Improved social functioning following social recovery therapy in first episode psychosis: do social cognition and neurocognition change following therapy, and do they predict treatment response?


This version is available from Sussex Research Online: http://sro.sussex.ac.uk/id/eprint/97708/

Copyright and reuse:
Sussex Research Online is a digital repository of the research output of the University.

Copyright and all moral rights to the version of the paper presented here belong to the individual author(s) and/or other copyright owners. To the extent reasonable and practicable, the material made available in SRO has been checked for eligibility before being made available.

Copies of full text items generally can be reproduced, displayed or performed and given to third parties in any format or medium for personal research or study, educational, or not-for-profit purposes without prior permission or charge, provided that the authors, title and full bibliographic details are credited, a hyperlink and/or URL is given for the original metadata page and the content is not changed in any way.

http://sro.sussex.ac.uk
IMPROVED SOCIAL FUNCTIONING FOLLOWING SOCIAL RECOVERY THERAPY IN FIRST EPISODE PSYCHOSIS: DO SOCIAL COGNITION AND NEUROCOGNITION IMPROVE FOLLOWING THERAPY, AND DO THEY PREDICT TREATMENT RESPONSE?

Running Head: Cognition and functional improvement in psychosis

Siân Lowri Griffiths PhD*1, Prof Stephen J. Wood PhD1,2,3, Prof David Fowler PhD4, Prof Nick Freemantle PhD5, Joanne Hodgekins, PhD6, Prof Peter B Jones PhD7, Prof Swaran Singh PhD9, Prof Vimal Sharma PhD10,11 & Prof Max Birchwood PhD9

1Institute for Mental Health, University of Birmingham, Birmingham, UK; 2Orygen, the National Centre of Excellence in Youth Mental Health, Melbourne, Australia; 3Centre for Youth Mental Health, University of Melbourne, Victoria, Australia; 4Psychology Department, University of Sussex, Brighton, UK; 5Institute for Clinical Trials and Methodology, University College London, London, UK; 6Norwich Medical School, University of East Anglia, Norwich, UK; 7University of Cambridge, Cambridge, UK 8University of Warwick, Coventry, UK; 9University of Chester, Chester, UK; 10Cheshire and Wirral Partnership NHS Foundation Trust, Chester, UK

Word Count: Text Body 3,996; Abstract 239.

*Correspondence: Siân Lowri Griffiths; Institute for Mental Health, University of Birmingham, Edgbaston, Birmingham, UK, B15 2TT; email: s.l.griffiths@bham.ac.uk; Tel: +44 7912497267.
Abstract

There is a need to develop and refine psychosocial interventions to improve functioning in First Episode Psychosis (FEP). Social cognition and neurocognition are closely linked to functioning in psychosis; examinations of cognition pre- and post- psychosocial intervention may provide insights into the mechanisms of these interventions, and identify which individuals are most likely to benefit.

Method: Cognition was assessed within a multi-site trial of Social Recovery Therapy (SRT) for individuals with FEP experiencing poor functioning (<30h weekly structured activity). Fifty-nine participants were randomly allocated to the therapy group (SRT + Early intervention), and 64 were allocated to treatment as usual group (TAU - early intervention care). Social cognition and neurocognition were assessed at baseline and 9 months; assessors were blind to group allocation. It was hypothesized that social cognition would improve following therapy, and those with better social cognition prior to therapy would benefit the most from SRT.

Results: There was no significant impact of SRT on individual neurocognitive or social cognitive variables, however, joint models addressing patterns of missingness demonstrate improvement across a number of cognitive outcomes following SRT. Further, regression analyses showed those who had better social cognition at baseline were most likely to benefit from the therapy ($\beta = .350; 95\% \text{ CI} = .830 \text{ to } 8.891; p = .019$).

Conclusion: It is not clear if SRT impacts on social cognitive or neurocognitive function, however, SRT may be beneficial in those with better social cognition at baseline.

Keywords: Cognition; Functioning; Psychosocial intervention; Psychosis; Social disability.
1. Introduction

Many young people with first episode psychosis (FEP) have persisting difficulties with their social functioning, even when receiving high quality care under an Early Intervention Service (EIS; Hodgekins et al., 2015a). This highlights the need for new interventions to target social impairments in early psychosis.

