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The Direct and Indirect Relationship Between Social Cognition and Psychosocial Dysfunction in Major Depressive Disorder

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Background: Recent evidence suggests that depressed patients experience social cognitive deficits (e.g., poor affect recognition). However, very little is known regarding the contribution of social cognitive deficits to psychosocial dysfunction (e.g., occupational functioning). In particular, the mechanistic roles of depression severity and cognitive deficits (e.g., memory) in this domain have not been explored. The current study evaluated the extent to which mood symptoms and cognitive deficits provide a mechanistic explanation for the relationship between social cognitive and psychosocial deficits in major depressive disorder (MDD).

Methods: Data were obtained from 111 participants with MDD (75 Female, mean age = 35, 84% Caucasian, 12% Asian, 4% Other) in the Cognitive Function and Mood Study (CoFaM-S), a cross-sectional study of mood, social cognition, cognition, and psychosocial functioning in mood disorders. Social cognitive abilities were assessed using the Social Perception subtest of the Wechsler Adult Intelligence Scale, and psychosocial dysfunction was clinically evaluated with the Functioning Assessment Short Test (FAST).

Results: Cognitive deficits and mood symptoms did not significantly mediate relationships between social cognitive ability and psychosocial dysfunction. The exception was executive function, which mediated an indirect relationship between meaning interpretation (i.e., theory of mind) and self-perceived cognitive dysfunction.

Conclusion: The results suggest that the relationship between social cognitive deficits and psychosocial dysfunction is not mechanistically explained by mood symptoms or nonsocial cognition. Development of treatment strategies targeting social cognitive deficits in patients with MDD is warranted.

Keywords: depression, social cognition, cognition, mediation, memory, facial recognition, psychosocial functioning

INTRODUCTION

“Social cognition” refers to the broad range of perceptual and cognitive abilities involved in the interpretation of social information, including affect recognition, eye contact, prosody interpretation, and inferring the mental states of others (i.e., theory of mind) (1, 2). Recent evidence suggests that major depressive disorder (MDD) is associated with social cognitive deficits in both the acute (3) and remitted stage of illness (4), primarily in domains of affect recognition and theory of mind (5–8). However,

the extent to which social cognitive deficits independently contribute to psychosocial dysfunction (e.g., occupational functioning, interpersonal relationships) has not been explored.

Social cognition is reliant on a number of cognitive abilities, as social information is processed through memory and attention, with more complex social abilities (e.g., theory of mind) likely facilitated by executive functions (e.g., problem solving) (9, 10). Evidence for the reliance of social abilities on cognitive function in mentally ill patients is available in domains of schizophrenia and attention deficit hyperactivity disorder (ADHD), with research demonstrating that deficits in processing speed, executive functioning, and working memory are associated with impaired theory of mind (1, 9, 11, 12). These findings highlight the close overlap between social and nonsocial cognition in clinical populations, and the need for research in the domain of MDD specifically.

A wide body of literature supports the notion that cognitive deficits are associated with psychosocial dysfunction, notably in domains of occupational functioning, interpersonal relationships, daily responsibilities, and self-perceived quality of life (13–18). Cognitive deficits acquired in the acute stage of illness commonly persist through diagnostic remission and are associated with higher rates of relapse and prolonged psychosocial dysfunction (19–21), highlighting the chronicity of dysfunction associated with cognitive deficits. Given the broad links between cognitive and psychosocial dysfunction, it is possible that social cognitive deficits also contribute to functional deficits in MDD (8, 22), particularly in domains highly reliant on social functioning (e.g., occupational functioning, interpersonal relationships).

An initial study by Knight and Baune (22) demonstrated that prosody interpretation was linked with reduced psychosocial function, whereas facial affect recognition and meaning interpretation (e.g., sarcasm detection) were not associated with functional deficits. These findings highlight the clinical importance of social cognition in MDD patients, such that remediating social cognitive deficits may be an important treatment target in alleviating psychosocial dysfunction. At present, there is a dearth of literature examining the role of social cognition in psychosocial function, highlighting the need for studies that show the impact and clinical mechanisms of social cognition in MDD.

As social cognition is likely reliant on cognitive abilities (e.g., memory), the question is raised as to whether social cognition contributes to psychosocial functioning independently of cognitive functioning. It is possible, for instance, that deficits in executive functioning could explain impaired analysis of social information and reduced psychosocial functioning. If executive function is the primary clinical mechanism, then patients presenting with social cognitive deficits may benefit more from treatment targeting executive function specifically than from treatments centered on social cognition. Given the importance of selecting appropriate treatment targets, it is crucial to elucidate whether cognitive domains mediate the relationship between social cognition and psychosocial function. The current study is the first to evaluate the extent to which the effect of social cognition on psychosocial function is mediated by cognitive performance.

