatic stress disorder".

If there are vested interests that are "pathologizing everyday life" (Kutchins and Kirk 1997), then there are cases of down-playing reality. There is always the issue that military personnel are unlikely to admit to psychiatric symptoms because of the "macho culture" of the military. But to down-play the effects of armed conflicts is to encourage the belief that war is normal. Rather than individuals naturally being disturbed by such situations, the experiences of war are brought into the range of everyday experience. This allows those vested interests that want armed conflicts and military situations to continue unopposed.

If war is a normal part of life, then people will not oppose going to start another one, and attempts will not be made to seek non-military conflict resolutions.

The US writers, Burbach and Tarbell (2004) were blunt in their views of the kind of vested interests here:

> The Bush administration does not care how many civilians die in Iraq; in fact it connives with the established media to keep the US public ignorant of the true human toll of the war. In Baghdad, the Pentagon refuses to count the number of civilian dead (p3).

2. The category of peripheral victims has also increased with media coverage of traumatic events.

This is most evident with the television pictures of the terrorist attacks in New York in September 2001, which were witnessed live or seen in replays throughout the world. Here the line is blurred between witnesses (who are usually seen as close to the event; ie: witness it in the flesh) and the peripheral victims (who witness the event live via television).

Another example relates to the "Asian Tsunami" in December 2004. Many television viewers of the aftermath were affected because they had been to the areas hit for holidays in the past and/or were considering going. In other words, actually knowing the spot where the event took place.

Whatever the number of people affected by a traumatic event and those diagnosed with PTSD, there are many individuals who do not suffer. In fact, in many cases, it is the majority. The key is to look for the risks (ie: the factors) that distinguish sufferers from non-sufferers.

Problems of Researching PTSD

A lot of research has looked at the effect of the events in the US on 11th September 2001. For example, Schuster et al (2001) obtained data between 14-16th September (ie: 3-5 days after). Of the sample, 44% reported at least one symptom of PTSD.

While Schlenger et al (2002) studied 2273 adults in New York and Washington one to two months after the event using internet-based questionnaires. The level of PTSD was higher in these two cities compared to other US cities, and certain characteristics emerged around who had PTSD (table 2). The amount of television viewing and images seen is an interesting variable.

North and Pfefferbaum (2002) addressed the methodological issues of the Schlenger et al study, and they highlighted relevant concerns for any study of PTSD.

1. Timing of the study

PTSD cannot be diagnosed for at least one month, and this limits the beginning of the study.

	CITY	NEW YORK *	11.2
		WASHINGTON	2.7
		OTHER US CITIES	3.6
		WHOLE USA	4.3
FAMILY/FRIENDS/CO-WORKER INJURED/KILLED			13.2
		NOT	3.9
	SELF OR CLOSE FAMILY IN MILITARY		6.7
		NOT	3.5
TELEVISION VIEWING PER DAY (hrs)		less 4	0.8 *
		12+	10.1
TELEVISION CONTENT INDEX (different types of graphic events viewed)			
		0-4	1.5 *
		7	11.9

(* = significant different)

(After Schlenger et al 2002)

Table 2 - Levels of PTSD (%) in different situation in Schlenger et al (2002).

2. Sampling

Levels of PTSD between different studies vary greatly, and some of this is due to the type of trauma being studied (eg: war or natural disaster), but it is also due to the sampling process used. Table 3 lists the main types of sampling used in surveys.

SAMPLING TECHNIQUE	ADVANTAGE	DISADVANTAGE
Random sample - every member of research population has equal chance of being chosen	easy to sample	no guarantee of representativeness
Opportunity sample - random sample of those available	convenient	limited choice
Volunteer sample	overcomes ethical problems	volunteers not typical of general population (Brewer 2005)
Purposive sample - eg: quota sample from different groups based on age or gender	cross-section of population	difficult to achieve

Table 3 - Main types of sampling used in surveys.

Schlenger et al (2002), for example, used a random household sample (by random phone digit dialling) recruited before 11th September, which reduced the bias of self-selection (who volunteers) or selectivity of participants (ie: those known to be affected by event) if recruitment was after the event. But only 41% of the random sample agreed to participate, and 73% of them filled in the Web-based questionnaire. Schlenger et al were able, though, to show that their sample had demographic similarities to the population as a whole using census data.

3. Comparison groups

Though Schlenger et al had comparison data from other cities in the US, they did not have a baseline measurement (pre-11th September) for their sample.

4. Measurement of PTSD

Ideally each participant would be diagnosed there and then face-to-face by a psychiatrist with a clinical interview (1), but this is rarely done for large-scale studies. More often psychometric questionnaires (2) (like the Impact of Event Scale (3)) are used. Studies can use symptom checklists, and respondents simple tick them. But are these symptoms new (ie: after the event) (memory can be inaccurate), and/or persistence as required by DSM-IV?

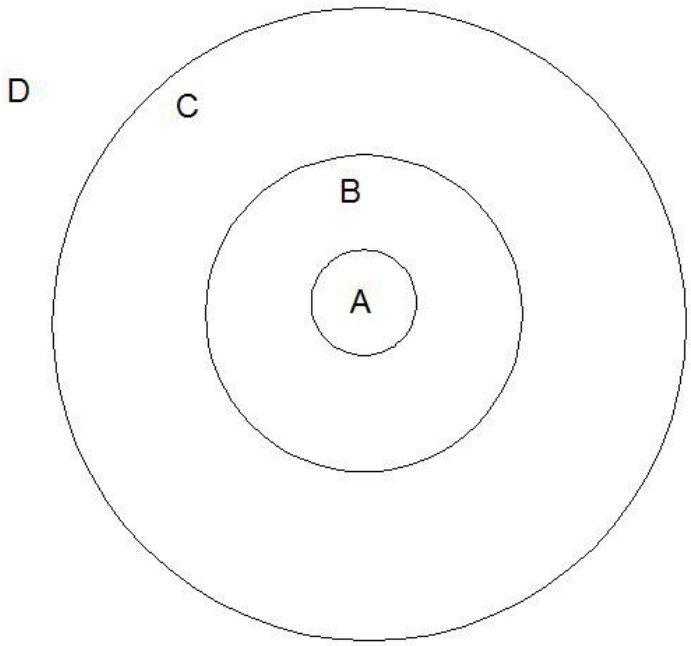
Individuals self-reporting their symptoms do so in a context of social understanding of PTSD. It is a balance between the existence of "normal" distress (which is not PTSD) and should not be diagnosed as so, and not to belittle the more extreme distress that is PTSD.