Widespread impairments in social cognition, defined as the mental operations underlying social interactions (Adolphs, 2008), and neurocognition (e.g. memory), are evident in psychosis and are closely associated with functional impairments (Allott et al., 2011a; Couture et al., 2006). Whilst there are number of evidence-based psychosocial interventions targeting poor functioning in psychosis, the impact of these interventions on cognitive impairment is not well established (Kurtz, 2011). There is also considerable heterogeneity in therapy response (Fizdon et al., 2020). Thus, to ensure that psychosocial interventions are being delivered appropriately, it is important to understand which factors contribute to change, and identify individuals who are most likely to benefit (Allott et al., 2011b).

Neurocognitive variables have been identified as robust predictors of treatments response to a number of psychosocial interventions, demonstrating that poorer neurocognitive function prior to intervention has a rate-limiting impact on treatment progress (Kurtz, 2011; Kurtz and Mueser, 2008). Verbal memory, working memory, executive functions, problem-solving and attention have been shown to predict progress on proximal measures designed to assess acquisition of specific therapy-related skills following: social skills training (Kern et al., 1992; Kurtz and Mueser, 2008), cognitive remediation (Fiszdon et al., 2020), and vocational rehabilitation (Bell and Bryson, 2001). The importance of baseline neurocognitive variables in predicting change in more distal outcomes has also been demonstrated. Verbal memory and
executive function were shown to predict long-term competitive employment outcomes following a supported employment programme (McGurk and Mueser, 2006), and composite neurocognition and social cognition predicted improvement in functioning following community rehabilitation (Brekke et al., 2007). Studies have failed to demonstrate a moderating influence of neurocognition on outcomes in response to Cognitive Behavioural Therapy (CBT) for psychosis, however, in these studies, the primary outcome was positive symptom reduction as opposed to improvement in functioning (Allott et al., 2011a; Garety et al., 1997).

Social cognition and neurocognition are also shown to improve following psychosocial interventions. In a meta-analysis evaluating a social cognition training programme, large effect sizes were found for facial affect recognition, moderate-sized effects for Theory of Mind (ToM), and small to medium effects for attribution style, in addition to moderate to large effects for community functioning (Kurtz et al., 2016). A meta-analysis has also reported on lasting improvements in social cognition, neurocognition, and functioning following and integrative psychosocial intervention with adjunctive cognitive remediation (Roder et al., 2006; Roder et al., 2011). These findings may demonstrate a reciprocal relationship between social cognition and neurocognition with functional improvement.

Despite these findings, interventions addressing poor functioning have largely focused on those with enduring illness. Interventions delivered earlier in a person’s functional trajectory might be more effective at preventing disability from becoming entrenched (Griffiths et al., 2018). In early psychosis, the effectiveness of a specialised Social Recovery Therapy (SRT) has been demonstrated in a recent randomized controlled trial (Fowler et al., 2017). Individuals’ receiving SRT alongside EI showed an 8.1-hour greater increase of weekly structured activity,
compared to those receiving EIS alone (95% CI 2.5 to 13.7; \( p = 0.005 \)). SRT is designed to motivate individuals to re-engage with their social environment, whilst using CBT techniques, such as cognitive reappraisal strategies, to overcome barriers to successful social engagement (Fowler et al., 2009).

Although cognition was not addressed by the SRT, given the evidence that cognition is implicated in a range of evidence-based psychosocial interventions (Kurtz, 2011), we might expect to see a change in cognition post SRT, or that baseline cognition may limit the progress made via therapy. Further, with regards to the nature of the SRT and reliance on social and interpersonal skills to engage in a therapeutic relationship and re-engage in a social environment, it was hypothesized that social cognition is more likely implicated with SRT. Finally, it was apparent \textit{a priori} that many individuals did not feel ready to return to work, and as such, the focus of the SRT was to encourage engagement in a range of social activities, rather than a return to main roles such as work or education. This, together with evidence showing that progress following vocational interventions is associated with neurocognition (e.g. attention and problem solving, which might be considered important for obtaining employment), we expect that SRT may be less reliant on neurocognitive functions.