Social cognitive deficits in MDD are also associated with the general severity of illness symptoms (e.g., pessimism, anxiety) (5, 23, 24), suggesting that general depression symptoms interfere with

the perception and interpretation of social information. Support for this notion was found by Air et al. (5), who found that greater affective symptom severity in currently depressed patients was negatively associated with ability to detect emotions and interpret meaning from verbal prosody. In addition, somatic symptoms (e.g., headaches, muscle pain) were negatively associated with ability to detect emotions from facial affect. The role of symptom severity in social cognition was also demonstrated by Gollan et al. (23), who presented MDD patients and healthy controls with pictures of facial expressions, which varied in the intensity of emotion conveyed. The results indicated that depressed patients detected sadness with greater sensitivity, but detection of other emotions (e.g., surprise) was impaired relative to healthy controls. Symptom severity enhanced this relationship, such that more severely depressed patients were increasingly sensitive to sadness, and less sensitive to other emotions. Taken together, available research suggests that greater depression severity may reduce social perception abilities in general (5), as well as facilitate hypersensitivity to negative social information (25).

Similarly to cognition, greater depressive symptom severity is associated with reduced social cognitive abilities and poor psychosocial functioning (14, 26). Accordingly, it is important to distinguish the contribution of social cognition to psychosocial functioning independently of illness severity, a key objective in the current study. This research will better inform clinicians as to the importance of treating social cognitive deficits as distinct from mood symptoms (27), or alternatively, whether treatment of mood symptoms by existing treatment approaches (e.g., cognitive behavioral therapy, antidepressants) is sufficient to remediate social cognitive deficits.

It is also possible that poor social cognition exerts a negative effect on psychosocial function indirectly by increasing symptom severity or cognitive dysfunction. These indirect effects would not have been detected by previous studies, which examined total (i.e., unmediated) relationships between social cognition and psychosocial function (22, 28). As a result, the present study fills an important gap in our understanding by examining the indirect relationship between social cognition and psychosocial function, as mediated by cognition and mood symptoms.

The primary goals were as follows:

1. To evaluate whether social cognitive domains (i.e., affect recognition, prosody interpretation, meaning interpretation) were directly associated with overall psychosocial dysfunction, independently of mood symptoms or cognitive deficits.
2. To examine whether social cognitive domains were associated with psychosocial dysfunction indirectly *via* mediating mood symptoms or cognitive deficits.

METHODS

Participants

The present data were obtained from the Cognitive Function and Mood Study (CoFaM-S) (19), a multisite cross-sectional analysis of cognitive, social, emotional, and functional status in patients with mood disorders. The CoFaM-S was reviewed by the Human Research

Ethics Councils (HRECs) of the Royal Adelaide Hospital (approval number: 111230) and the University of Adelaide (approval number: H-160-2011) and was conducted in accordance with the Declaration of Helsinki. All participants were verbally informed regarding study procedures, and read a study information sheet, before providing written consent to participate. CoFaM-S participants were at least 18 years of age, and no age limit was imposed. Inclusion criteria included lifetime diagnosis of a mood disorder according to *diagnostic and statistical manual of mental disorders, fourth edition, text revised (DSM-IV-TR)* criteria (29). Participants were recruited from inpatient and outpatient clinics throughout Eastern, Western, and Northern Mental Health Networks in Adelaide, South Australia, as well as through advertisement in the general community. Exclusion criteria included diagnosis of psychotic disorders, dementia, learning disorders, eating disorders, autism spectrum disorder, or medical illnesses, which could affect cognitive functioning (e.g., multiple sclerosis) (19). The mini international neuropsychiatric interview (MINI) Neuropsychiatric Diagnostic Interview was used to screen patients for psychiatric illness (30), and the Hamilton Depression Rating Scale (HAM-D) was used to measure depression symptom severity (31).

Participants ($N = 111$) with a lifetime diagnosis of MDD were included in the current study on the basis of completing standard measures of social cognition, cognition, depression symptom severity, and psychosocial functioning (see specific scales below). Inclusion criteria were a current ($n = 42$) or previous ($n = 69$) diagnosis of MDD following *DSM-IV-TR* criteria (29) according to the MINI 600 (31). Average time since first depressive episode was 15.2 years ($SD = 14.04$), and mean number of previous depressive episodes was 1.4 ($SD = 1.39$). Antidepressants were being used by 29 (26%) of the included participants. Participants' mean age was 34.95 ($SD = 16.38$), 75 (68%) participants were female, and mean years of education were 13.48 ($SD = 2.24$). Ninety-three (84%) participants were Caucasian, 13 (12%) were Asian, and 5 (4%) were Indigenous/African/Hispanic.

Social Cognitive Assessment

Social cognition was evaluated with the Wechsler Adult Intelligence Scale Advanced Clinical Solutions (ACS) Social Perception Subtest (WAIS-ACS). The assessment involves the detection of emotional states and meaning interpretation by appraisal of facial affect, prosody, and body language (32, 33). A previous study by Air et al. (5) indicated that social cognitive abilities measured by the ACS were associated with mood and anxiety symptoms in currently depressed, but not remitted, individuals. The findings of Air et al. (5) suggest that social cognition as measured by the ACS is a state-like feature of illness affected by patients' present diagnostic status. Accordingly, the scale is appropriate for the current purpose of testing associations between social cognition and psychosocial function. The ACS assessment tasks are explained below:

- **Affect Naming (ACS Affect):** The participant is presented with 24 images of male and female faces, each displaying a particular emotion (i.e., happiness, sadness, surprise, fear, anger, disgust, and neutral). An answer sheet is provided to participants, indicating each of the seven potential responses (i.e., emotions) that could be displayed by each face image.

Participants are instructed to select which emotion on the answer sheet best matches that conveyed by the image. Correct answers award a single point, leading to a maximum possible raw score of 24, demonstrating ability to detect facial affect.

