Meta-analysis of theory of mind in anorexia nervosa and bulimia nervosa: A specific impairment of cognitive perspective taking in Anorexia Nervosa?

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Abstract

Objective: Deficits in theory of mind (ToM), ability to infer mental states of others, can play a significant role in interpersonal difficulties and/or unawareness of illness observed in AN and other eating disorders including bulimia Nervosa (BN).

Method: Current meta-analysis aimed to summarize available evidence for deficits in ToM in AN and BN and examine the effects of number of study-level variables on observed findings. In this meta-analysis, 15 studies (22 samples with eating disorders) investigating ToM performances of 677 individuals with AN or BN and 514 healthy controls were included.

Results: AN was associated with significant deficits in ToM ($d=0.59$) which were more pronounced in the acute patients ($d=0.67$). Small sized deficits in ToM were observed in BN ($d=0.34$) and recovered AN ($d=0.35$). Both cognitive perspective-taking (ToM-PT) ($d=0.99$) and decoding mental states (ToM-decoding) ($d=0.61$) aspects of ToM were impaired in acute AN. ToM-decoding impairment in BN was modest. There was no evidence for significant ToM-PT deficit in BN. Several study-level variables including longer duration of illness, lower BMI and depressive symptoms were associated with more severe deficits in ToM in AN.

Discussion: ToM deficits, particularly in ToM-PT, can be a specific feature of AN but not BN. ToM impairment can contribute to poor insight, treatment resistance and social impairment in AN.

Key words: Social cognition; theory of mind; eating disorders; anorexia nervosa; bulimia nervosa

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Anorexia nervosa (AN) is a severe mental disorder characterized by distorted body image, fixed beliefs about being overweight and behavioral changes including restriction of food intake and engaging in excessive exercise. **Bulimia Nervosa (BN) is another eating disorder characterized by recurrent episodes of binge eating with (BN) compensatory purging behaviours.** Eating disorders (ED) are predominantly observed in females and associated with significant problems in physical health (1).

While the majority of individuals with AN improve and no longer meet the criteria for this condition in long-term follow-up (2-5), AN is a treatment-resistant condition.

One important feature of eating disorders is impairment in social functioning. AN is associated with poorer social functioning that predates the emergence of ED symptoms (6-8). There is also evidence for social disability and lack of social support in BN (9-10). Cognitive deficits which are evident in eating disorders can contribute to impairment in social functioning. AN has been associated with deficits in central coherence, executive functions and social cognitive deficits including theory of mind (ToM) impairment (11). Weak central coherence is characterized by impaired global but superior local processing (focusing on individual elements at the expense of global picture) and has been reported in AN by a number of studies in AN (12-15). Executive dysfunction, especially, set-shifting ability is also evident in AN (16-18). Recently, meta-analytical studies provided evidence for medium-sized deficits for central coherence and set-shifting in AN (19-20).

These reports also suggested that other eating disorders including bulimia nervosa (BN) have similar deficits in central coherence and set shifting.

Social cognitive impairment might be particularly relevant to understand deficits in social functioning in AN and other eating disorders. Social cognitive abilities are involved in how people perceive and interpret information about themselves, others, and social situations. Individuals with eating disorders have significant difficulty in understanding and accepting the perspectives of others about their body image problems, physical health issues, the need of treatment and behavioral consequences of their condition (21-22).

ToM, the ability to attribute mental states (intentions, feelings, beliefs) to others and explain and predict others' behaviour based on this information, is a critical ability for effective social adaptation and communication.

ToM is a complex aspect of social cognition and is not a unitary concept. One important aspect of ToM is cognitive perspective taking (23). Cognitive perspective taking to infer others' thoughts, beliefs, emotions is an important part of ToM (ToM-PT). This aspect of ToM, is most typically assessed by tasks measuring false belief and indirect speech comprehension (23).

Another aspect of ToM involves processing of affective stimuli (ToM-decoding).

Decoding of affective perceptual stimuli is not only critical for basic emotion recognition, it is also important for inferring complex social emotions, thoughts and other mental states (mental state decoding). The Reading of the mind in the eyes test (RMET) (24) is the most commonly used task to assess mental state decoding aspect of ToM (25).

The majority of available studies in AN have used the RMET to assess ToM. In 2011, Oldershaw et al (26) conducted a very small meta-analysis including only three studies of RMET and reported a deficit of medium effect size. Since 2011, a number of other studies have investigated ToM using the RMET in AN. While some of these studies have found impaired ToM in AN (27-28), a number of them have found no significant differences between AN and healthy controls (29-32). In addition to studies investigating decoding aspects of ToM, emerging evidence suggests that ToM-PT might be also impaired in AN (6,28). However, it is not clear any aspect of ToM is particularly impaired in AN.