The amount of people diagnosed with PTSD (or any mental disorder) can be viewed as three circles (figure 1). The size of the circles will vary, and depending on things like public awareness of a problem or the "pathologizing of normal experience".

It is not easy to apply figures to the different circles, but Child (2003), explaining the diagnosis of ADHD, proposed 2% for the "core group" (A in figure 1), 25% for the "broad group" (B), 33% (C), and the remainder 40% (D). It is not clear how those figures were arrived at.

It is interesting to note that the criteria for diagnosing PTSD included eleven changes in DSM-IIIR (APA 1987) from DSM-III (APA 1980), and fifteen more in DSM-IV (APA 1994).

Ziskin (1995) noted 175 combinations of symptoms by



KEY:

A = Individuals who clearly have PTSD, and show all the symptoms over time. It does not necessarily require a psychiatrist to know that this person has problems, it can be seen in their everyday lives. This is the "pure" number of PTSD sufferers.

B = Individuals who have some of the symptoms and/or are borderline for a diagnosis of PTSD. This group may be included in the number of sufferers depending on the research design and sampling etc.

C = This is the area of concern where individuals are showing "normal" reactions to distressing events, but these are "pathologized" as part of PTSD. Self-reported symptom checklists could inflate this group.

D = The remainder of the population who clearly do not have any of the symptoms of PTSD.

Figure 1 - Layers of diagnosis of PTSD (or any mental disorder).

which to diagnose PTSD, and it was also possible to have or not have PTSD depending on the edition of DSM (even if the individual's symptoms remain unchanged). This will have an effect upon the size of the circles in figure 1.

5. Problems of diagnosis

North and Pfefferbaum (2002) asked "how to categorise experiences such as jumpiness, loss of concentration, and difficulty in sleeping reported among individuals outside the directly endangered area" (p635) of the World Trade Centre on 11th September. DSM-IV was not that helpful here because it included the phrase "confronted with" the event.

As to whether those who viewed an event of television can be included as PTSD victims, North and Pfefferbaum (2002) preferred to see the symptoms as "distinct from post traumatic stress disorder" though deserving recognition. DSM-IV-TR (APA 2000) did not specify that witnessing the event should be in person.

Prevalence of PTSD in Different Situations

Prevalence is the total number of cases in the population at a given time. Studies on the prevalence of PTSD vary from a few percent to over three-quarters of cases. The differences are a product of the sample studied and research design as much as the actual level of PTSD. This section gives some examples of studies of PTSD in relation to different events. It does not intend to be comprehensive.

1. General population

The current risk of PTSD in the general population varies from 1% (Kinuchin 1994) or 2-4% in North America (Stein 2002) to 10% in a lifetime (Stein 2002).

2. Armed conflicts

a) Directly involved - combatants

The rate of PTSD among soldiers varies from 20-50% (Haughton 1997) with most research on Vietnam veterans (table 4).

The studies in table 4 do not have baseline scores (ie: before the war) even if they use comparison groups (like Goldberg et al 1990). This makes the studies similar to quasi-experiments (or natural experiments) (4) which means caution about claiming causation.

Overall, the rate of PTSD among combatants will be influenced by the length of the conflict, and the amount of action seen.

STUDY	CURRENT *	LIFETIME **
Helzer et al (1987) 64 US soldiers	-	6.3
Snow et al (1988)*** 2858 US soldiers in SE Asia	15.0	-
Centers for Disease Control Vietnam Experience Study (1988) 2490 US Vietnam veterans	2.2	13.0
Goldberg et al (1990) 715 US MZ pairs (1 fought Vietnam/1 not)	16.8/5.0	-
National Vietnam Veterans Readjustment Study (1990)*** US SE Asia veterans 1173 males/424 females	15.2/8.5	30.6/26.9

* Current = at time of study
** Lifetime = at any point in life even if not currently
*** Quoted in Saigh 1992

Table 4 - Four studies of US Vietnam veterans and prevalence (%) of PTSD (using DSM-III criteria).

b) Indirectly involved - refugees and others

Armed conflicts also affect civilians in terms of refugees, journalists, partners of those exposed, caregivers, peacekeepers, and body handlers (Karam and Bou Ghosa 2003). Table 5 gives some examples of studies of these groups.

STUDY	DETAILS	PTSD (%)
Kinzie et al (1986)	40 Cambodian adolescent refugees taken to USA (DSM-III criteria)	50.0
Kinzie et al (1989) *	3 year follow-up	29.6
Saigh (1989) *	840 Lebanese child refugees referred to Red Cross (DSM-III criteria)	27.5
Dyregov et al (2002) **	Iraqi children and bombing of shelter in 1991 Persian Gulf War (IES score 17+) after 1 year after 2 years	 88 79
Mehlin & Weisth (2002) **	Norwegian UN peacekeepers in Lebanon	16

(* Quoted in Saigh 1992)
(** Quoted in Karam and Bou Ghosa 2003)

Table 5 - Four studies of PTSD and indirect victims of armed conflicts.

Journalists are often forgotten about in terms of individuals present during armed conflicts, and exposed to the effects. Feinstein et al (2002) compared 140 war journalists, who had spent an average of fifteen years covering war abroad, and domestic journalists. The former had higher IES scores and greater weekly consumption of alcohol.

As shown in table 5 many of those indirectly hit by armed conflicts are children. Somasundaram (1993) studied 625 adolescents in Vaddukoddia (North Sri Lanka), who were caught up in the armed conflict there. About one-third (31%) were seen as suffering from PTSD. But many other psychological problems were also evident; eg: anxiety (34% of sample) and depression (29%).

Thabet et al (2002) assessed ninety-one Palestinian children exposed to home bombardment and demolition during Al Aqsa Intifada, and eighty-nine controls exposed to other political violence in Gaza. The data were collected in January and February 2001. The children exposed to home bombardment were more prone to PTSD, and this exposure (and the feelings of anxiety and helplessness) was key to the severity of PTSD (table 6).