- **Prosody-Face Matching (ACS Prosody):** Participants are presented with six faces, each displaying a particular emotion (i.e., the seven emotions used in the Affect Naming task). A voice is played to the participant, who must match the emotional tone of the speaker with one of the six presented faces. This process is repeated 12 times, for a maximum possible raw score of 12. This task reflects the ability to interpret prosody in the context of associated facial affect.
- **Prosody-Pair Matching (ACS Pairs):** In this task, the participant is presented with four images, each depicting two people interacting. Similar to the face matching task, a voice is played to the participant, who must indicate which image best matches the tone and intention of the speaker. In addition, the participant is instructed to indicate what feeling is conveyed by the tone of voice, and whether the tone of voice changes the meaning of the comment (e.g., sarcasm, humor). If a meaning change does occur, the participant indicates the true meaning/intentions of the speaker. Taken together, performance in the prosody-pair matching task leads to a maximum raw score of 42, indicating ability to interpret meaning from prosody and body language in social interactions.

Cognitive Assessment

Cognitive functioning was assessed with three cognitive test batteries: the Repeat Battery for the Assessment of Neuropsychological Status (RBANS), the Colorado Assessment Tests (CATS), and the Psychology Experiment Building Language (PEBL). The RBANS is a brief screening instrument designed to detect neuropsychological deficits in individuals with physical or mental illness (34). Five cognitive domains are assessed: immediate memory, delayed memory, visuospatial/constructional memory, language, and attention. RBANS tests included in the current study were the digit span, list learning, list recognition, store memory, figure recall, picture naming, figure copy, and line orientation tests (34). The CATS battery included the n -back, visual span, word recall, Tower of London, and Wisconsin card sorting tests (35). The PEBL battery includes computerized versions of many psychological tests (e.g., the Tower of London, Corsi Blocks) (36). We utilized the Stroop and Wisconsin Card Sorting tasks in the PEBL battery as additional measures of executive functioning and attention. Individual participants completed either the CATS or PEBL executive tasks (i.e., Tower of London, Wisconsin Card Sorting test), as each test battery was applied at a different site of the study.

Domain-specific cognition composites were derived from the RBANS, CATS, and PEBL batteries by transforming raw performance in specific tasks (e.g., digit span, list learning) into Z-scores, which were averaged into composite Z-scores (e.g., immediate memory), giving equal weighting to each test from which the composite cognitive domain Z-score was derived. Individual participants completed either the CATS or PEBL executive tasks, as these test batteries were applied at different study sites. **Table 1** demonstrates the derivation test results

TABLE 1 | Calculation of composite domain-specific Z-scores by cognitive domain in the Repeat Battery for the Assessment of Neuropsychological Status (RBANS), Colorado Assessment Tests (CATS), and Psychology Experiment Building Language (PEBL) batteries.

Cognitive domain	Cognitive tests		
	RBANS	CATS	PEBL
Executive Functioning		Tower of London (excess moves total) Wisconsin Card Sorting test (perseverative responses)	Stroop (incongruency errors) Tower of London (total excess moves) Wisconsin Card Sorting test (perseverative responses) Stroop (incongruency errors)
Attention	Digit Span Coding	VSPAN [†] (total average time) NBACK (accuracy)	
Immediate Memory	List Learning Store Memory Digit Span	Word Recall (total words recalled) VSPAN (correct trials forward) VSPAN (correct trials backward)	
Delayed Memory	List Recognition List Recall Story Recall Figure Recall	Word Recall (primacy total)	
Semantic Fluency	Picture Naming Semantic Fluency	Word Recall (total words recalled)	
Spatial Cognition	Figure Copy Line Orientation	Tower of London (percentage above optimal)	

[†]VSPAN, verbal memory span.

to composite Z-scores in domains of executive functioning, attention, immediate memory, delayed memory, semantic fluency, and spatial cognition. For a similar procedure, see Godard et al. (37).

It is noteworthy that several of the cognitive tests employed overlap across more than one cognitive domain. However, this should not preclude the current domain-specific use of these tests, as each is primarily associated with a specific domain. For instance, despite being an “executive” task, Beck’s Card Sorting Test (BCST) places significant load on visuospatial cognition due to its reliance on analyzing color, shape, and number of visual features. As a result, the BCST is not a “pure” measure of executive function. However, the primary cognitive challenge of the BCST is to adapt to new logical rules in sorting cards. In contrast, analysis of the visual information alone (i.e., identifying color/shape/number features) is relatively simple. As a result, we contend that it is appropriate to consider the BCST as a measure of executive function, as executive demands comprise the primary cognitive challenge of the task. Similarly, other cognitive tests also overlap several cognitive domains, but the most parsimonious explanation is to consider the primary cognitive domain of a particular cognitive test.