A smaller set of studies also investigated ToM deficits in BN but provided conflicting findings. This is a relatively neglected area as social difficulties are also evident in BN. While some studies found ToM deficits in BN (32), others reported no significant difference between healthy controls and BN (28, 30). Like in AN, RMET has been the most common measure used to assess ToM in BN. Several studies also assessed ToM-PT in BN (28, 33). However, studies investigating ToM in BN have typically small sample sizes. Therefore, studies with larger sizes and meta-analyses can potentially provide evidence of ToM impairment in BN.

Another important consideration is potential effect factors related to the severity of acute illness on observed ToM deficits in eating disorders. Depressive symptoms which are evident in clinically unstable patients with AN and BN are associated with ToM deficits (34). Starvation has also negative effects on central coherence and set-shifting abilities in healthy individuals (35) and can potentially impair social cognition.

A meta-analysis of ToM findings in AN and BN to date can estimate the severity of these deficits during acute illness and recovery and can help to understand the heterogeneity by investigating the effects of the potential confounding factors including severity of weight loss and depressive symptoms. A meta-analysis of ToM findings can also overcome the limitation related to small sample size of the studies and can provide a more reliable estimate of the magnitude of deficits in AN and BN. The primary goal of the current meta-analysis was to investigate ToM abilities and their relationship with demographic and clinical variables in eating disorders. Another goal of the meta-analysis was to compare the severity of ToM deficits in AN and BN.

Methods

Study selection

We followed PRISMA guidelines in conducting this meta-analysis (36). A literature search was conducted using the databases Pubmed, PsycINFO, ProQuest and Scopus to identify the relevant studies (January 1990 to March 2016) using the combination of keywords as follows: ('Theory of mind' or 'social cognition') and (eating disorder or anorexia or bulimia). Reference lists of published reports were also reviewed for additional studies. Inclusion criteria were studies that: (1) Compared ToM performances of females with eating disorders and healthy controls; (2) Having a diagnosis of anorexia nervosa or bulimia nervosa. Studies reporting data for eating disorder not-otherwise-specified (EDNOS) bulimic type were included; (3) reported sufficient data to calculate the effect size and standard error of ToM measures. All of the studies meeting inclusion criteria 1 and 2 reported sufficient information to calculate effect sizes. For the appraisal of the study quality, we considered several criteria including peer review status, medium

of publication, diagnostic procedure and procedure for performing social cognitive tests in MRI studies (only outside scanner assessment included). We only included studies that are published in peer-reviewed journals and not included conference papers and theses.

All of the available studies used DSM-IV criteria. In some studies, DSM-IV diagnosis was given by treating team, in others, a structured clinical or eating disorder interview was also used. We conducted a sensitivity to analysis to compare the effect of the diagnostic procedure on results. Two functional MRI studies that used a behavioral task in the scanner to assess neural response to ToM task were not included as they were not designed to detect subtle deficits in social cognition (37-38). Having an unspecified type of EDNOS (1 sample from Medina-Pradas et al (32)), the inclusion of male patients (39), being reported as a conference abstract (1 study) and being based on an overlapping sample with an included study (40-41) were other reasons for exclusion from the meta-analysis.

22 ED samples (reported in 15 published studies) involving 677 (all females) individuals with eating disorders and 514 (females) healthy controls were included (6, 25-30, 42-48) (Table 1) (see the figure 1 for the flow chart of the study selection process). Fourteen of these samples involved 450 individuals with AN including three samples that were in clinical recovery (recovered AN). Other AN samples (acute AN) either included patients in the acute phase of the illness (9 studies, low mean BMI, active symptoms) or patients in subacute phase (2 studies, including a mixture of patients with low and near-normal BMI and relatively improved symptoms) (Table-1). BN group (8 samples) included six samples meeting full diagnostic criteria for BN and two having EDNOS-BN type.

There were no significant differences for age ($d=0.01$, $CI=-0.15-0.17$, $Z=0.13$, $p=0.90$) between individuals with ED and healthy controls. Different depression rating scales were used across studies. Therefore, rather than using raw scores of depression rating scales, effect size (Cohen d) for differences between ED and healthy individuals for depression scores were calculated. ED was associated with significantly more depressive symptoms compared to healthy controls ($d=1.82$, $CI=1.38-2.25$, $Z=8.2$, $p<0.001$) and there were no significant differences between AN and BN ($Q_{bet}=1.7$, $p=0.19$).