	EXPOSED TO HOME BOMBARDMENT	EXPOSED TO POLITICAL VIOLENCE ONLY
NO PTSD	3	3
SEVERE/VERY SEVERE	59	25
MILD/MODERATE	38	72

Table 6 - Severity of PTSD (%) in two groups of children in the Palestinian territories.

As mentioned earlier, these studies will not have baseline measurements (ie: pre-conflict). Studies like Saigh (1989) were using a self-selected sample which may not be representative of the whole research population.

3. Political violence and terrorism

The rates of PTSD for victims of political violence and terrorism vary depending on the type of event; eg: bombing survivors (50%), and hijack victims (37%) (Kinchin 1994), or torture victims (70%) (Haughton 1997).

Wilson and Cairns (1992) summarised the work on the effect of political violence in Northern Ireland in the 1970s and 1980s. For example, a 1970s study found that 92% of uninjured bystanders at bomb explosions had

"serious affective disturbances". Around a quarter of the victims of violence in other studies had PTSD.

Cross-cultural comparisons of PTSD are relatively rare. One exception (North et al 2005) looked at the terrorist bombings of the US Embassy in Nairobi, Kenya in 1998 and of the Murrah Federal Building in Oklahoma City, USA in 1995. The latter event was studied by interviewing 182 survivors using the Diagnostic Interview Schedule - Disaster Supplement (DIS/DS) (North et al 2001) four to six months after the event (North et al 1999).

The Kenyan bombing was studied with the same interview schedule eight to ten months after the event, and involved 227 Kenyan civilians. Of this group, about one-third of the men and about one half of the women had "bombing-related PTSD", which were similar rates to the Oklahoma city group.

Confirming other research findings, more women than men suffered from PTSD in Kenya, and pre-existing psychiatric disorders were a risk factor for PTSD (table 7).

		OKLAHOMA CITY	NAIROBI
Pre-disaster psychiatric disorder			
	YES	41	60
	NO	26	36
	sig	0.035	0.002

Table 7 - Amount of PTSD (%) based on pre-disaster psychiatric disorder.

But there were differences between the two countries. In Oklahoma City, the number of injuries positively correlated with PTSD as did death or injury to a family member or friend. Neither of these were so in Nairobi.

Torture victims are a separate category of political violence. For example, Weiseath (1989 quoted in Saigh 1992), using DSM-III criteria, found, six months later, a current rate of 58.3% for PTSD among thirteen Norwegian sailors held in Libya for 67 days.

Ehlers et al (2000) interviewed eighty-one former political prisoners about their experiences of prison. Those who were suffering from PTSD were more likely to have felt alienated about the world, had perceived negative changes in their personality from prison, and perceived mental defects (eg: talking under interrogation and/or torture).

4. Natural disasters

Again the level of PTSD among survivors depends upon the type of natural disaster experienced. For example, shipwreck survivors (75%) and air crash (22%) (Kinchin 1994). Table 8 gives two examples of studies using DSM-III criteria.

Experiencing traumatic events like natural disasters has long-term consequences. Hull et al (2002) interviewed survivors of the Piper Alpha oil platform disaster in the North Sea ten years after. Thirty-three of fifty-nine survivors were interviewed, and 7 (21%) were diagnosed with PTSD at that time. The key characteristics for sufferers of PTSD were physical injury, and "survivor guilt".

A key variable for PTSD and natural disasters is how quickly things get back to normal.

STUDY	DETAILS	PTSD (%)	
Madakasira & O-Brien (1987)	116 tornado victims in South Carolina; five months after event	59.5	current
Green et al (1990; quoted in Saigh 1992)	120 flood victims; fourteen years after	28.3 44.2	current lifetime

Table 8 - Two studies of PTSD and natural disasters.

Studies comparing types of events are rare. But Goenjian et al (2000) compared survivors of an earthquake with those who had seen political violence in the same part of the world in 1988. Table 9 shows the estimated levels of PTSD (using DSM-IIIR criteria) in the three groups. However, the study was only based on a total of 78 participants, and the estimated rate of PTSD.

GROUP	N	AFTER EVENT	
		1.5 yrs	4.5 yrs
Severe earthquake survivors from Gumri, Armenia	30	86.7	73.3
Mild earthquake survivors from Yerevan, Armenia	29	13.8	6.9
Political violence survivors from Sumgait, Azerbaijan	19	89.5	94.9

(After Goenjian et al 2000)

Table 9 - Rates of PTSD(%) for three groups of survivors.

5. More common events

i) Car crashes

Haughton (1997) quoted the rate of PTSD as 12% for severe car crash survivors, while Stallard et al (1998) reported around one-third for children involved in road traffic accidents (which included being hit by a vehicle).

ii) Crime

Kilpatrick et al (1987) found a current rate of 7.5% and a lifetime rate of 27.8% for PTSD among 295 female victims of crime (half of sexual crimes). Kinchin (1994) quoted up to 50% for rape victims.

Among 157 victims of crime, shame, and anger with others predicted PTSD at one month after the event (Andrews et al 2000).

From a different point of view, Kruppa (1991) noted that perpetrators of violent crime (including murder) can suffer from PTSD.

McLeer et al (1992) showed that nearly half (48.8%) of thirty-one multiple child sexual abuse sufferers had current PTSD. Childhood trauma is also a predictor for PTSD in response to future trauma.

iii) Medical conditions

Czarnocka and Slade (2000) found 3% of 264 Sheffield women suffering from PTSD following childbirth. While Cohen et al (2002) calculated a rate of 57% for 77 fibromyalgia (rheumatism) patients in Israel.

6. Rescue and medical services

Kinchin (1994) quoted PTSD rates of 15% for emergency service staff and carers.

Paton (1992) summarised studies of three groups of relief workers: (a) police and medical workers after the Piper Alpha oil platform disaster; (b) fire-fighters and volunteers after an earthquake in Armenia; and (c) volunteer nurses in Romanian orphanages. The last group were most affected as measured by the Impact of Event Scale. Their mean score was 17.39 on the "Intrusion sub-scale" (which measures intrusive thoughts) compared to less than six for the other groups. The key was the importance of preparation about what the workers would

find, and the nurses were not prepared compared to the other groups.

7. Miscellaneous

i) Children

Studies (eg: Somasundaram 1993) showed that children suffer badly from PTSD symptoms.