Nevertheless, exploration of both social cognition and neurocognition pre- and post-SRT may be important to understand the dynamics of therapy, and explore which individuals are most likely to benefit. No studies to date have examined the impact of cognition on treatment response to a CBT intervention targeting functional impairment in psychosis. This study was designed to add value to the assessment of the effectiveness of SRT in a recent NIHR SUPEREDEN trial (reported elsewhere in Fowler et al. 2017), by addressing two main questions:
(1) Does SRT lead to improvements in social cognition and neurocognition?

(2) Do baseline social cognitive and/or neurocognitive variables predict which individuals are more likely to benefit from the SRT?

2. Method

2.1. Design

This was a sub-study within the multi-site NIHR SUPEREDEN trial, which was a single-blind, proof-of-principle trial, comparing SRT plus EIS care, against standard care from EIS alone (referred to as treatment as usual – TAU). SRT draws on psychological intervention and multi-systemic assertive outreach case management to promote social recovery. The trial is registered (ISRCTN61621571), and further details on the specific therapeutic approach can be found in Fowler et al. (2017). EIS care was provided under four recognised centres of excellence with high fidelity to the EIS model, indicating that the centres provided a comprehensive range of interventions including: CBT for psychosis, family interventions, supported employment, social support, assertive outreach case management, and medical and psychopharmacological management (Fowler et al., 2017).

2.2. Sample

The inclusion criteria were as follows: (1) individuals aged 16-35 years with non-affective psychosis; (2) low level of structured activity after 12 months of EIS [defined as 30 hours or less per week on the Time Use Survey; (Short, 2006)]; (3) receiving EIS care between 12-30 months. Participants were excluded if they were: (1) not proficient in English language to engage in SRT; (2) deemed too unwell to partake; (3) diagnosed with a neurological disorder;
(4) had a learning disability; (5) had a history of severe head injury (more than 5 minutes loss of consciousness, or an overnight hospital stay).

One hundred and fifty-five individuals from four UK EIS centers (Birmingham, Lancashire, Norfolk and Sussex) consented to the trial and 122 completed cognitive assessments. The breakdown of group allocation post-randomization was as follows: 59 participants were allocated to the SRT plus EIS group, and 63 participants were allocated to the TAU group (EIS alone). An analysis comparing those who completed the cognitive assessments at baseline with those who did not (Table 1), showed no group differences on demographics, structured activity and clinical characteristics.

2.3. Measures

2.3.1. Functioning

The primary hypothesis of the trial was that SRT (plus EIS) would lead to improvements in social recovery (assessed as time spent in structured activity at 9 months). Structured activity was assessed using the Time Use Survey [TUS (Short, 2006)]. The TUS measures time spent over the last month in activities such as work, education, socializing, leisure, sports, chores and childcare. The average hours per week spent in ‘structured activity’ was calculated.

2.3.2. Cognitive study measures (Secondary outcome measure)

2.3.2.1. Neurocognition

To minimize participant burden and maximise engagement, a brief neurocognitive battery was selected. Verbal skills are shown to be most impaired in psychosis (Allott et al., 2011b), and as such, the battery included two verbal assessments: verbal learning and memory [Logical
2.3.2.2. Social Cognition

Given the interpersonal nature of the intervention, we hypothesized that SC was more likely to explain response to SRT. We therefore included four social cognition sub-domains most commonly impaired in psychosis: Theory of Mind [Picture sequencing task; (Langdon et al., 2014)]; emotion recognition (Mayer-Salovey-Caruso Emotional Intelligence Test – Perceiving Emotions; MSCEIT; (Mayer, 2002); Attribution of blame bias [Ambiguous Intentions Hostility Questionnaire; AIHQ; (Combs et al., 2007)]; and social perception [The Social Knowledge Questionnaire; SKQ; (Cutting and Murphy, 1990)].

