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Semantic priming in schizophrenia: systematic review and meta-analysis

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Background
Increased semantic priming is an influential theory of thought disorder in schizophrenia. However, studies to date have had conflicting findings.

Aims
To investigate semantic memory in people with and without thought disorder.

Method
Data were pooled from 36 studies comparing patients with schizophrenia and normal controls in semantic priming tasks. Data from 18 studies comparing patients with thought disorder to normal controls, and 13 studies comparing patients with and without thought disorder were also pooled.

Results
There was no support for altered semantic priming in schizophrenia as a whole. Increased semantic priming in patients with thought disorder was supported, but this was significant only in comparison with normal controls and not in comparison with patients without thought disorder. Stimulus onset asynchrony (SOA) and general slowing of reaction time moderated the effect size for priming in patients with thought disorder.

Conclusions
Meta-analysis provides qualified support for increased semantic priming as a psychological abnormality underlying thought disorder. However, the possibility that the effect is an artefact of general slowing of reaction time in schizophrenia has not been excluded.

Declaration of interest
None.

Increased semantic priming or semantic hyperpriming is currently one of the most influential theories of thought disorder, the incoherent speech seen in some patients with schizophrenia. It originated with Maher, who proposed that a pathological heightening of the normal associative processes between words could lead to the intrusion into speech of material which would normally be excluded on the basis of its irrelevance in the current context.[1] Maher's associative process between words was similar in all essential respects to the concept of spread of activation in network theories of semantic memory, and this meant that the proposal could be readily tested by means of the lexical decision or semantic priming task[2]. In this paradigm, a string of letters is flashed up on a computer screen and the participant has to decide by a key press whether it is a word or a non-word (Fig.1). Immediately beforehand, another word – the prime – is briefly shown which may or may not be semantically related to the target word (if this happens to be a real word). The time taken to decide that the letter string is a word is significantly reduced when the prime is related to the target, a phenomenon that is most easily explained as the prime producing spread of activation to associated words in semantic memory, and so reducing the amount of activation needed to identify that it is a real word.

Over 30 studies of semantic priming in schizophrenia have now been carried out. Influential early studies by Manschreck et al[3] and Spitzer et al[4] found evidence for increased semantic priming in patients with thought disorder. Other studies, for example by Ober and co-workers,[5,6] then also suggested that priming is reduced in schizophrenia as a whole. The basic priming design has been subjected to many variations across the studies, one of the most important of which has concerned the use of a short or a long prime–target interval (stimulus onset asynchrony or SOA). Priming at SOAs of up to approximately 400 ms captures the 'automatic' process of spread of activation in semantic memory. However, it is clear that other 'controlled' processes become important at longer SOAs.[2] In a review by Minzenberg et al,[7] findings were divided over whether priming was increased in patients with thought disorder; however, a relatively consistent finding was reduced priming at long SOAs in schizophrenia as a whole. This report also drew attention to the fact that few studies had corrected for a well-recognised psychometric artefact whereby, if patients with schizophrenia are slower to respond on both the unprimed and primed versions of the task, the value for priming will tend to be spuriously inflated.

Method

Papers reporting semantic priming studies in patients with schizophrenia were searched electronically from 1988 (the year of the publication of the first study[3]) to April 2007. Studies were identified initially through PubMed, and then MEDLINE, PsychINFO and EMBASE, using the keywords schizophrenia, priming and semantic priming. The electronic search was supplemented by checking of review articles and the reference lists of all research papers obtained. To be included, studies had to report reaction time data in patients with schizophrenia and normal controls in any type of semantic priming paradigm. Age and gender matching was not required. Use of diagnostic criteria for schizophrenia was also not required. Non-English language papers and unpublished studies (e.g. theses) were included.