Clinical Assessments

The Functioning Assessment Short Test (FAST) was used to clinically evaluate psychosocial functioning. The FAST is a clinician rated interview, in which the clinician conducts a semistructured interview to evaluate the extent of dysfunction across six functional domains (i.e., autonomy, occupational functioning, cognitive functioning, financial issues, leisure time, and interpersonal relationships). Functional impairment is rated on a four-point scale, with 0 indicating no dysfunction and 3 indicating severe impairment. Six composite FAST

scores were derived from the mean score of impairment in each functional subdomain [i.e., autonomy (range: 0–12), occupational dysfunction (range: 0–15), subjective cognition (range: 0–15), financial issues (range: 0–6), interpersonal relationships (range: 0–18), and leisure time (range: 0–6)]. Overall psychosocial functioning (i.e., FAST total score) is indicated by the sum of FAST subdomains (range: 0–72). FAST total and FAST subdomain scores were used as the dependent variable in regression analyses.

Depression symptom severity was evaluated with the Hamilton Depression Rating Scale (HAM-D) (31). The scale involves 17 interview items that measure a range of mood and somatic symptoms, ranging in severity from 0 to 4, with greater scores indicating more severe depression. A total score of 0–7 indicates normal mood, 8–13 indicates mild depression, 14–18 indicates moderate depression, 19–22 indicates severe depression, and ≥23 indicates very severe depression. The scale has been widely used in MDD literature, with indicated severity associated with measures of psychosocial functioning (14).

Statistical Analyses

Analyses were conducted with statistical package of the social sciences (SPSS) for Windows, version 24. As the current research was exploratory and employed an existing data set, *post hoc* power analyses were not used (8, 38, 39). Descriptive statistics (Ms, SDs) for the psychosocial dysfunction in the FAST and social cognition in the ACS are presented in **Supplementary eTable 1**. Mediation analyses were conducted with the “Process” macro for SPSS developed by Hayes (40). The Process macro enables nonparametric testing of the indirect relationship $a \times b$ using a bootstrap test of 5,000 resamples. In comparison to the Sobel test, the nonparametric nature of the bootstrap test enables greater statistical power to detect an indirect effect and was therefore appropriate for the present analyses (41).

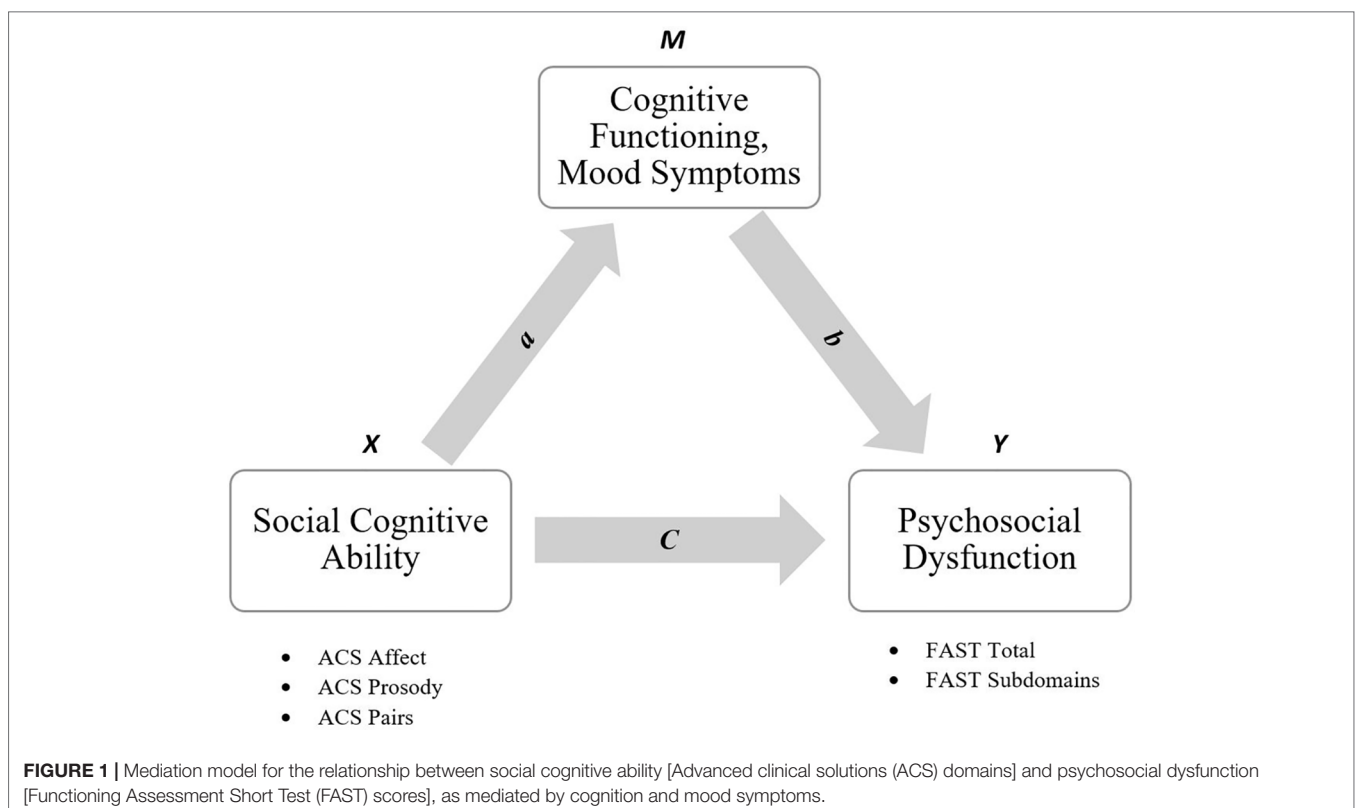
Mediation analyses were performed following the procedure recommended by Zhao et al. (41). The notation for this mediation model and the mediation pathways evaluated in the present analyses are presented in **Figure 1**. Following Zhao et al. (41), the only criteria for mediation is a significant indirect effect of X (the independent variable) on Y (the dependent variable) *via* M (the mediator). The indirect effect is calculated as the product of the coefficients of relationships a and b (i.e., $a \times b$). If $a \times b$ is significant, then mediation subtype is determined by the sign (i.e., + or -) of $a \times b$, and whether the direct effect of X on Y (i.e., " c ") is significant, after adjusting for the removal of variance explained by $a \times b$ (41). Mediation may occur in three patterns: 1) *complementary mediation* if the mediated effect ($a \times b$) is in the same direction as the direct effect (c), 2) *competitive mediation* if $a \times b$ is in the opposite direction to c , or 3) *indirect-only mediation* if $a \times b$ is significant while c is not significant. In contrast, nonmediation may occur in two patterns: 1) *direct-only nonmediation* occurs if $a \times b$ is not significant while c is significant, 2) *no-effect nonmediation* occurs if neither $a \times b$ nor c is significant. The total effect (c') indicates the relationship between X and Y , before adjusting for variance explained by $a \times b$.