2.4. Study Procedure

Participants were randomly allocated using a computer program to either the treatment group (SRT + standard EIS care) or TAU (standard EIS care). Those allocated to the treatment group received SRT for 9-months by a CBT accredited therapist. Those allocated to TAU group continued to receive standard care under EIS. Participants completed a battery of cognitive assessments in addition to the main trial assessments at baseline (prior to randomization), and at 9-month follow-up. Although all trial participants were given the opportunity to complete the cognitive test battery at baseline, only 79% agreed to complete (see section 2.2.). Researchers were blind to group allocation. Where un-blinding occurred,
assessments were conducted by another researcher blind to group allocation. Consent was obtained on one occasion at the start of the trial. Trial researchers were trained on the use of cognitive assessments and completed regular inter-rater reliability checks in line with the NIHR SUPEREDEN protocol (Fowler et al., 2017). The study was approved by the Black Country NHS research ethics committee (REC reference: 12/WM/009).

2.5. Statistical Analyses

2.5.1. Analysis 1: Impact of SRT on Social Cognition and Neurocognition

To explore the impact of SRT on each of the social cognitive and neurocognitive variables, a mixed 2x2 multivariable Analysis of Variance (MANOVA) was used. To determine a treatment effect, the ‘group x time’ interaction was inspected. A Bonferroni correction was applied to reduce the likelihood of a Family-Wise-Error (Haynes, 2013). Cohen’s $f$ was used as a measure of effect size, with small, medium and large effects corresponding to values of .01, .025, and .04, respectively (Cohen, 1988).

The assumption of multivariable normality was met, and there were no problematic outliers. There was no indication from scatterplots of non-linearity, and there was no evidence of multicolinearity or singularity in the data (tolerance >.2, variance inflation factor <5; Pearson correlations <.7).

In the initial analysis, no imputation was conducted, but due to higher attrition in the control group by follow-up, data was assumed missing not at random. Missingness was addressed using joint multivariate modelling, where the continuous outcome score (using Gaussian error) was modelled with the observed loss to follow-up (with Bernoulli error) to describe the joint probability of the observed outcome. Joint modelling assumes a relationship between the
observed outcome and missing data points via an informative missingness parameter; although such a parameter cannot be estimated from the observed data, it can be specified with associated uncertainty (Chaimani et al., 2018). In this instance, we made the assumption that those who did not remain in the study, had poorer outcomes on the cognitive measures than those who remained. The justification of this assumption is given by the main trial analysis, whereby in this group who uniformly displayed extreme social withdrawal at baseline, by 15-months, the proportion of those lost to follow-up was more than two-times higher in the TAU group compared to the SRT group, implying a benefit on maintenance of engagement via therapy, which was reflected by an improvement on the primary outcome (Fowler et al., 2017). Joint modelling was undertaken here and in the main trial analysis, and was conducted using SAS Proc Glimmix (version 9.4, SAS Institute, CARY, NC).

2.5.2. Analysis 2: Social cognition and neurocognition as predictors of response to SRT

A Change score (difference in hours per week spent in structured activity between pre- and post SRT), was entered as a dependent variable in a linear regression, with social cognitive and neurocognitive scores entered as predictor variables. To handle the large number of variables entered into the regression, a backward method was employed to find an optimal model of the most parsimonious predictors of treatment response (Lindsey and Sheather, 2010). A post-hoc Bonferroni correction was applied in the case of significant regression models (Haynes, 2013).

3. Results

3.1. The Sample: cognitive study

Of the full trial sample, 79% (122) completed cognitive assessments at baseline. Demographic and clinical comparisons between the trial sample and the sub-group completing cognitive assessments (by group allocation) are shown in Table 1. There were no differences on demographic or clinical characteristics between the two groups at baseline.
At 9-month follow-up (post SRT), data were available for 109 of the 122 participants (89%) on the primary outcome (TUS). Not all cognitive assessments were completed by each participant who returned for follow-up. Table 2 provides a breakdown of missing data for each of the cognitive measures at 9-month follow-up. There was higher percentage of missing cognitive data in the control group, which supported an assumption that data were missing not at random (Table 2).

3.2. Analysis 1: Impact of SRT on social cognition and neurocognition

Group by time MANOVAs were conducted for each of the social cognitive and neurocognitive variables. Means and standard deviations are presented in Table 2. The only significant group differences over time was observed for attribution of blame bias (95% CI = 0 to 0.437; p = 0.49), but this did not retain its significance following a Bonferroni correction. There were no other significant differences observed on either of the social cognitive or neurocognitive variables. The group x time interactions are presented in Table 3. Due to the bias in the pattern of missingness (Table 2); the data was regarded as missing not at random. Based on the assumption that loss to follow-up was associated with poor scores on the cognitive measures, results of the joint models indicated significant differences on several of the cognitive variables (Table 3), including: verbal comprehension, non-verbal problem solving, ToM, social knowledge and attribution of blame bias.