Most studies reported data on the lexical decision task; however, a few used the alternative procedure of word pronunciation (where the subject speaks the target word or string, and reaction time to do this is recorded by a voice key). Most studies reported 'subtractive' values for priming, that is, mean unprimed reaction time minus mean primed reaction time. Some studies instead
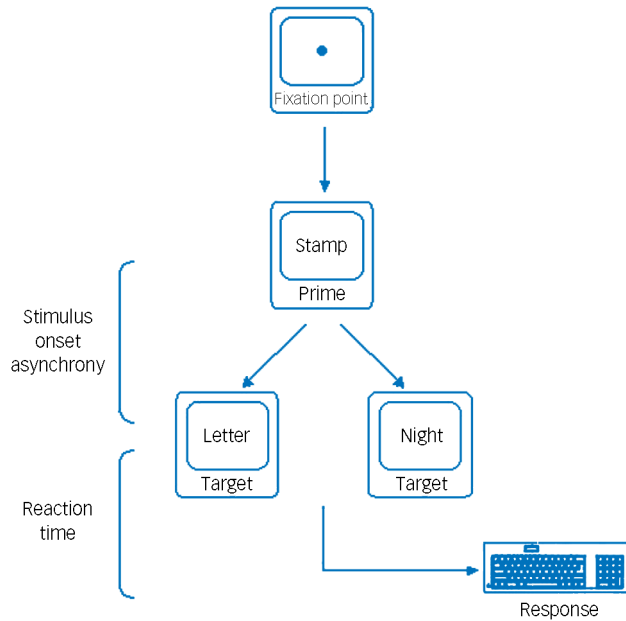
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Fig. 1 The lexical decision task

reported priming as 'percentage gain' in reaction time, in an attempt to avoid the above-mentioned tendency for overall slowing of reaction time to artefactually increase the value for priming when calculated by subtraction. As less than a third of studies used this technique, subtractive measures were used in the analyses, unless percentage gain was the only measure reported. Other variations in experimental design were either examined as moderator variables or ignored.

Data obtained from each study were converted into an effect size Cohen's *d*, the difference between the means for the patient and control groups divided by their pooled standard deviation. Hedges' correction was used; this corrects for the tendency of studies with small sample sizes to overestimate the effect size. Where means and standard deviations were not available, effect sizes were derived from *t*-values, *F*-values or *P*-values. In some cases, priming effects were presented only as interaction *F*-values in a two-by-two (between and within) ANOVA on the raw reaction times in the primed and unprimed conditions. In such cases, the interaction effect is comparable to one way ANOVA using difference scores – although the sums of squares will probably not be identical (taking the difference scores makes all scores closer to the overall mean), the ratio of sum of squares, degrees of freedom, *F* ratios and η^2 should be the same (M. Aitken, personal communication). For some studies, data were extracted from graphs or scatter plots using a digitising program (UnGraph; http://www.biosoft.com). Authors were contacted if effect sizes could not be extracted from any of the published data. All effect sizes were extracted a second time and differences resolved.

The meta-analysis was carried out using DSTAT 1.10[8] which uses a fixed effects model. Individual effect sizes were combined to produce an overall effect size, with each *d*-value weighted by the reciprocal of its variance. Analysis of moderator variables was based on the weighted effect size for each study using the *Q* statistic for categorical variables and Rosenthal's focused comparison for continuous variables.[9] Moderator variables included SOA, age, duration of illness and neuroleptic treatment. The potential confounding effect of general slowing of reaction time was also examined.

Results

Thirty-six studies were located. This number included two studies that both reported two experiments on separate samples of patients and controls[14,38] and three unpublished PhD theses.[10,33,37] The studies are summarised in the online data supplement, which shows sample sizes, values for mean patient age and duration of illness, the SOA or SOAs used and how patients were dichotomised into those with and without thought disorder. Some studies additionally measured performance on a variety of neuropsychological tests but this information is not shown because of the relatively small number of such studies. All studies except three[18,12,21] employed diagnostic criteria for schizophrenia.

In two studies the standard deviations reported for priming appeared to be standard errors of the mean, based on the fact that they were much smaller than the means (standard deviations for difference scores in priming studies are typically as large as or larger than the means). The authors of one of these studies agreed that this was the case,[16] and in the other this interpretation was supported by calculating the effect size from other data provided in the paper.[30]

Priming in schizophrenia

For this analysis, when studies examined two or more groups of patients with schizophrenia (e.g. patients with and without thought disorder), these were combined. Similarly, when studies examined priming at two or more SOAs, the effect sizes were averaged. A positive effect size indicates that priming is increased.