Stallard et al (1998) compared 119 children (aged 5-18 years old) involved in road traffic accidents and sixty-six with sports injuries at Bath Hospital in 1996-7. Only 2-3% of the latter group had PTSD compared to 41 (34.5%) of the children in road traffic accidents. For this group, PTSD was significantly associated with girls; previous experience of trauma in last twelve months; and belief that would die, but not age nor type or severity of injury.

ii) Cross-cultural studies

These types of studies are increasing in number, but most of the research on PTSD is in the Western or Developed countries. Table 10 gives two examples of studies from Africa.

STUDY	DETAILS	PTSD (%)
Carey et al (2003)	sample = those seeking medical help; diagnostic instrument used	42 male lifetime 46 female lifetime
Seedat et al (2004)	adolescents in South Africa (20% sample = black) and Kenya (97% black)	22 South Africa 5 Kenya

Table 10 - Two recent studies of PTSD from Africa.

Risk Factors for PTSD

What distinguishes those who will experience PTSD and those who will not ? There are certain common risk factors:

i) The event is life-threatening or perceived as so;

ii) The length of event;

iii) Vulnerability of the individual to psychological problems or had them before the event;

iv) Individual has an anxious disposition;

v) Actual or perceived lack of support afterwards.

In Czarnocka and Slade's (2000) study of Sheffield women showing PTSD after childbirth, four variables emerged to distinguish the minority who suffered: perceived low level of support from partner or staff; low perceived control in labour; previous mental health problems; and "trait anxiety" (anxious disposition).

Level of post-event support was crucial in a study of armed robbery victims in UK Building Societies (Richards 2000). The victims were assessed at one month after the event (n = 51) and at six months after (n = 31). Higher levels of PTSD symptoms at both time periods was associated with poor crisis support.

King et al (1999) developed a path model from the data of 1632 male and female US Vietnam veterans who had seen traditional combat. Three groups of factors were involved in distinguishing those who developed PTSD. Table 11 gives some examples.

In a similar study, Nishith et al (2000) found that prior trauma (eg: childhood sexual abuse or other adult victimization, but not childhood physical abuse) was important among 117 adult rape victims as to who developed PTSD.

Stein (2002) found that females were more affected by PTSD after motor vehicle collisions as well as the usual factors of perceived threat. The level of injury was inconsistent with PTSD.

Karam and Bou Ghosa (2003) added Anti-Social Personality Disorder and substance use as pre-exposure variables. But Vietnam veterans with high IQ "fended off" PTSD (Staff Writer 2002).

FACTORS	INCREASES RISK OF DEVELOPING PTSD	DECREASES RISK OF DEVELOPING PTSD
PRE-WAR FACTORS	family instability	family stability
WAR-ZONE STRESSORS	additional life stresses; high perceived threat	no additional life stresses; low perceived threat
POST-WAR RESILIENCE RECOVERY VARIABLES	no social support; no hardiness	social support; hardiness as personality characteristic

Table 11 - Examples of risk factors among Vietnam veterans for PTSD as found by King et al (1999).

O'Brien (1998) summarised the risk factors under following headings:

i) Trauma itself is important, but it is not the sole cause of PTSD;

ii) The severity of the trauma is important;

iii) Pre-trauma risk factors are important. These include family instability, child abuse, family mental illness, own mental illness, and childhood behavioural problems;

iv) Post-trauma experience including level of support.

Conclusions

Trying to establish exact numbers of sufferers of PTSD is difficult because of the factors mentioned earlier (eg: type of trauma), but here is a general attempt using the types of victims (table 12) and the three cicles idea (table 13). These should be taken as approximations.

The terrorist events of 11th September 2001 are a good example where social meaning and construction can worsened any distress. For example, the replay of images, and place in social history as "world post 9/11", even "9/11" which links to the US emergency phone number (911). This event also destroyed the myth of invulnerability that existed in the USA, and thus the event means so much more than the actual damage and deaths. In other words, the symbolic value. Is it possible to have PTSD based on the symbolic meaning of an event as much as the actual nature of the damage?

TYPE OF VICTIMS	RISK OF PTSD	FACTORS
Primary	one-third to over three-quarters	- severity of event - child or adult - type of event
Secondary	up one-third	- suddenness or unexpected of event - social support
Tertiary	can be high	- social construction of event: eg: replay of images
Rescuers/medical	one-fifth	- preparation - expectations of society about caring - experience of such situations
Peripheral	one quarter	- social meaning of event; eg: 11th Sept - media coverage - value placed on distress

Table 12 - Approximate figures for different types of PTSD victims.

CATEGORY		PTSD
A	Core sufferers with all symptoms; no disagreement over diagnosis of PTSD	less 5%
B	Individuals with some symptoms; included or not depending on diagnosis used in study eg DSM-III or DSM-IV	20%
C	Individuals with "normal" distress who include themselves as PTSD on self-reporting checklists or part of "pathologizing of everyday life"	25-40%
D	Remainder with no symptoms of PTSD or distress (5)	35-50

Table 13 - Prevalence of PTSD applied to the three circles in figure 1.

Footnotes

1. The bedrock of psychiatry is the clinical interview. Based on a combination of structured and unstructured questions, the psychiatrist builds up the information for diagnosis. It allows an individual assessment of the patient, but is dependent on the interaction between psychiatry and the patient (eg: impression formation and management) (Brewer 2002).

2. Psychometric tests are standardised questionnaires with comparative norms of behaviour to use. Good tests have reliability (consistency), validity (measure what claim to be measuring), and discrimination (ability to distinguish between high and low scorers).

3. Impact of Event Scale (Horowitz et al 1979) has 15 items and uses a scoring of 0-4 on frequency and intensity; It has sub-scales for "intrusion" and "avoidance".

4. Quasi-experiments and natural experiments are similar to experiments, but lack the control to establish the causality of behaviour. A "true" experiment should have randomisation of participants, standardised procedures, and control over variables. Studying individuals after an event, even with a comparison group, does not fulfil the criteria for a "true" experiment. Therefore care should be taken in talking about an event causing PTSD.

5. It could be asked if not showing distress to certain situations is a good thing.

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Dual Diagnosis: Learning Disability and Mental Disorder - Thinking About the Nature of the Relationship

INTRODUCTION

Mental disorders are more common in individuals with learning disability than the general population, and the nature of the disorders can be both similar and different to the general population (Volkmar and Dykens 2002).

