Psychological Medicine, 2004, **34**, 1365–1369. © 2004 Cambridge University Press
Printed in the United Kingdom

CORRESPONDENCE

Psychological Medicine, **34** (2004).
DOI: 10.1017/S0033291704213848

To the Editor:

Pilling and colleagues (2002) recently published a meta-analysis examining the effects of social skills training on schizophrenia (this review also included a meta-analysis of research on cognitive remediation for schizophrenia which is not discussed in this comment). The stated advantages of this review over previous ones were that it considered only randomized controlled trials (RCTs), and that it focused only on studies of patients with schizophrenia-spectrum diagnoses. The authors' concluded that 'there was no clear evidence for any benefits for social skills training on relapse rate, global adjustment, social functioning, quality of life or treatment compliance' (p. 783) and that 'social skills training and cognitive remediation do not appear to confer reliable benefits for patients with schizophrenia and can not be recommended for clinical practice' (p. 783). In this commentary we point out that the empirical basis for these conclusions is insufficient and that the authors' review of the research literature on social skills training was both ill-informed and misleading.

A look at the evidence

As discussed in greater detail by Bellack (in press), the research literature on social skills training for schizophrenia and other severe mental illnesses has been reviewed multiple times over the past 15 years (e.g. Donahoe & Driesenga, 1988; Benton & Schroeder, 1990; Corrigan, 1991; Halford & Hayes, 1991; Scott & Dixon, 1995; Dilk & Bond, 1996; Penn & Mueser, 1996; Smith *et al*. 1996; Wallace, 1998; Heinssen *et al*. 2000). While there are minor differences in the conclusions of these reviews, by and large they are consistent with respect to several outcomes. Specifically, most reviews conclude that skills training has few or no effects on symptom severity or relapses and rehospitalizations, but does have modest effects on social functioning. These findings are not in agreement with Pilling and colleagues, who concluded that social skills training has no effect on social functioning, quality of life, or global adjustment. However, an examination of the empirical basis for Pilling *et al*.'s conclusions casts serious doubts on their claims.

Pilling *et al*. examined a total of nine studies of social skills training, but of these studies only one evaluated social functioning (Marder *et al*. 1996), one evaluated quality of life (Liberman *et al*. 1998), and only two examined global adjustment (Lukoff *et al*. 1986; Hayes *et al*. 1995). The one study that actually examined the effects of skills training on social functioning reported significant effects (Marder *et al*. 1996). Pilling *et al*.'s conclusion that skills training had no effect on social functioning was due in part to the fact that another study reported that skills training had no effect on social skills (Hayes *et al*. 1995). However, social skills should not be confused with social functioning; the former refers to the cognitive, verbal, and non-verbal behaviors used during interactions with others, whereas the latter refers to the actual meeting of instrumental and affiliative needs (Mueser *et al*. 1990; Penn *et al*. 1995).

The one study that examined the effects of skills training on quality of life actually produced mixed findings (Liberman *et al*. 1998), with significant improvements reported on one measure of quality of life but not another. The two studies that evaluated the effects of skills training on global adjustment (Lukoff *et al*. 1986; Hayes *et al*. 1995) both employed rigorous control interventions that conceivably had beneficial effects on overall functioning.

Pilling and colleagues' interpretations

The foregoing discussion of the evidence included in Pilling *et al*.'s review clearly indicates that there is an insufficient number of RCTs examining the effects of skills training on the social functioning, quality of life, or overall adjustment in patients with schizophrenia to

https://doi.org/10.1017/S0033291704213848 Published online by Cambridge University Press

draw any firm conclusions. However, rather than acknowledging this, Pilling *et al*. concluded that 'it is hard to recommend the use of social skills training in routine clinical practice' (p. 789) and 'the unimpressive results from our analysis certainly make a case that requires to be answered by clinicians who support the use of social skills training' (p. 790).

These conclusions do not consider the severe limitations of the research studies upon which their review was based. Rather than acknowledging these limitations and calling for more controlled research on skills training for schizophrenia, Pilling *et al*. suggested that their findings differ from previous reviews of the literature because they employed more methodologically rigorous meta-analytic techniques. In truth, practically all other reviews of the social-skills training literature have agreed with the conclusions of Pilling *et al*. concerning the lack of effects of skills training on symptoms and relapses; the disagreement lies in the area of psychosocial functioning, which was examined in only one or two studies included in Pilling *et al*.'s review. In addition, Pilling *et al*. suggested the need to reformulate the social-skills training model to improve its effectiveness. It is unclear why the authors believe the model needs modification when there is a paucity of controlled research that has examined the intended effects of social skills training on psychosocial functioning.