The pooled effect size from the 36 studies was non-significant at 0.07 (95% CI −0.02 to 0.16). The data were significantly heterogeneous ($Q(35)=59.82$, $P=0.008$), but homogeneity was achieved by excluding two studies with outlying effect sizes. This made little difference to the effect size (pooled $d=0.08$, 95% CI −0.02 to 0.17). Excluding the three studies which used pronunciation rather than lexical decision[20,16,29] also made little difference (pooled $d=0.07$, 95% CI, −0.02 to 0.17).

A funnel plot of the studies is shown in Fig. 2a and does not suggest publication bias.

Semantic priming in patients with thought disorder

The pooled effect size for 18 studies which compared patients with schizophrenia and thought disorder with normal controls was 0.16 (95% CI 0.01 to 0.31), indicating significantly increased priming. These studies were again heterogeneous ($Q(17)=52.31$, $P<0.0001$) and the effect size climbed further to 0.38 (95% CI 0.21 to 0.55) when five studies with outlying effect sizes were excluded. As shown in Fig. 2b, the funnel plot of these studies was asymmetrical, but it suggested a lack of small studies with positive findings, rather than the pattern typical of publication bias, where there is an absence of small studies with negative findings. Also, it should be noted that this analysis did not include the study of Manschreck et al,[3] because an effect size could not be extracted from any of the data in the paper. This study had a small sample size and positive findings and so would have tended to make the funnel plot more symmetrical.

In contrast, pooling the 14 studies which compared patients without thought disorder with controls yielded an effect size of 0.00 (95% CI −0.15 to 0.16) for semantic priming. These studies were not significantly heterogeneous ($Q(13)=21.29$, $P=0.07$).

Thirteen studies allowed comparisons between patients with and without thought disorder. The pooled effect size was 0.06 (95% CI −0.12 to 0.24). This was non-significant, but once again the findings were heterogeneous ($Q(12)=28.79$, $P=0.004$). The
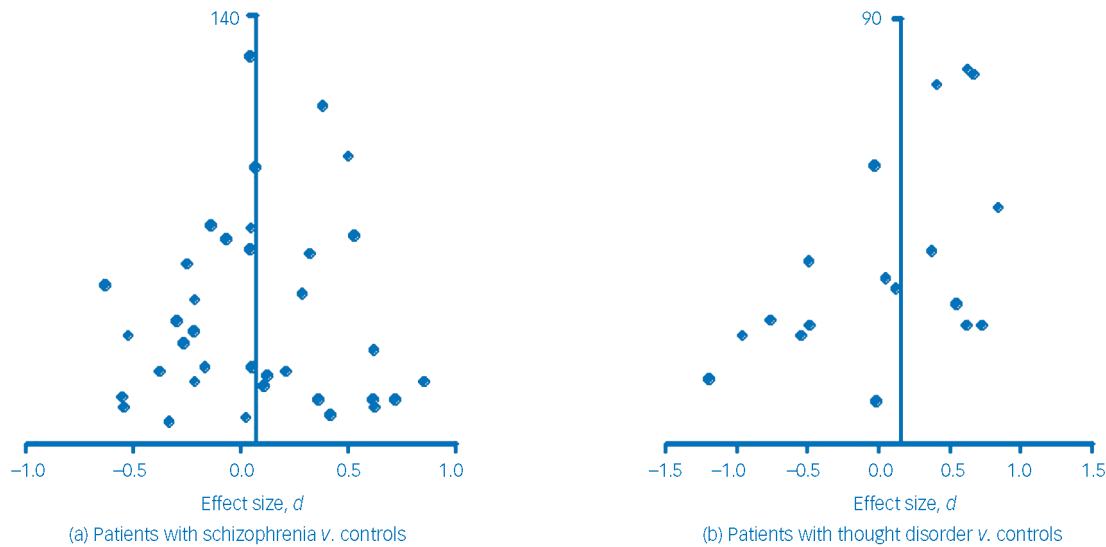
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Fig. 2 Funnel plot of effect sizes for semantic priming in studies of schizophrenia and studies of patients with thought disorder

value increased after excluding two outliers (pooled effect size=0.16, 95% CI −0.02 to 0.35) reaching trend level (*P*=0.08).