In the current design, a single ACS domain was entered as X , the HAM-D score and composite cognitive domain Z-scores were entered as M , and FAST total score was entered as Y . If the HAM-D score or composite cognition Z-scores produced a significant $a \times b$ for a specific ACS domain, then follow-up analyses were conducted with FAST subdomains

entered as Y in subsequent models. This exploratory approach enabled testing of domain-specific mediation relationships with each of the three ACS domains, evaluating the potential mechanistic role of cognition and mood symptoms in specific functional deficits. Age, sex, and years of education were entered as covariates, as these factors can influence the relationship between cognition and psychosocial functioning (14, 42).

As the participant sample was composed of currently ($n = 42$) and previously ($n = 69$) depressed individuals, it was appropriate to test whether these groups differed in their degree of psychosocial dysfunction (i.e., FAST total score). After adjusting for age, sex, and years of education with an analysis of covariance (ANCOVA), currently depressed participants demonstrated greater overall psychosocial dysfunction ($M = 31.18$, $SD = 17.38$) than participants remitted of MDD ($M = 15.97$, $SD = 16.02$, $p < .001$). Following this result, mediation analyses were conducted, which either included or excluded diagnostic status (current MDD, remitted MDD) as a covariate. The inclusion of diagnostic status as a covariate did not alter the significance or magnitude of relationships detected, and hence results that excluded this covariate are presented below.

The present analyses were exploratory in nature, and as such were uncorrected for multiple comparisons (43). Following this approach, any significant results were interpreted as reflecting a narrow (i.e., domain specific) and speculative statistical relationship subject to further testing by future confirmatory analyses (43).



RESULTS

Initial analyses with ACS affect as X and FAST total score as Y (see **Figure 1**) indicated no significant indirect relationships when the HAM-D score or composite cognition Z -scores were entered as mediators (all 95% CIs overlap 0). In addition, the direct effect between ACS affect and FAST total score was nonsignificant (all 95% CIs overlap 0). **Table 2** presents coefficient values, confidence intervals, and mediation status for the analysis of ACS affect.

When ACS Prosody was entered as X and FAST total score as Y , no significant indirect relationships were detected with

HAM-D score or composite cognitive domains entered as M (all 95% CIs overlap 0). The direct effect between ACS Prosody and FAST total score was not significant (all 95% CIs overlap 0). Statistics for the ACS Pairs analysis are presented in **Table 3**.

Including ACS Pairs as X and FAST total score as Y demonstrated a significant indirect effect with executive function ($a \times b = .08$, CI = .01, .19). As the direct effect was not significant ($c = -.01$, CI = $-.49$, .48), the pattern of mediation was indirect only. In contrast to executive function, neither HAM-D score nor any other composite cognitive domains resulted in significant indirect effects (all 95% CIs overlap 0).

TABLE 2 | Mediation criteria for the ACS Affect–FAST total relationship as mediated by Hamilton Depression Rating Scale (HAM-D) score and composite cognitive domains ($N = 111$).

Potential mediators	Mediation pathway				Mediation type
	a	b	Indirect effect value and 95% CI	Total (c') and direct (c) effect value and 95% CI	
HAM-D Score	-.04	1.38**	-.05 [-.99, .97]	$c' = .17$ [-1.09, 1.42] $c = .15$ [-.81, 1.10]	No effect (nonmediation)
Executive Functioning	-.09*	-1.72	.16 [-.06, .41]	$c' = .17$ [-1.09, 1.42] $c = .15$ [-.81, 1.10]	No effect (nonmediation)
Attention	.11*	.36	.04 [-.16, .30]	$c' = .17$ [-1.09, 1.42] $c = .15$ [-.81, 1.10]	No effect (nonmediation)
Immediate Memory	.07	-1.90	.14 [-.54, .07]	$c' = .17$ [-1.09, 1.42] $c = .15$ [-.81, 1.10]	No effect (nonmediation)
Delayed Memory	.12**	1.05	.12 [-.20, .47]	$c' = .17$ [-1.09, 1.42] $c = .15$ [-.81, 1.10]	No effect (nonmediation)
Semantic Fluency	.07	-1.83	.13 [-.34, .02]	$c' = .17$ [-1.09, 1.42] $c = .15$ [-.81, 1.10]	No effect (nonmediation)
Spatial Cognition	.02	.15	.01 [-1.01, .97]	$c' = .17$ [-1.09, 1.42] $c = .15$ [-.81, 1.10]	No effect (nonmediation)

Model adjusted for gender and years of education.

c' = Relationship between IV, Independent Variable; DV, Dependent Variable; unadjusted for other variables.

c = Relationship between IV and DV, after subtracting variance explained by the indirect effect $a \times b$.