3.3. Data Analysis 2: Social cognition and Neurocognition as predictors of response to SRT.

The change score on TUS was entered into a backward linear regression as a dependent variable. The final model included social knowledge and attribution of blame bias, accounting
for 11% of variance (Table 4). Baseline social knowledge was the only significant independent predictor of change in structured activity ($\beta = .350; 95\% \text{ CI} = .830 \text{ to } 8.891; p = .019$); individuals with better social knowledge at baseline appeared more likely to improve their structured activity post SRT. To illustrate the magnitude of this effect, individuals in the SRT group scoring in the top quartile at baseline on average, increased their structured activity by 11 hours more than those who scored below the 50th percentile.

4. Discussion

4.1. Main Findings

First, we investigated whether there was a change on the social cognitive and neurocognitive variables following a specialized psychosocial intervention. In our main analysis, we did not identify robust statistically significant benefits of SRT on either neurocognition or social cognition over the follow-up interval, compared with TAU. Although attribution of blame bias was significant post SRT, this effect was lost once a Bonferroni correction was applied, suggesting this finding was perhaps due to chance.

Overall, findings may suggest that SRT had no impact on social cognition or neurocognition, but given the high level of missing data at 9 months, this finding might be biased if those who dropped out had poorer outcomes on the cognitive measures. As data were considered missing not at random, with the assumption that missingness was associated with a poorer outcome (an assumption strongly supported by the primary and secondary analyses of main trial), joint models may be considered to provide the least biased assessment of the outcome (Hickey et al., 2016). Results of the joint modelling (multivariate) analyses demonstrated nominally significant treatment effects for several neurocognitive and social cognitive outcomes. Whilst
this is encouraging, we are unable to make firm conclusions due to the lack of treatment effect in our main analysis.

Plausible reasons for the lack of treatment effect could be as follows: first, the SRT did not seek to address cognitive impairments. Second, in prior studies evaluating psychosocial interventions, these studies focused on homogenous groups with enduring illness, where the effects of illness chronicity, such as medication and repeated relapse, may contribute to impaired functioning (Roder et al., 2011). In contrast, the present sample represented a clinically heterogeneous subgroup with FEP who were deemed to have ‘severe social disability’, and likely to be a ‘chronic’ sample of the future. Comparing the severity of cognitive impairment reported in other early psychosis studies (typically 1 SD below the mean), impairments were also evident in this sample, and most pronounced for social knowledge, ToM, verbal memory and verbal comprehension (Cutting and Murphy, 1990; Langdon et al., 2014; Lin et al., 2011; Combs et al., 2007; Simons et al., 2016). Participants were mildly impaired on problem solving, and no impairment was observed for social perception, hence it is not surprising that we didn’t see a treatment effect for these variables. Interestingly, by follow-up, the neurocognitive scores were above the impairment threshold for both groups. Whilst this might represent a natural recovery process, improvement over time might simply reflect a practice effect.

A second aim of this study was to investigate whether social cognition and neurocognition predicted response to SRT. We found that individuals who had better social knowledge prior to therapy were more likely to improve their structured activity post therapy. Social knowledge may therefore have a moderating influence on response to SRT, expanding on prior research showing that baseline cognition has a rate-limiting impact on functional change following
psychosocial rehabilitation (Brekke et al., 2007). We postulated that those who have a better understanding of the social world may be more able to use adaptive cognitive reappraisal strategies to engage in social activity and a psychosocial intervention, compared to those who struggle to understand social situations (Rowland et al., 2013). They may also form a better therapeutic alliance leading to a better therapy response (Allott et al., 2011b). But it must be acknowledged that no other social cognitive sub-domain predicted treatment response, and chance is a plausible explanation for the observation given the high number of variables in the model.