There can also be differences in mental disorders between the types of learning disability (eg: obsessive-compulsive features are greater in Prader-Willi syndrome than other learning disabilities; Dykens et al 1996), and common characteristics between them (eg: self-injury in 5p (cri du chat) and Smith-Magenis syndromes; Volkmar and Dykens 2002).

However, the similarities in characteristics may be manifest in qualitatively different ways. Inattention is manifest as part of anxiety in Williams syndrome, and associated with hyperarousal in Fragile X syndrome (Volkmar and Dykens 2002).

PREVALENCE OF MENTAL DISORDERS AMONG INDIVIDUALS WITH LEARNING DISABILITIES

Historically the presence of psychiatric diagnosis were assumed to be part of the learning disability. This was known as "diagnostic overshadowing" (Reiss et al 1982).

The difference in the presence of psychiatric disorders can be quite large; eg: 30% among children with developmental delay versus 6% of the control group (Rutter et al 1976), and even higher for severe learning disability (Corbett 1979).

In terms of the seriousness of the psychiatric problems, about a quarter of individuals with learning disabilities can be said to have significant problems including behavioural difficulties (Jacobson 1999).

The exact prevalence of psychiatric disorders among individuals with learning disabilities varies with the type of study performed.

i) Clinical or hospital samples

For example, Philips and Williams (1975) found that 87% of 100 consecutive children with learning disabilities referred to a psychiatric clinic were also

diagnosed with a mental disorder. Table 1 lists some examples of similar studies (Volkmar and Dykens 2002).

STUDY	SAMPLE	FINDINGS	GENERAL POPULATION
Reid (1972)	500 adult	3.2% psychosis	6% (Miles 1987)
Meyers (1986)	62 adult	19% schizophrenia 24% psychosis 11.0% depression	0.5 - 1.0% (APA 1994) 10% worldwide (Bhugra & Mastrogianni 2004)
Grizenko et al (1991)	176 adolescent	2.8% schizophrenia 2.3% psychosis	very low (Meltzer et al 2000)
LaMalfa et al (1997)	176 adult	4.5% schizophrenia 1.1% depression	

Table 1 - Examples of studies of prevalence of mental disorders in clinical and hospital samples of individuals with learning disabilities.

ii) General samples of individuals with learning disabilities

The rates of mental disorder vary between 10-15% to 30-40% depending on the sample, the definitions of mental disorder used, and the methods of behaviour measure (eg: behaviour checklists) (Volkmar and Dykens 2002). Table 2 summarises some of the studies.

STUDY	SAMPLE	FINDINGS
Lund (1985)	302 adult	1.3% schizophrenia; 5% psychosis; 1.7% depression
Gillberg et al (1986)	149 adolescent	1.0% schizophrenia; 10.0% depression (1-6% in general population; Harrington 2002); 11.0% ADHD (3-5% in general population; APA 1994)
Cooper (1997)	602 older adults	9.0% schizophrenia; 5-10% depression

(After Volkmar and Dykens 2002)

Table 2 - Examples of studies of prevalence of mental disorders in general samples of individuals with learning disabilities.

The specific reasons for the increased prevalence of mental disorders among individuals with learning disabilities are open to debate, and no comprehensive model of explanation exists (Volkmar and Dykens 2002).

Furthermore, the causal direction is not entirely clear - for example, are behavioural problems a precursor or consequence of psychopathology?

Pennington et al (1993) looked at the co-morbidity of dyslexia and attentional problems in three groups of children: dyslexic readers with no attentional problems (RD), dyslexic readers with ADHD (co-morbidity group), and a group of children with attention-deficit-hyperactivity-disorder (ADHD) only.

The experiment was based upon two tasks - a phonological task which measures dyslexia, and an executive function task requiring attention and planning that distinguishes ADHD. The co-morbidity group showed the same pattern as the RD group (table 3), which would suggest they develop attentional problems as a consequence of dyslexia (Snowling 2002).

GROUP	PHONOLOGICAL TASK	EXECUTIVE TASK	
RD	difficulty	no difficulty	expected
ADHD	no difficulty	difficulty	expected
Co-morbidity	Difficulty	no difficulty	as RD group

Table 3 - Performance of two tasks by three groups of children in Pennington et al (1993).

There are certain risk factors have been found to be associated with mental disorders among individuals with learning disabilities (Volkmar and Dykens 2002) (table 4).

- Aberrant personality styles; eg: too wary or disinhibited
- Atypical motivational styles; eg: need for attention
- Risk of failure experiences in life and at school
- Negative evaluation of whole self concept rather than just parts
- Reinforcement of negative behaviours
- Poor communication skills
- Stressful social interactions
- Social stigma
- Peer rejection
- Poor social skills
- Risks of abuse and exploitation
- Family stress
- Seizure disorders (Caplan et al 1998)
- Abnormal neurological functioning
- Biochemical anomalies
- Genetic risks

Table 4 - Risk factors for mental disorders in individuals with learning disabilities.

GENETIC SYNDROMES OF LEARNING DISABILITIES

Looking at dual diagnosis, genetic syndromes of learning disabilities have proved most fruitful. Table 5 gives some of the key behavioural problems associated with genetic syndromes of learning disabilities.

SYNDROME	CAUSE	KEY BEHAVIOURAL PROBLEMS
Fragile X	FMR-1 gene amplification of CGG	Inattention; hyperactivity; social anxiety; males - hyperactivity, attentional deficits; females - schizotypal disorder
Williams	micro-deletion on chromosome 7	Anxiety; phobias; inattention; social disinhibition; anxiety; ADHD
Prader Willi	paternal gene missing on chromosome 15 through deletion or disomy	Non-food obsessions and compulsions; skin picking; underactivity; depression; impulse control disorder; obsessive-compulsive disorder
Down	chromosome 21 trisomy	Physical signs; eg: short stature, facial differences; affective disorder; dementia
Smith-Magenis	chromosome deletions on 17	Hyperactivity; aggression; impulsivity; self-injurious behaviour; anxiety; ADHD; sleep disorders
Velocardiofacial	deletion on chromosome 22	Cleft palate; long face with prominent nose; small stature; psychosis

Table 5 - Key behavioural problems associated with genetic syndromes of learning disabilities.