TABLE 3 | Mediation criteria for the ACS Prosody–FAST total relationship as mediated by HAM-D score and composite cognitive domains ($N = 111$).

Potential mediators	Mediation pathway				Mediation type
	a	b	Indirect effect value and 95% CI	Total (c') and direct (c) effect value and 95% CI	
HAM-D Score	-.72	1.36**	-.97 [-1.96, .04]	$c' = -1.85$ [-3.38, -.31]* $c = -.87$ [-2.03, .28]	No effect (nonmediation)
Executive Functioning	-.09	-2.32*	.21 [-.13, .53]	$c' = -1.85$ [-3.38, -.31]* $c = -.87$ [-2.03, .28]	No effect (nonmediation)
Attention	.09	.37	.04 [-.16, .26]	$c' = -1.85$ [-3.38, -.31]* $c = -.87$ [-2.03, .28]	No effect (nonmediation)
Immediate Memory	.15**	-1.51	-.21 [-.91, .15]	$c' = -1.85$ [-3.38, -.31]* $c = -.87$ [-2.03, .28]	No effect (nonmediation)
Delayed Memory	.10	1.22	.12 [-.05, .92]	$c' = -1.85$ [-3.38, -.31]* $c = -.87$ [-2.03, .28]	No effect (nonmediation)
Semantic Fluency	.08	-1.63	-.14 [-.39, .04]	$c' = -1.85$ [-3.38, -.31]* $c = -.87$ [-2.03, .28]	No effect (nonmediation)
Spatial Cognition	-.01	-.03	.01 [-.14, .17]	$c' = -1.85$ [-3.38, -.31]* $c = -.87$ [-2.03, .28]	No effect (nonmediation)

Model adjusted for gender and years of education.

c' = Relationship between IV and DV, unadjusted for other variables.

c = Relationship between IV and DV, after subtracting variance explained by the indirect effect $a \times b$.

Statistics for the analysis of ACS Pairs with FAST total score are presented in **Table 4**.

To further analyze the significant indirect effect of ACS Pairs and executive function on FAST total score, follow-up analyses were conducted with FAST subdomains entered as *Y*. A significant indirect effect was demonstrated with subjective cognitive deficits entered as *Y* ($a \times b = .03$, CI = .01, .07), with the direct effect of ACS Pairs on subjective cognition remaining nonsignificant ($c = -.12$, CI = $-.26, .02$), resulting in an indirect-only mediation

pattern. This mediation relationship is depicted in **Figure 2**. The indirect effect was not significant when any of the remaining FAST subdomains were entered as *Y* (see **Table 5**).

DISCUSSION

The current study investigated whether the relationship between social cognitive deficits and overall psychosocial dysfunction

TABLE 4 | Mediation criteria for the ACS Pairs–FAST total relationship as mediated by HAM-D score and composite cognitive domains ($N = 111$).

Potential mediators	Mediation pathway				Mediation type
	Coefficients		Indirect effect value and 95% CI	Total (c') and direct (c) effect value and 95% CI	
	a	b			
HAM-D Score	-.20	1.34**	-.28 [-.78, .19]	c' = -.35 [-.91, .21] c = -.01 [-.49, .48]	No effect (nonmediation)
Executive Functioning	-.04*	-2.09	.08 [.01, .19]*	c' = -.35 [-.91, .21] c = -.01 [-.49, .48]	Indirect only (mediation)
Attention	.06**	.32	.02 [-.11, .17]	c' = -.35 [-.91, .21] c = -.01 [-.49, .48]	No effect (nonmediation)
Immediate Memory	.05**	-1.90	-.09 [-.31, .04]	c' = -.35 [-.91, .21] c = -.01 [-.49, .48]	No effect (nonmediation)
Delayed Memory	.06**	1.20	.07 [-.06, .30]	c' = -.35 [-.91, .21] c = -.01 [-.49, .48]	No effect (nonmediation)
Semantic Fluency	.08**	-1.79	-.14 [-.35, .03]	c' = -.35 [-.91, .21] c = -.01 [-.49, .48]	No effect (nonmediation)
Spatial Cognition	-.01	.18	-.01 [-.05, .06]	c' = -.35 [-.91, .21] c = -.01 [-.49, .48]	No effect (nonmediation)

Model adjusted for gender and years of education.

c' = Relationship between IV and DV, unadjusted for other variables.

c = Relationship between IV and DV, after subtracting variance explained by the indirect effect $a \times b$.