4.2. Implications and future directions

Firstly, the potential to identify individuals at an early stage who are more likely to respond to therapy could ensure that costly interventions (for both health service and service user), are delivered to those who are more likely to benefit. An *a priori* study comparing response to SRT between those with ‘poor’ and ‘good’ social knowledge would be a direct test of this assumption. A second implication is the potential to guide interventions for those who are less responsive; improving social cognition might improve response to SRT. Refining SRT by incorporating a social cognition intervention may enhance its efficacy in those with deficits in social cognition (Bartholomeusz et al., 2013). Indeed, integrative psychosocial interventions with adjunctive cognitive training are shown to have greater cognitive and real-world functional improvements in schizophrenia (Horan and Green, 2017; Roder et al., 2011). The application of an integrated, multi-dimensional treatment approach combining SRT with a form of social cognitive intervention may produce more durable and sustained therapeutic effects for individuals with poor functioning in FEP.
4.3. Study Strengths and Limitations

The study benefits from being part of a large scale, multi-site, randomized controlled trial, where researchers were blind to group allocation. Participants were recruited from several centers across the UK, which included urban, rural and town settings, making the sample highly diverse and representative. Nevertheless, despite a substantial proportion of the overall sample completing cognitive assessments at baseline, 20% of the trial sample did not. This may have biased the findings, as those with poorer cognitive function may have chose not to engage in cognitive assessments. There was also a significant number of participants who did not complete the cognitive assessments (up to 40%; Table 4), resulting in inadequate power to detect a posited significant post-intervention effect. Further, there was a bias for missing data in the control arm at follow-up, and it is plausible that individuals with missing data, on average, had lower scores on the cognitive measures compared to those who remained in the study, making the findings difficult to interpret. Although we attempted to address this bias with joint modelling, we are unable to draw on conclusions without replication in a larger sample. Further, we cannot rule out that the higher percentage of missing cases simply reflected the challenge of retaining TAU participants in trials. This group of young people in particular are an incredibly challenging group to engage and retain in research. The pattern of missingness was evident across all measures in the main trial, and twice-higher in the TAU group 6-months post-intervention (Fowler et al., 2017). As the SRT aims to promote social engagement, and research participation requires a level of social engagement, this may demonstrate that missingness in this instance was related to the outcomes of interest. A final limitation is the selection of cognitive subtests and their ecological validity and sensitivity to detect changes following improvements in structured activity. In particular, vocabulary and block design may be insensitive to detect fluctuations in cognitive performance (Nuechterlein et al., 2008). Further, a number of social cognitive measures, including the AIHQ used in this study, have
been deemed unsuitable for assessing social cognition in early psychosis (Ludwig et al., 2017; Pinkham et al., 2014). Future research should aim to select appropriate measures identified by consensus groups (e.g. MATRICS), to ensure the reliability of findings across studies (Nuechterlein et al., 2008; Pinkham et al., 2014).

4.4. Conclusion

Results of the main analysis suggest that SRT had no impact on social cognition or neurocognition, however, joint models suggest an improvement on several social cognitive and neurocognitive variables following treatment. Those who had better social knowledge at baseline were also more likely to improve their structured activity post SRT. Although no firm conclusions can be made about the treatment effect on cognition, the findings have implications for targeted intervention for individuals in the early phase of psychosis with poor social cognition. Combining SRT with a social cognitive intervention may maximize its impact by providing more durable and sustained treatment effects, particularly for those who may not respond to SRT alone.

5. Acknowledgments

We acknowledge the SUPEREDEN programme for incorporating this study into the trial. SUPEREDEN is funded by the National Institute of Health Research (NIHR) under the Programme Grants for Applied Research programme (RP-PG-0109-10 074). The NIHR clinical research network helped with data collection, but had no involvement in the design, analysis and interpretation of the findings, nor the write up. M.B. and S.P.S are part funded by the National Institute for Health Research through the Applied Research Collaboration West Midlands (ARC-WM). P.B.J. is part funded by the NIHR ARC East of England.
Views expressed here are not necessarily those of the NIHR or Department of Health. We thank all research participants, research assistants, and trial therapists. Declarations of interest: none.
6. References


Cutting, J., Murphy, D., 1990. Impaired ability of schizophrenics, relative to manics or depressives, to appreciate social knowledge about their culture. British Journal of Psychiatry, 157, 355-258. https://doi.org/10.1192/bjp.157.3.355


### Tables

Table 1. Comparison of demographic and clinical characteristics (by trial allocation) between the full trial sample compared with the sub-group completing cognitive assessments.