Certain psychiatric problems (anxiety, depression, and schizotypal symptoms) seem to occur in many genetic syndromes of learning disabilities (table 6).

CONDITION	ANXIETY	DEPRESSION	SCHIZOTYPAL
Fragile X	yes	yes	yes
Williams	yes		
Prader Willi	yes	yes	
Velocardiofacial		yes	yes

(After Skuse and Kuntsi 2002)

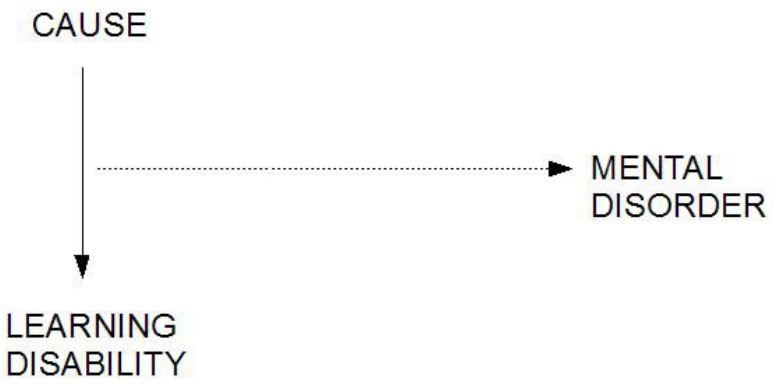
Table 6 - Anxiety, depression and schizotypal symptoms in four learning disability conditions.

THE NATURE OF THE RELATIONSHIP BETWEEN LEARNING DISABILITIES AND MENTAL DISORDERS

This section considers the possible relationships or causes for learning disabilities and mental disorders together under a number of headings. The aim is to open the ideas for thought and discussion as much as to give a definitive answer.

Finding the causes for learning disabilities and mental disorders will also depend upon whether the learning disability is organic (with a clear cause like genetic disorders) or "cultural-famalial" (no clear organic cause) (Zigler 1967).

MODEL 1: The cause of the learning disability leaves the individual vulnerable to a mental disorder.



Examples:

Velocardiofacial syndrome (VCFS) is caused by a deletion on chromosome 22, and the missing genes produce an enzyme (catechol-O-methyltransferase; COMT) related to dopamine; Dopamine malfunctioning can be related to psychosis. Psychosis is prevalent in between 22-40% of individuals with VCFS (Thomas 2003). The missing genes which caused the VCFS make the individual vulnerable to the mental disorder.

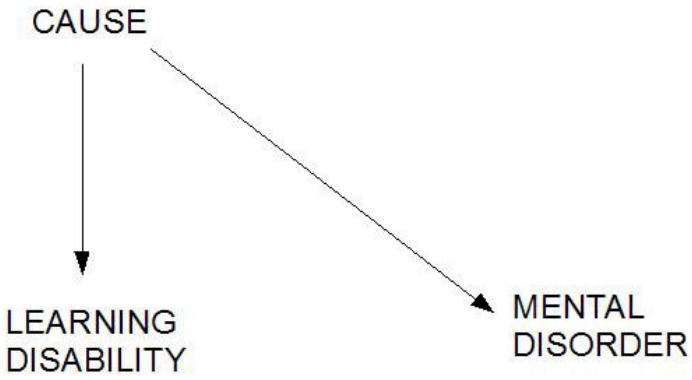
There is a higher prevalence of dementia in adults with Down syndrome than in the general population and among adults with other learning disabilities (McCallion and McCarron 2004). Focus upon adults with Down syndrome has found an overproduction of amyloid precursor protein (Head et al 2003), which is known to be involved in dementia in the general population.

Down syndrome can also lead to early onset menopause for female sufferers, which is a risk for early onset

dementia (Schupf et al 2003).

Adults with Down syndrome are vulnerable to depression as well, and this may be "overshadowed" by the diagnosis of dementia (Dykens 1998).

MODEL 2: The same cause for the learning disability and the mental disorder.

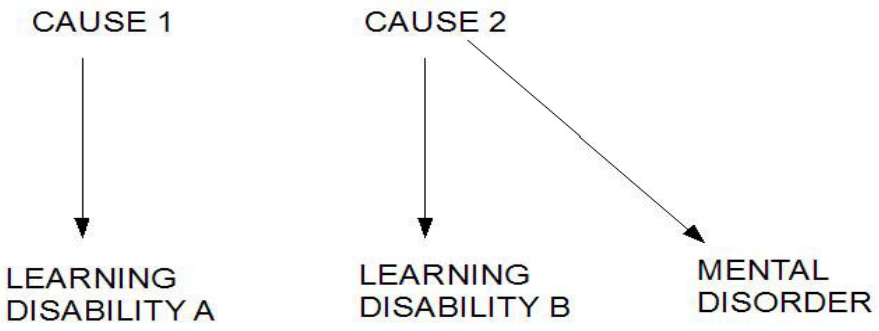


This may be manifest in similar symptoms for both a learning disability and a mental disorder, for example, in terms of the same genes implicated.

One piece of evidence to back up this model would be high co-morbidity between particular learning disabilities and mental disorders; eg: VCFS and ADHD (Gothelf et al 2003); or over 90% of clinic-referred boys with Fragile X syndrome had ADHD (Dyken 1998).

But the problem with this evidence is that many children have ADHD with no learning disability. Unless there are different causes for ADHD depending on whether the individual has a learning disability or not.

MODEL 3: There are different causes for learning disabilities, some of which also cause a mental disorder.



It could be that the learning disability originating from environmental causes, for example, does not also cause a mental disorder, whereas when the cause is biological then there is also a mental disorder. Or different biological causes have different effects.

Examples:

Researchers have examined the different causes of Prader Willi syndrome (PWS): deletion of a genes (in about 70% of cases; Nicholls et al 1989) or uniparental disomy (UPD) (1) (in 30% of cases) and mental disorder (table 7).