Indirect semantic priming

In this version of the semantic priming paradigm the target words are only indirectly related to the prime, usually via a mediating word, for example, *lemon – sweet* (mediating word sour), *black – chalk* (mediating word white). This experimental design aims to examine the hypothesis that activation of associations is not just greater than normal in schizophrenia, but that it extends to more distant associations.

The pooled effect size for nine studies which employed an indirect semantic priming condition was 0.19 (95% CI 0.03 to 0.36), a significant increase. These studies were homogeneous ($Q(8)=4.67$, *P*=0.80). Six of these studies included comparisons of patients with thought disorder with controls, and in these the pooled effect size was greater (pooled *d*=0.56 (95% CI 0.31 to 0.80). These studies were also homogeneous ($Q(5)=7.74$, *P*=0.17).

Stimulus onset asynchrony as a moderator variable

The studies employed a wide range of SOAs, from zero (i.e. the prime and target were presented simultaneously) to 1500 ms. In order to examine this factor, studies were coded as employing a short SOA (⩽400 ms) or long SOA (>400 ms).

Schizophrenia as a whole

The pooled effect size for 23 studies with short SOAs was 0.09 compared with 0.00 in 22 studies using long SOAs. This difference was not significant ($QB(1)=1.30$, *P*=0.25). The larger number of studies in the analyses reflects the fact that some studies tested their subjects at both short and long SOAs, or at multiple SOAs.

We was also examined SOA as a continuous variable. This analysis indicated that effect sizes tended to become more negative with increasing SOA, but once again the effect was not significant (for 45 studies $Z=-1.066$, *P*=0.29). A plot of the effect size for priming against SOA is shown in Fig. 3 and, despite the lack of significance, suggests that there may be a more complex pattern of interaction. At very short SOAs (0–200 ms), there is little

evidence of increased priming in schizophrenia. As SOA increases beyond 200 ms, positive effect sizes start to appear among the negative ones. After around 600–800 ms, negative effect sizes are in the majority, and they then incline back towards 0 at ⩾1000 ms.

Patients with thought disorder

When compared with normal controls, the pooled effect size for ten studies with a short SOA was 0.25 compared with −0.14 in seven studies with a long SOA; this difference was significant ($QB(1)=6.33$, *P*=0.01). When patients with thought disorder were compared with those without thought disorder the difference was significant at trend level (pooled *d* for eight studies with short SOA and six studies with long SOA=0.15 *v.* −0.17 respectively; $QB(1)=3.39$, *P*=0.06).

Other moderator variables

Among the other moderator variables examined, age was not significant ($Z=0.31$ in 34 studies, *P*=0.76). Duration of illness was
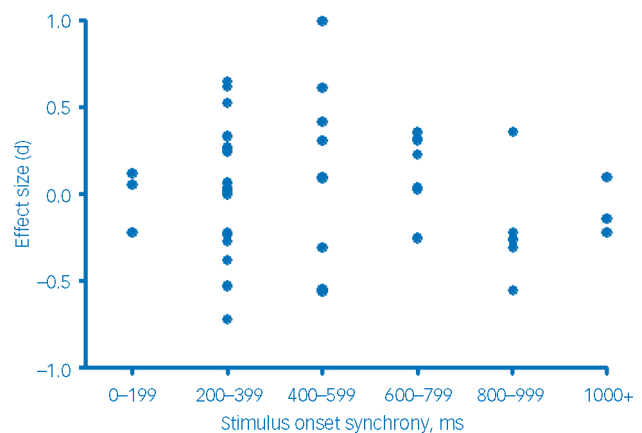

Fig. 3 Plot of studies of semantic priming in schizophrenia as a function of stimulus onset asynchrony (SOA). Two studies where the prime was self-terminated by the subject after a minimum period of time were excluded: Aloia *et al*,[20] 1998; SOA ⩾350 ms, effect size =−0.17; Baving *et al*,[36] 2001; SOA ⩾800 ms, effect size=0.85

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also not significant ($Z=-0.97$ in 26 studies, $P=0.33$) – priming tended to be greater with a shorter length of illness, but nowhere near significantly so. Only four studies were carried out on unmedicated patients, or included a subsample of unmedicated patients, and so it was not considered appropriate to examine medication status as a moderator variable.