<table>
<thead>
<tr>
<th>Item</th>
<th>SRT group allocation in the cognitive sub-sample (n=59)</th>
<th>SRT group allocation in the trial sample (n=75)</th>
<th>95% CI of the difference and p-value</th>
<th>TAU group allocation in the cognitive sub-sample (n=63)</th>
<th>TAU group allocation in the trial sample (n=79)</th>
<th>95% CI of the difference and p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean Age</strong> (years)</td>
<td>25.89</td>
<td>24.8</td>
<td>-1.88 to 1.56 (p=.851)</td>
<td>24.8</td>
<td>24.2</td>
<td>-1.35 to 1.87 (p=.750)</td>
</tr>
<tr>
<td><strong>Gender:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male (n; %)</td>
<td>44; 72.9</td>
<td>56; 74.7</td>
<td>0.52 to 2.38 (p=.929) a</td>
<td>54; 84.4</td>
<td>60; 76.0</td>
<td>0.69 to 3.81 (p=.369) a</td>
</tr>
<tr>
<td><strong>Years in Education</strong> (Mean)</td>
<td>12.1</td>
<td>12</td>
<td>-0.66 to 0.37 (p=.584)</td>
<td>12.2</td>
<td>12</td>
<td>-0.65 to 0.46 (p=.738)</td>
</tr>
<tr>
<td><strong>DUP (days; mean)</strong></td>
<td>341</td>
<td>240</td>
<td>-411.21 to 209.62 (p=.522)</td>
<td>270</td>
<td>285</td>
<td>-118.94 to 148.33 (p=8.28)</td>
</tr>
<tr>
<td><strong>Time Use Survey</strong></td>
<td>10</td>
<td>11</td>
<td>-1.85 to 3.31 (p=.577)</td>
<td>270</td>
<td>285</td>
<td>-2.49 to 3.16 (p=.814)</td>
</tr>
<tr>
<td><strong>Symptoms (PANSS; mean)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>13.3</td>
<td>13.3</td>
<td>-1.64 to 1.66 (p=.989)</td>
<td>14.1</td>
<td>14.6</td>
<td>-1.39 to 2.39 (p=.603)</td>
</tr>
<tr>
<td>Negative</td>
<td>15.6</td>
<td>15.5</td>
<td>-2.27 to 2.11 (p=.940)</td>
<td>16.8</td>
<td>16.6</td>
<td>-2.24 to 1.81 (p=.834)</td>
</tr>
<tr>
<td>General</td>
<td>32.4</td>
<td>32.8</td>
<td>-2.60 to 3.42 (p=.790)</td>
<td>32.3</td>
<td>33.7</td>
<td>-1.59 to 4.27 (p=.367)</td>
</tr>
</tbody>
</table>

aYates’ Correction for continuity. SRT = Social Recovery Therapy. TAU = Treatment as Usual. Cognitive sub-sample = participants who completed cognitive assessments and were included in the present study.
Table 2. Means and standard deviations for the individual cognitive variables by group allocation, along with missing data at 9-month follow-up.