In their discussion, Pilling and colleagues provided a spirited challenge to clinicians to justify their use of social skills training in light of the weak data supporting it. This challenge fails to appreciate either the difficulties of improving social functioning in schizophrenia or the lack of good alternative treatments. Impaired social functioning is a defining characteristic of schizophrenia, one which often predates the onset of the illness by many years (Zigler & Glick, 1986), and is stable over time (Leary *et al*. 1991). In contrast to psychotic symptoms and relapse rates, which are readily modified by a variety of interventions such as medication (Davis *et al*. 2003), family therapy (Pitschel-Walz *et al*. 2001), training in relapse prevention skills (Mueser *et al*. 2002), and cognitive behavior therapy (Gould *et al*. 2001), there are no established interventions that consistently improve social functioning in schizophrenia. Clinicians' selection of treatment strategies needs to be based not only on the strength of research supporting a particular intervention, but the availability of other interventions that improve the same area of functioning. In the case of social functioning, while the data supporting the effects of social skills training are modest, few would argue that there are better candidate treatments for improving the pernicious deficits in this area.

Conclusions

The most sweeping conclusions reached by Pilling *et al*. regarding the effects of social skills training on social functioning, quality of life, and general functioning were based on an insufficient number of studies. There was also no attempt to reconcile the conclusions of this meta-analysis with the numerous other reviews of the skills-training literature that have reached very different conclusions. The consequences of Pilling *et al*.'s rush to judgment are not trivial. For example, clinicians may avoid using skills training for fear of it not being an 'evidence-based' practice, thus depriving their clients of a potentially effective treatment, and research on skills training may be squashed as funding agencies shy away from interventions shown to be 'ineffective'. An important responsibility of reviewing a clinical research area is to provide a fair, balanced, and accurate evaluation of a treatment approach. Pilling and colleagues failed to objectively review the research literature on social skills training. Their conclusions are not supported by the research, and thus are not valid.

Declaration of Interest

None.

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Psychological Medicine, **34** (2004).
DOI: 10.1017/S0033291704223844

To the Editor:

We read with great interest the article by Nielen & Den Boer (2003), who found that patients with obsessive–compulsive disorder (OCD) displayed cognitive deficits consistent with a dysfunction of the dorsolateral–striatal circuit (DLSC) (i.e. impairments in planning ability, spatial memory, and motor speed). According to the report, the 'successful' treatment of patients with OCD with fluoxetine did not alter cognitive functions 'to any significant degree'. The authors argued that cognitive impairments in OCD may form a trait-feature of the disorder and that fluoxetine produces its clinical effects by acting on a neural system whose cognitive functions were not measured in their study (presumably those subserved by the orbitofrontal–striatal circuit).

There are, however, a certain number of empirical findings that apparently challenge the authors' conclusions regarding the relatively minor role played by the DLSC in the treatment of OCD with serotonin reuptake inhibitors (SRIs). Hollander & Wong (1996) found that impairment in cognitive functions presumably subserved by the DLSC (Trail Making Test B – A) are associated with a blunted prolactin response to m-CPP (a probe for serotonergic function) in patients with OCD. A recent study reported that cognitive deficits that are suggestive of a dysfunction in the DLSC (verbal fluency-letters) in patients with OCD may be state-related and, therefore, more amenable to treatment (Kim *et al.* 2002). Abbruzzese *et al.* (1995) found that patients with OCD treated with fluvoxamine exhibited a better performance in the WCST (a test thought to tap the DLSC) as compared to their unmedicated counterparts. Finally, at least one study (Fontenelle *et al.* 2001) observed that a poorer performance in the WCST in patients with OCD

https://doi.org/10.1017/S0033291704213848 Published online by Cambridge University Press

was associated with a better therapeutic response to SRIs.