STUDY	MENTAL DISORDER	DELETION	UPD
Verhoeven et al (2003)	psychosis or bipolar disorder	2	14 individuals
Vogels et al (2003)	psychosis		5 of 6 individuals had chromosome 15 maternal UPD
Einfeld et al (2004)	psychosis major depression	3 of 4 4 of 4 individuals	

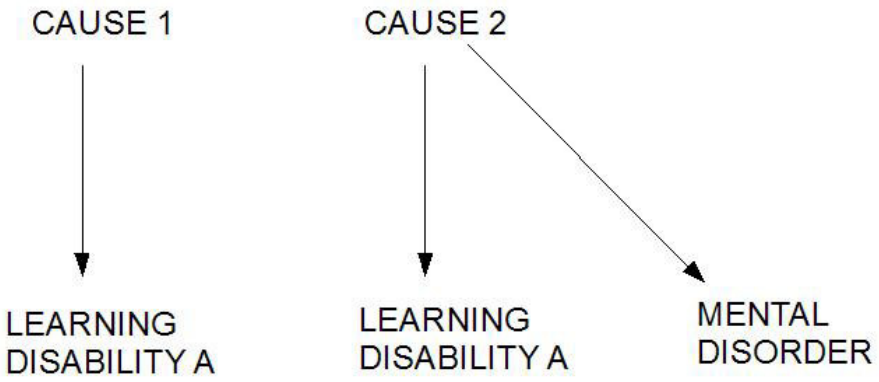
Table 7 - Prader Willi syndrome and mental disorders.

Einfeld (2004) concluded that in PWS individuals, compulsive behaviour appears to be associated with gene deletion, and psychosis with "imprinting defects" (eg: UPD), "but these associations have not been found in all studies" (p344).

Another example here could be the differences found by Clarke et al (2002) between individuals with PWS and those with other learning disabilities who were overweight. The former showed higher rates of ritualistic behaviours (eg: the need to tell something), hoarding, and repetitive actions. But they did not show other behaviours associated with obsession-compulsion conditions, like obsessive thoughts or compulsive checking.

This can be seen as an argument against model 5. There are not common symptoms of obsessive-compulsive behaviour irrelevant of whether they manifest themselves as part of a learning disability or just as a mental disorder.

MODEL 4: Different causes for the same learning disability, and some of the causes also produce a mental disorder.



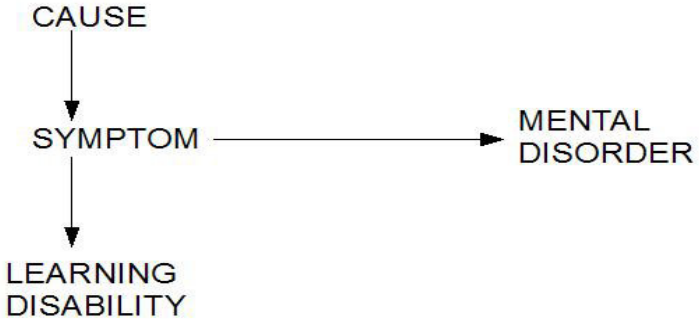
The most obvious version of this relationship would be in terms of specific genes. In the figure above, both cause 1 and 2 produce the same learning disability, but only cause 2 also leads to a mental disorder.

Example:

An example of this model could be the differences found in individuals with PWS relating to word search skills depending on the genetic cause. Individuals with gene deletions scored the same as non-learning disabled peers on word searches, whereas PWS individuals with UPD did not (Dykens 2003).

Butler et al (2004) found that PWS individuals with type I deletions had worse adaptive behaviour scores and more obsessive-compulsive behaviours than UPD individuals with PWS.

MODEL 5: All symptoms have the same cause.



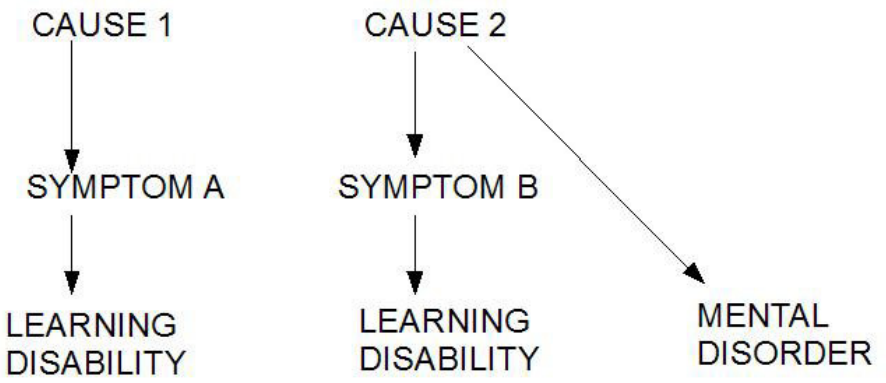
This explanation focuses upon the cause of the

symptom rather than the whole disability or disorder. So, for example, hyperactivity has a common cause whether that appears as part of ADHD or as seen in Fragile X syndrome.

Example:

Russell et al (2005) compared a group of adults with high-functioning autistic-spectrum disorder and a matched group with obsessive-compulsive disorder (OCD) on obsessive-compulsive symptoms. The two groups had similar frequencies of obsessive-compulsive symptoms in the main, but repeating rituals, for example, was more common in the OCD group.

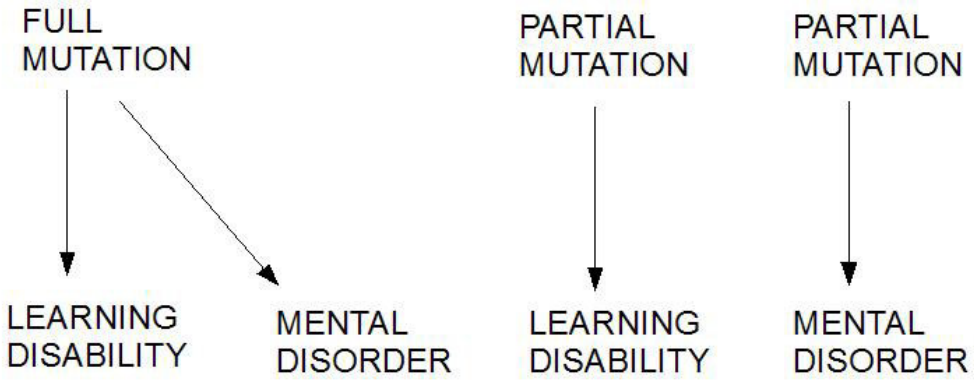
MODEL 6: Each symptom of a learning disability has different causes, and some of the causes may also produce a mental disorder.