<table>
<thead>
<tr>
<th>Cognitive measures</th>
<th>Baseline</th>
<th>9-month Follow-up</th>
<th>Missing data at 9-month follow-up (n; %)</th>
<th>Missing data at 9-month follow-up (n; %)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SRT M±SD</td>
<td>SRT M±SD</td>
<td>TAU M±SD</td>
<td>SRT M±SD</td>
<td>TAU M±SD</td>
</tr>
<tr>
<td>Verbal learning and memory&lt;sup&gt;a&lt;/sup&gt;</td>
<td>8.65 (3.75)</td>
<td>6.88 (2.95)</td>
<td>8.94 (3.77)</td>
<td>7.83 (3.53)</td>
</tr>
<tr>
<td>Verbal comprehension&lt;sup&gt;a&lt;/sup&gt;</td>
<td>8.49 (3.51)</td>
<td>7.38 (2.85)</td>
<td>9.14 (3.23)</td>
<td>8.31 (3.22)</td>
</tr>
<tr>
<td>Non-verbal problem solving&lt;sup&gt;a&lt;/sup&gt;</td>
<td>8.71 (3.08)</td>
<td>7.74 (3.33)</td>
<td>9.39 (3.37)</td>
<td>8.76 (2.78)</td>
</tr>
<tr>
<td>Theory of Mind&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.44 (0.98)</td>
<td>4.35 (1.07)</td>
<td>4.74 (0.94)</td>
<td>4.56 (1.05)</td>
</tr>
<tr>
<td>Social Knowledge&lt;sup&gt;b&lt;/sup&gt;</td>
<td>7.00 (1.87)</td>
<td>6.48 (1.55)</td>
<td>7.16 (1.75)</td>
<td>6.95 (1.53)</td>
</tr>
<tr>
<td>Attribution of blame bias&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.94 (0.91)</td>
<td>2.85 (0.88)</td>
<td>2.49 (0.98)</td>
<td>2.84 (0.93)</td>
</tr>
<tr>
<td>Social perception&lt;sup&gt;b&lt;/sup&gt;</td>
<td>98.36 (18.60)</td>
<td>95.44 (22.59)</td>
<td>101.92 (18.69)</td>
<td>96.86 (18.22)</td>
</tr>
</tbody>
</table>

<sup>a</sup>Neurocognitive sub-domain. <sup>b</sup>Social Cognition sub-domain. Group = Social Recovery Therapy + Early Intervention vs. Treatment as usual (Early Intervention). †p value from joint modelling (multivariate) analyses. The p value is the effect of treatment allocation, derived from the joint model of loss to follow up and item.
Table 3. MANOVA results testing for treatment effects in each of the social cognitive and neurocognitive variables, along with results of the joint models.

<table>
<thead>
<tr>
<th>Group x Time Interaction Effect</th>
<th>Effect size (95% CI)</th>
<th>p – value</th>
<th>‡p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verbal learning and memory&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.088 (0 to 0.346)</td>
<td>.197</td>
<td>0.138</td>
</tr>
<tr>
<td>Verbal comprehension&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0 (0 to 0.263)</td>
<td>.603</td>
<td>0.004</td>
</tr>
<tr>
<td>Non-verbal problem solving&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0 (0 to 0.288)</td>
<td>.452</td>
<td>0.025</td>
</tr>
<tr>
<td>Theory of Mind&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0 (0 to 0.272)</td>
<td>.565</td>
<td>0.011</td>
</tr>
<tr>
<td>Social Knowledge&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.092 (0 to 0.346)</td>
<td>.187</td>
<td>0.012</td>
</tr>
<tr>
<td>Attribution of blame bias&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.196 (0 to 0.437)</td>
<td>.049&lt;sup&gt;‡&lt;/sup&gt;</td>
<td>0.008</td>
</tr>
<tr>
<td>Social perception&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0 (0 to 0.281)</td>
<td>.562</td>
<td>0.359</td>
</tr>
</tbody>
</table>

<sup>a</sup>Neurocognitive sub-domain. <sup>b</sup>Social Cognition sub-domain. <sup>‡</sup>No longer significant after applying a Bonferroni correction. <sup>‡p</sup> value from joint modelling (multivariate) analyses – the p value is the effect of treatment allocation, derived from the joint model of loss to follow up and item.

Table 4. Optimal regression model predicting change in Time Use post Social Recovery Therapy using baseline social cognitive and neurocognitive predictors.

<table>
<thead>
<tr>
<th>Predictors</th>
<th>β</th>
<th>95% CI</th>
<th>Adjusted R&lt;sup&gt;2&lt;/sup&gt;</th>
<th>p – value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Knowledge</td>
<td>.350&lt;sup&gt;‡&lt;/sup&gt;</td>
<td>.830 to 8.891</td>
<td>0.110</td>
<td>0.029</td>
</tr>
<tr>
<td>Attribution of Blame Bias</td>
<td>.278</td>
<td>-.358 to 15.866</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<sup>‡</sup><sub>≤0.05</sub>. <sup>‡</sup>Retains Significance with a post-hoc Bonferroni Correction.