In our opinion, it would be counter-intuitive to expect that patients with OCD who have not responded to treatment with fluoxetine (44% of the total) would exhibit significant improvements in their neuropsychological performance. Instead of investigating the OCD group as a whole (responders and non-responders), it would be interesting if Nielen & Den Boer could focus their analysis in the treatment-responders group. Did this group of patients display improvements in their cognitive function, while the treatment non-responders did not? We believe that this kind of analysis might provide us with additional relevant findings. Maybe the authors can take a second look at their data under this perspective.

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The Authors reply:

Fontenelle, Mendlowicz and Versiani make a plausible case for the necessity to analyse not only the OCD group as a whole, but also to compare responders and non-responders to treatment in a separate analysis. In view of their comments we have taken up their suggestion to re-analyse our data. As described in the original paper (Nielen & Den Boer, 2003), responders ($n=12$) were defined as patients with a minimum reduction of 40% on the total score on the Y-BOCS.

Performance on the four executive tasks (SWMT, TOL, IDED and Stroop) was re-analysed with repeated-measures ANOVA using session as the within-subject factor. With respect to the between-subject factor 'group', we compared performance of RESP and NRESP. In case of significant differences between these OCD subgroups, we subsequently tested whether responders differed from the normal controls.

For measures on the SWMT (total between-errors and strategy score), TOL accuracy (number of perfectly solved solutions and total number of excess moves), TOL latency, and ID-EDS (number of trials on ID and ED stage) there were no significant group × session interactions or main effects. Only for the Stroop task (level of interference) did we find a main effect of group, indicating that non-responders were in general more susceptible to interference ($F_{1,16}=5{\cdot}56$, $p=0{\cdot}031$) than responders. When we subsequently compared performance of responders with that of normal controls, there was a significant group × session interaction ($F_{1,16}=5{\cdot}93$, $p=0{\cdot}020$). However, this just seems to replicate the subtle interaction effect that was already present in the whole OCD sample (Nielen & Den Boer, 2003). In other words, re-analysing the data by comparing performance of responders and non-responders, and responders with normal controls did not essentially alter the findings we already observed in the entire OCD sample.

In their critique, Fontenelle and colleagues propose that the DLPFC plays a more important role than is suggested by our findings (Nielen & Den Boer, 2003). They argue that there are several empirical studies supporting an association between DLPFC function and treatment response in OCD. However, we are

https://doi.org/10.1017/S0033291704213848 Published online by Cambridge University Press

not quite sure whether the findings of these studies unequivocally demonstrate a direct relationship between DLPFC function and therapeutic response to an SRI. First, except from our own report, it was only Kim *et al*. (2002) who *directly* investigated the effect of pharmacological treatment on neuropsychological performance. In this study, there was an effect of treatment on the COWA-letter task, however, it should be added that this task does not seem to be a very specific marker of DLPFC function. For instance, COWA-letter fluency has been reported to recruit medial and orbital prefrontal regions as well (Phelps *et al*. 1997; Kim *et al*. 2002; Ravnkilde *et al*. 2002). In addition, directly manipulating activity in the central serotonergic system of remitted depressive patients has been shown to affect neural activity elicited by verbal fluency tasks (Smith *et al*. 1999). Serotonin is increasingly associated with the functions of medial and orbital PFC, and not DLPFC (Robbins, 2000), so this makes it less likely that verbal fluency is exclusively linked to the DLPFC.

In their own study, Fontenelle and colleagues investigated the relationship between treatment and performance on tasks for the DLPFC rather indirectly. That is, Fontenelle *et al*. (2001) associated baseline WCST performance to treatment outcome but it was not quite clear whether the two groups of responders and non-responders were carefully matched before they entered treatment. For instance, performance on the WCST is significantly influenced by factors such as education or the presence of depressive symptoms (Gambini *et al*. 1992; Beats *et al*. 1996). Unfortunately, Fontenelle *et al*. (2001) do not report whether responders displayed comparable levels of education or depressive symptoms as non-responders. In our opinion, this hampers firm conclusions about the significance of the reduced WCST performance in OCD responders.

Taken together, we believe that there is, as yet, no strong evidence in the literature for a prominent role of the DLPFC in the treatment of OCD. This conclusion is supported by our own data showing no differential performance of responders and non-responders on tasks for DLPFC function.

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https://doi.org/10.1017/S0033291704213848 Published online by Cambridge University Press