The effect of overall slowing

It is universally accepted that reaction time is slower than normal in patients with schizophrenia.[43] However, this in itself will tend to inflate the value for priming due to a simple arithmetical artefact: the difference between a mean reaction time of, say, 900 ms in the unprimed condition and 600 ms in the primed condition is numerically greater than that for the difference between, say, values 600 ms and 400 ms in controls, even though the proportional increase is the same.

To examine this potential confounding effect, a value for general slowing of reaction time in schizophrenia was first calculated for each study. This was taken as the difference between schizophrenic and control means in the unprimed (unrelated word) condition. If data for the unprimed reaction time was not available, reaction time across both unprimed and primed conditions was used. This was standardised across the studies by converting it to an effect size and this was then entered as a moderator variable in the analysis. For obvious reasons, studies which reported percentage priming were not included in the analysis.

General slowing of reaction time was a significant moderator of effect size in schizophrenia as a whole (for 29 studies $Z=2.82$, $P=0.004$), with the positive sign indicating that the greater the slowing the greater the amount of priming. This also held true in the comparison between patients with thought disorder and controls (for 16 studies $Z=3.23$, $P=0.001$).

Discussion

The proposal that associative processes in semantic memory are pathologically altered in schizophrenia has now been tested in over 30 studies using the semantic priming paradigm. Meta-analysis of these studies provides no evidence to support the view that priming (at least direct semantic priming) is increased or decreased in the disorder as a whole, but there is support, albeit qualified, for increased priming in patients with thought disorder. This increase is seen particularly at short SOAs, consistent with an underlying mechanism of increased spread of activation.

Considering first priming in schizophrenia as a whole, the negative findings of our meta-analysis broadly mirror the results of Minzenberg *et al*'s 'vote counting' review which found that the studies were approximately evenly divided into those reporting increased, normal and reduced semantic priming.[7] However, they also commented that studies which employed long SOAs were relatively consistent in finding reduced priming. A similar pattern was discernible in our plot of effect sizes against SOA in which slightly more studies had positive than negative values for priming at short SOAs, but from around 600–800 ms negative effect sizes became increasingly the rule. Furthermore, at least some of the strategic or 'controlled' processes that have been proposed to take place at longer SOAs require a degree of conscious attention and effort,[2] and impairment might, therefore, be expected in schizophrenia. Nevertheless, appearances may be deceptive: the two meta-analytic procedures we carried out provided no grounds to support reduced semantic priming at long SOAs in schizophrenia as a whole.

In relation to thought disorder, Minzenberg *et al*[7] again found that the studies were divided among those finding increased, normal and decreased priming and concluded that 'it is presently unclear how semantic priming disturbances (should they be reliably demonstrated) may be related to thought disorder as manifested clinically'. Meta-analysis, by contrast, yields clearer results here: the effect size for priming in patients with thought disorder compared with controls was small but significant at 0.16 rising to 0.38 in a homogeneous set of studies. This finding could be considered to be strengthened by the facts that: (a) the comparison of patients without thought disorder with controls found no increase in priming; and (b) the meta-analysis of smaller sets of studies of indirect semantic priming also found that the effect size was increased, with the rise being particularly marked in patients with thought disorder. However, it is weakened again by the fact that the analysis comparing patients with and without thought disorder had ambiguous results: the pooled effect size was positive, but only substantially so after outliers were excluded and even then it still did not reach significance, although by this time there were relatively few studies.