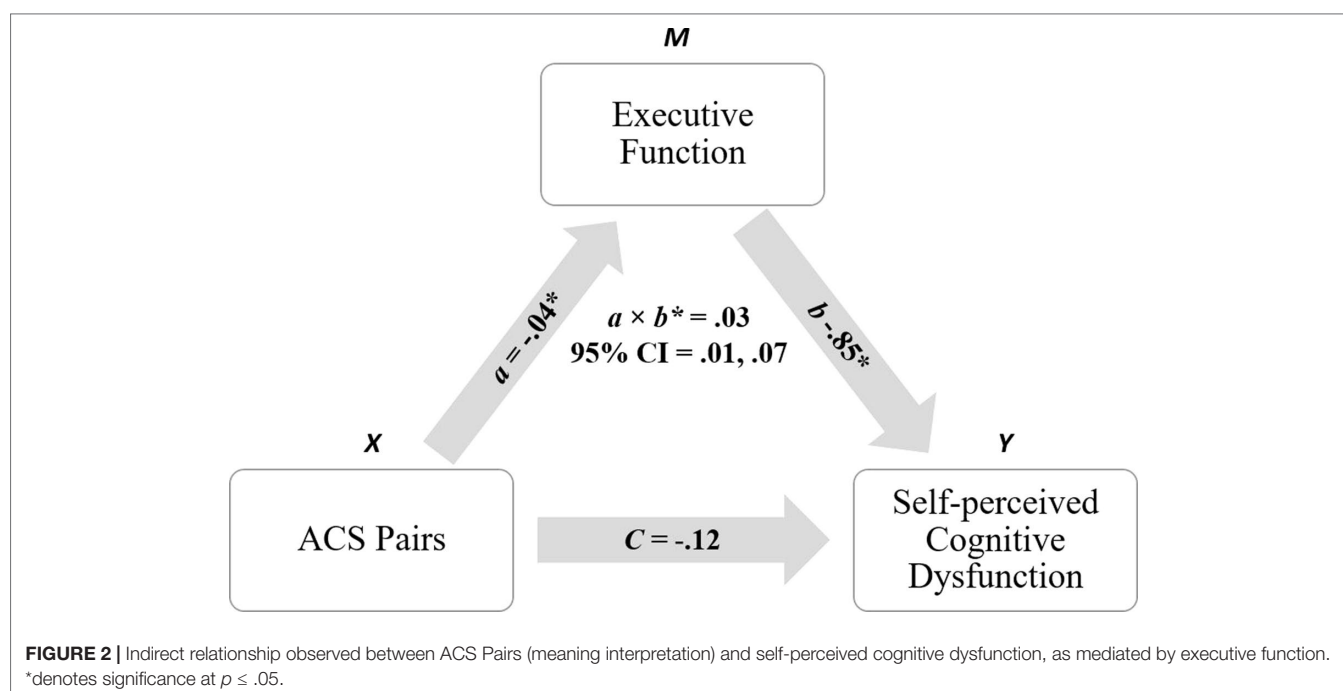


TABLE 5 | Mediation criteria for the ACS Pairs-FAST subdomain relationship as mediated by executive function ($N = 111$).

Potential mediators	Mediation pathway			Mediation type	
	Coefficients		Indirect effect value and 95% CI		Total (c') and direct (c) effect value and 95% CI
	a	b			
Autonomy	-.04*	-.61	.02 [.01, .6]	c' = -.08 [-.20, .03] c = -.11 [-.22, .01]	No effect (nonmediation)
Occupational Functioning	-.04*	-.39	.01 [-.04, .08]	c' = -.01 [-.17, .15] c = -.02 [-.18, .14]	No effect (nonmediation)
Subjective Cognition	-.04*	-.85*	.03 [.01, .07]*	c' = -.09 [-.23, .05] c = -.12 [-.26, .02]	Indirect only (mediation)
Leisure Time	-.04*	-.33	.01 [-.01, .03]	c' = -.04 [-.11, .03] c = -.05 [-.12, .01]	No effect (nonmediation)
Financial Issues	-.04	-.24	.01 [-.01, .03]	c' = -.01 [-.06, .04] c = -.02 [-.07, .03]	No effect (nonmediation)
Interpersonal Relationships	-.04*	-.14	.01 [-.04, .06]	c' = -.12 [-.31, .06] c = -.13 [-.31, .06]	No effect (nonmediation)

Model adjusted for gender and years of education.

c' = Relationship between IV and DV, unadjusted for other variables.

c = Relationship between IV and DV, after subtracting variance explained by the indirect effect $a \times b$.

*significant at .05; **significant at <.001.

in lifetime MDD patients is mediated by symptom severity or cognitive function. Our analyses indicated an indirect relationship between meaning interpretation (i.e., theory of mind) and total psychosocial dysfunction, as mediated by executive functioning. Follow-up analyses revealed that executive functioning mediated an indirect relationship between meaning interpretation and self-perceived cognitive dysfunction. While a direct relationship was found between poor prosody interpretation and impaired overall psychosocial functioning, neither HAM-D nor cognitive performance mediated this relationship. Finally, facial affect recognition was not significantly associated with psychosocial functioning either directly or indirectly *via* symptom severity or cognitive function. These results are the first to demonstrate the interaction between social cognition, mood symptoms, and cognitive deficits in their effect on psychosocial dysfunction.