This explanation also concentrates upon the symptoms and their causes. Some symptoms are specific to learning disabilities, but others will have a cause that also produces a mental disorder. It is possible to refer to earlier examples with PWS in model 3 here (eg: table 7).

MODEL 7: The degree of mutation

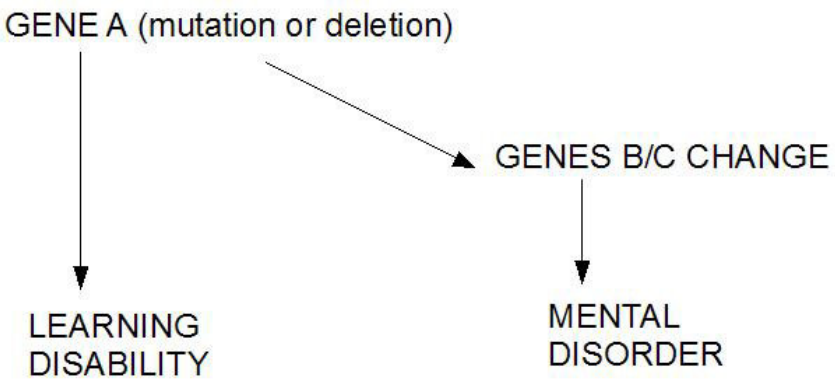
This model relates to a genetic explanation only, and is the degree or level of mutation of a gene(s). For example, a full mutation leads to a learning disability and a mental disorder, while a partial mutation produces only a learning disability or a mental disorder.



Example:

The full mutation of Fragile X Mental Retardation (FMR1) gene produces Fragile X syndrome, while a pre-mutation version (2) produces tremor and cognitive decline in older males (Hagerman et al 2003). Female carriers of the pre-mutation version (not the full mutation) have increased rates of premature ovarian failure (Einfeld 2004).

MODEL 8: The effect of genes upon other genes.



This explanation is also based in genetics. The mutation or absence of a particular gene leads to a learning disability, but this change in the gene causes other genes to behave differently which produces a mental disorder.

Examples:

Studies of Fragile X Mental Retardation protein (FMRP) in Fragile X syndrome have shown changes in other

biological processes (Einfeld 2004).

Holland et al (2003) suggested that PWS may originate from the absence of a paternal version of a gene that controls satiety. Could the absence of the paternal version have a knock-on effect in terms of other genes, and subsequently mental disorders?

AFTERTHOUGHT

Any attempts to explain mental disorders and learning disabilities must face the fact that some psychiatric problems are stable across the lifespan (eg: PWS and tantrums), while others vary between child and adulthood (eg: Down syndrome children and disruptive behaviour; adults and depression and dementia) (Dykens 1998).

APPENDIX: BASIC GENETICS

Each cell in the body contains a complete set of chromosomes. The chromosomes are numbered 1 to 22 in humans based on decreasing size.

The chromosome complement is known as a karotype, which for humans is 46: 22 pairs (known as autosomes) the same for either sex and then XX or XY.

On occasions, there can be extra copies of a chromosome (known as trisomy); eg: Downs syndrome and trisomy 21 (an extra copy of chromosome 21). Other chromosomal abnormalities include deletions or duplications of part of a chromosome.

Chromosomes are made up of genes, which are based on DNA (deoxyribonucleic acid). DNA is made up of four bases: adenine (A), guanine (G), cytosine (C), and thymine (T). These are ordered around two chains wrapped together as the double helix.

The nature of DNA is such that A always pairs with T, and C and G. The sequence of bases is the genetic information.

Changes in the sequence of bases leads to changes in cell development. Point mutation is the subsitution of one base for another, and is the most obvious example. Others include deletions (loss of sequence of bases), insertions (gaining of a piece of DNA) (3), frameshift mutations (the loss of one base affecting the coding of others), and translocations (the breaking of part of a chromosome and reforming at a different site on a different chromosome) (figure 1).

NORMAL SEQUENCE	ACCGTTTTA
DELETION	ACCGTTT..
POINT MUTATION	ACCG**A**TTTA
INSERTION	ACC**TTGTTG**

Figure 1 - Examples of changes in sequence of bases.

It is possible to focus upon a particular loci (position on chromosome), and to see which allele exists.

At any given genetic locus (position), there are two alleles (copies) of the DNA sequence. One of the alleles is from the mother, and one from the father.

Most diseases are complex and not linked to single genes. There are a number of genetic mechanisms involved (Craddock and Owens 1996):

i) Epistasis - this is the interaction of multiple genes to produce the illness. This process is known to be the cause of retinitis pigmentosa (progressive degeneration of the retina).

ii) Locus heterogeneity - this is where multiple genes are involved, but any one gene can produce the illness on its own also. Retinitis pigmentosa is linked to 14 different loci (gene positions).

iii) Allelic heterogeneity - there are multiple alleles at a single disease locus. This is a number of possible genes at one particular situation, and a certain combination produces the illness.

iv) Dynamic mutation - here at a single disease locus, the allele mutates between generations. A particular gene mutates between parents and offspring. This is the case for Huntington's chorea.

v) Parent of origin effect - the expression of the allele depends upon the parental origin. The gene at a particular locus from the biological father will have a different effect to that from the biological mother.

The absence of maternal chromosome 15 (q11-13) leads to Angelman syndrome (eg: spasms, "puppet-like" movements, and low intelligence), while absence of the paternal version causes Prader Willi syndrome.

vi) Mitochondrial gene mutation - the illness is linked to the maternal pattern of inheritance as genes in the mitochondria are only inherited from the mother. This mechanism may be involved in some forms of deafness.

FOOTNOTES

1. Uniparental disomy means that both genes in a pair come from one biological parent instead of one from each biological parent. Maternal disomy from the mother, and paternal disomy from the father.

2. Full mutation means a certain number (threshold) of repetitions of bases (CGG), and pre-mutation is less than that threshold number.

3. Eg: Fragile X syndrome and the X chromosome. Normally there are between 6-50 copies of the sequence CGG, but in fragile X, there are between 230-2000 copies of the sequence. This is an example of a disorder associated with expanded triplet repeat.

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