Another finding cautioning against acceptance of increased semantic priming in patients with thought disorder is that general slowing of reaction time significantly moderated the effect size. In other words, meta-analysis fails to exclude the possibility that some or all of the differences found merely reflect the fact that patients with schizophrenia have slower than normal reaction times. This does not automatically invalidate the conclusion that semantic priming is increased in patients with thought disorder – this confounding factor does not apply to the comparison of patients with *v.* patients without thought disorder, which had results in the same direction, although not reaching significance – but it does mean that it needs to be addressed in future studies. One simple remedy would be to use percentage gain as the index of priming, or the regression-based correction suggested by Chapman *et al*,[44] rather than a subtractive measure. Two studies have avoided the problem altogether by examining errors rather than slowing of reaction time. Kwapil *et al*[45] presented a prime followed by a visually degraded target and used accuracy of identification (by pronunciation) as the measure of semantic priming. They found that patients with schizophrenia unselected for presence of thought disorder showed more than twice the priming shown by the controls. In contrast, Quelen *et al*[46] found no increase in priming in unselected patients with schizophrenia, but in this study there was an association between increased priming and presence of thought disorder.

If semantic priming is increased in patients with schizophrenia and thought disorder, the effect is seen predominantly at short SOAs. This finding thus supports an interpretation in terms of an increase in the automatic element of the processes underlying priming in the lexical decision task, that is, increased spread of activation in semantic memory. How might this lead to thought disorder being manifested clinically? According to network theories of semantic memory, when nodes are activated, for example by hearing or reading words, the activation spreads to other nodes for words conceptually associated with them. Maher plausibly argued that the same process takes place when an individual is speaking and thinking about what to say next.[1] This would then cause activation of nodes which were only distantly related to the topic of discourse, and in such circumstances it could become difficult to prevent the intrusion of irrelevant associations which, as he put it, 'lie like a web of distractions around each element in the sentence'. In Maher's words, depending on the severity of the disturbance, the result would be speech that was either merely richer in associations than usual or, at the other end of the spectrum, was strewn with multiple intrusions which seriously compromised its intelligibility.[1]

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The central finding of this meta-analysis is that increased semantic priming may be a psychological mechanism underlying thought disorder but is not relevant to the wider clinical picture of the disorder. In some ways, this conclusion echoes the changing status of the symptom of thought disorder in schizophrenia over the years. Originally, Bleuler considered association disturbance to be one of the fundamental symptoms of schizophrenia, 'present in every case and at every period of the illness'.[47] Uncritical acceptance of Bleuler's views, particularly in the USA, led to thought disorder being considered central to the understanding of the disorder, as well as a certain way to distinguish patients with and patients without schizophrenia clinically. Eventually, however, studies began to cast doubt on its universality and also made it clear that the symptom could be seen in mania and probably other disorders as well.[48,49] Following influential work by Andreasen,[50] thought disorder is now regarded as a relatively uncommon symptom in schizophrenia, which broadly speaking can be either present or absent in the same way as other symptoms, such as auditory hallucinations or first-rank symptoms. Nevertheless, unlike these symptoms, it is widely believed that thought disorder can also be present 'subclinically' and can be detected in a greater proportion of patients than those in whom it is clinically obvious when special procedures such as interpreting proverbs are used to elicit it.

This meta-analysis touches on two final issues of relevance to semantic priming in schizophrenia. First, Maher *et al*[51] found evidence that priming changes from hyperpriming to hypopriming with increasing duration of illness, a finding which is of considerable potential significance given the important clinical differences between patients with acute and chronic illness. This meta-analysis, however, found no evidence to support such an association. Second, dopaminergic mechanisms regularly feature in theoretical analyses of semantic priming in schizophrenia.[52,53] It is, therefore, somewhat disappointing to find that the vast majority of studies have restricted their examination to patients treated with antipsychotics. This meta-analysis suggests that while there is clearly scope for further investigation of priming in schizophrenia at least some future studies should be carried out on patients who are drug-free.