While our previous research on this topic established the link between poor prosody interpretation and psychosocial dysfunction (22), a significant limitation to this research was the possible conflation of reduced social cognition with greater symptom severity and cognitive deficits, as these symptoms typically co-occur. This limitation restricted the conclusion that prosody interpretation was related to psychosocial function directly, as mood symptoms or cognitive deficits could explain this relationship. The present results provide clarity on this point, as the association between prosody interpretation and psychosocial dysfunction was not mediated by mood symptoms or cognitive deficits. In addition, our previous research showed no links between facial affect detection and meaning interpretation with psychosocial dysfunction. However, it remained possible that these social cognitive abilities were associated with psychosocial function indirectly *via* mood symptoms or cognitive deficits. The present results found some evidence for this notion in meaning interpretation, which was indirectly related to psychosocial dysfunction *via* executive function.

Indirect mediation suggests that meaning interpretation does not exert an effect on psychosocial function directly, but rather that meaning interpretation is related to executive function,

which in turn is related to perceived cognitive performance. This finding supports the notion that depressed patients' ability to read the emotions and intentions of others is reliant on executive function, which in turn affects daily functioning. The fact that mediation was only shown for executive function, but not for any other cognitive domains, suggests that greater cognitive complexity inherent to executive functions (e.g., cognitive flexibility, mental updating) is more crucial to theory of mind and psychosocial status than other cognitive functions (e.g., immediate memory). Importantly, follow-up analyses with psychosocial subdomains indicated that executive function was indirectly related to subjective cognitive deficits, but unrelated to other psychosocial subdomains (e.g., autonomy, executive function). The demonstrated link with subjective cognitive deficits suggests that reduced executive function in MDD patients may explain both poor theory of mind and reduced self-perception of cognitive ability. It follows that treating executive deficits may be crucial to attenuating the negative effect of interpersonal difficulties on self-perceived cognitive ability.

The finding that symptom severity did not mediate any relationship between social cognitive abilities and psychosocial function was unexpected. Previous literature has identified that social cognitive deficits and psychosocial dysfunction are elevated in more severely depressed patients (14, 23, 26, 44). Accordingly, it was plausible that greater symptom severity may be the primary mechanism of both social cognitive and psychosocial deficits or, alternatively, may be a significant mechanism by which poor social cognition leads to psychosocial deficits. The present findings provide evidence against this notion, as mood symptoms and cognitive deficits do not mechanistically explain the role of prosody interpretation in reduced psychosocial function. It follows that attenuating mood symptoms is likely insufficient to reduce functional deficits associated with prosody interpretation. Our results underscore the need to develop social cognitive treatments targeting prosody interpretation (27), as current treatments for social cognition in MDD are highly

limited (28). In addition, it may be important to incorporate measures of social cognition in clinical assessment/screening procedures for individuals with major depression. At present, no social cognitive screening tools are specialized for depressed patients; however, our results suggest that the ACS battery may serve for this purpose until such an instrument is developed.

It should be acknowledged that the results demonstrated in this study are modest in size and likely reflect subtle relationships between social cognition and functioning. These results confer with previous studies, suggesting social cognitive deficits are more nuanced in MDD by comparison with other psychiatric illnesses (8). In addition, the cross-sectional nature of our results does not enable conclusions regarding the longitudinal association of social cognition with psychosocial function. Also of note is our primarily Australian-Caucasian cultural-ethnic sample, as the present conclusions may not generalize to other social cohorts. In particular, the relationship between prosody and psychosocial functioning may be different in cultures with different languages or social-perceptive features (e.g., Asian or African). It is possible, for instance, that certain cultures place greater emphasis on facial recognition/body language relative to prosody. Finally, our analyses grouped together currently and previously depressed individuals, and our conclusions thus refer to persons with a lifetime occurrence of MDD. Further validation of the present results is warranted in currently depressed and remitted groups specifically, as well as in other cultural samples.

While existing research had identified links between social cognition and psychosocial function in MDD (22, 28, 45), it was previously unclear whether this relationship was conflated by mood symptoms or nonsocial cognitive function. The present findings provide some clarity in this domain, as symptom severity and cognitive deficits did not explain the relationship between social cognitive abilities and psychosocial dysfunction. The exception was executive function, which indirectly mediated a relationship between theory of mind ability and self-perceived cognitive dysfunction, highlighting the importance of treating executive deficits. Taken together, the present results suggest that social cognitive deficits are a subtle yet distinct feature of MDD, which may contribute independently to the pathology of psychosocial dysfunction.

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ETHICS STATEMENT

This study was carried out in accordance with the recommendations of the National Statement on Ethical Conduct in Human Research, Human Research Ethics Committee, with written informed consent from all subjects. All subjects gave written informed consent in accordance with the Declaration of Helsinki. The protocol was approved by the Royal Adelaide Hospital Human Research Ethics Committee.

AUTHOR CONTRIBUTIONS

BB conceived, designed, coordinated, and supervised the CoFaM-S, from which the current results were derived. MK and BB mutually developed the hypotheses and conceptual outline of the present manuscript. MK analyzed data and wrote the manuscript, while BB edited and provided feedback during the write-up.

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SUPPLEMENTARY MATERIAL

The Supplementary Material for this article can be found online at: <https://www.frontiersin.org/articles/10.3389/fpsyg.2019.00347/full#supplementary-material>

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