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Author: Please state which department] (Ref 37, Column 1) (AQ8)

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Data supplement. Studies included in the meta-analysis

First author and date	Patient n	Control n	Mean patient age, years	Mean duration, years	Stimulus onset asynchronies	Measure of priming	Measure of thought disorder
Meyer 1991[10]	25	25	36.7	13.3	317	Subtractive	TDI highest and lowest scores
Chapin 1992[11]	45	15	–	–	0	Subtractive	–
Spitzer 1993[4]	50	50	32.3	–	200	Subtractive	BPRS <3 v.
					700	Percentage gain	=3
Spitzer 1994[12]	70	44	32.8	–	200	Subtractive	BPRS <4 v.
					400		=4
					700		TD
Blum 1995[13]	18	9	38	–	350	Subtractive	Unspecified scores on TLC
Henik 1995[14] (experiment 1)	16	16	34.5	–	Data not usable	Subtractive	Patients with TD only based on unspecified SADS score
Henik 1995[14] (experiment 2)	16	16	38	–	Data not usable	Subtractive	Patients with TD only based on unspecified SADS score
Ober 1995[15]/Vinogradov 1992[5]	16	21	32	8	250	Subtractive	—
Barch 1996[16]	100	28	33.5	9.2	200	Subtractive	BPRS =3 v. <3
					300	Also used regression	
					450	based correction	
					700		
					950		
Besche 1997[17]	34	20	33.7	11	500	Subtractive	TLC total =7 v. >7 check
Ober 1997[6]	31	20	40	—	250	Subtractive	–
					1000		
Passerieux 1997[18]	22	11	27.75	6.2	500	Subtractive	TLC total <7 v. =7
Spitzer 1997[19]	20	20	32.5	—	250	Subtractive	–
						Percentage gain	
Aloia 1998[20]	20	21	35.8	17	=350	Percentage gain	TLC global =2 v. >2
Weisbrod 1998[21]	40	38	—	6.9	250	Subtractive	BPRS =3 v. <3
						Percentage gain	
Barch 1999[22]	56	25	38.3	16.7	300	Subtractive	–
					950		
Condray 1999[23] & 2003[24] (patients on medication only)	30	34	38.5	13.2	350	Subtractive	–
					950		
Rossell 2000[25]	42	28	34	10.9	700	Subtractive	Patients without TD only based on SAPS score =3
Baving 2001[26]	17	20	31.7	5.9	=800	Subtractive	–
Moritz 2001[27]	44	30	31.8	7.5	200	Subtractive	PANADSS <3 v. =3
Surguladze 2002[28]	20	26	34	8.6	400	Subtractive	–
						Percentage gain	
Moritz 2003[29]	32	65	32.53	4.85	200	Subtractive	PANADSS <3 v. =3
						Also used regression based correction	
Besche-Richard 2003[30]	15	15	34.9	–	500	Subtractive	Patients with TD only based on TLC total >7
Gouzoulis-Mayfrank 2003[31]	53	20	32.2	4.7	700	Percentage gain	SAPS median split
Hokama 2003[32]	18	18	28.9	7.3	1500	Subtractive	–
Irwin 2003[33]	20	20	41.3	–	300	Subtractive	–
					900	Percentage gain	
Minzenberg 2003[34]	57	20	40.2	16.2	250	Subtractive	–
					1000		
Chenery 2004[35]	14	12	33	8.21	Data not usable	Subtractive	–
Rossell 2004[36]	40	40	39.2	15.7	700	Subtractive	–
Leeson 2004[37]	32	16	44	22	200	Subtractive	CASH global TD =2 v. >2
Besche-Richard 2005[38] (experiment 1)	21	20	31	9	0	Percentage gain	Patients with TD only based on TLC >7
Besche-Richard 2005[38] (experiment 2)	19	20	34.7	10.5	0	Percentage gain	Patients with TD only based on TLC >7
Nestor 2005[39]	14	14	42.8	18.6	575	Subtractive	–
						Percentage gain	
Lecardeur 2006[40]	15	15	36.3	12.2	250	Subtractive	–
					500		
Rossell 2006[41]	30	32	36.4	13.6	250	Percentage gain	–
					750	Subtractive	
Kuperberg 2007[42]	17	15	45.1	19	800	Subtractive	–

Author:
Where more than one value, should this be shown as a range i.e. 200–950?]
(AQ10)

Author:
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Author:
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