Social cognition measures

Several different ToM tasks have been utilized across studies, most commonly the reading the mind from the eyes (RMET) (22) and it was the only measure of mental state decoding aspect of ToM. In RMET, individuals are instructed to look at a series of photographs of just the eye region of the face, and picking which of four words best describe what the person in the photo is thinking or feeling. Studies also included a number of measures of cognitive perspective taking/mental state reasoning aspect of ToM including the faux pas recognition (the task involves recognizing faux pas in series of short stories), the Happe cartoons (attribution of mental states to characters in a cartoon story) and stories (infer mental states in a verbal story context) (49-51).

Statistical analyses

For studies that reported more than one ToM task, pooled effect sizes and standard error values were calculated. Individual tasks analysis for the RMET was also possible. Other than total ToM score and RMET (ToM-decoding), we also had calculated effect sizes for mental state reasoning (ToM-PT).

Meta-analyses were performed using packages in the R environment (OpenMetaAnalyst, Metafor) (52-53). Effect sizes were weighted using the inverse variance method. A random effects model (DerSimonian–Laird estimate) was used. We preferred random effects rather than fixed effects model as heterogeneity of the distributions of effect sizes are expected for social cognition tasks in a clinical sample. The Q and I^2 tests were used to assess the homogeneity of the distribution of weighted effect sizes. Tau-squared (τ^2), an estimate of between-study variance, was used as a measure of heterogeneity in the random effects model. Other than inspection of the funnel plot, Egger's test and Trim and fill method were used to assess publication bias (54-56).

Meta-regression analyses were conducted for **study-level** predictors including the mean age of ED group, the mean duration of ED, the age of onset, body mass index (BMI) and depressive symptoms (effect size for between-group difference). Outcome variables were ToM-Decoding and ToM-PT scores. Meta-regression analyses were conducted using the restricted-information maximum likelihood method with a significance level set at $p < 0.05$. Q_{bet} test was used to statistically compare the severity of ToM deficits in AN and BN. A meta-analysis for the subgroup of individuals with recovered AN was also conducted.

Results

ToM was significantly impaired in ED ($d=0.50$) (Figure-2). Both AN ($d=0.59$) and BN ($d=0.34$) groups had significant ToM impairment. Impairment in ToM was not only significant in **acute** AN ($d=0.67$) but also in recovered AN ($d=0.35$). There was no

significant effect of diagnostic procedure (with or without structural interview) on the severity of ToM deficits ($Q_{bet}=0.99, p=0.32$).

Individuals with AN were impaired in both ToM-decoding (RMET) ($d=0.55$) (Figure-3) and ToM-PT ($d=0.75$) tasks (Figure-4). Effect sizes for the level of impairment in ToM-decoding ($d=0.61$) and ToM-PT ($d=0.99$) were even larger in acute AN. BN was associated with significant impairment in ToM-decoding ($d=0.33$) but not in ToM-PT. The distribution of effect sizes was significantly but modestly heterogeneous for most measures (Table-2). There was evidence of publication bias for mental state reasoning in BN. Trim and fill method suggested a reduced effect size ($d=-0.01$) for ToM-PT in BN.

AN vs BN

AN was significantly more impaired than BN in ToM ($Q_{bet}=4.5, p=0.03$ ($Q_{bet}=7.5, p=0.006$ for acute AN vs BN)). Between-group difference for ToM-PT was the most pronounced ($Q_{bet}=13.0, p<0.001$). AN was also more impaired in the ToM-decoding compared to BN ($Q_{bet}=4.7, p=0.03$). There were no significant differences in severity of ToM deficits of BN and clinically recovered AN ($Q_{bet}=0.28, p=0.60$).

Meta-regression analyses

In AN, the longer duration of eating disorder was significantly associated with more severe deficits in the ToM-decoding ($Z=3.48, p=0.005$) and ToM-PT ($Z=4.78, P<0.001$) measures. Lower BMI was associated with ToM-PT ($Z=-3.37, p=0.008$) but not with the ToM-decoding ($Z=-0.76, p=0.45$). There was a significant relationship between depressive symptoms and deficit in ToM-PT ($Z=3.20, p=0.001$). There was trend level but a non-significant relationship between depressive symptoms and impairment in the ToM-decoding ($Z=1.78, p=0.07$). Age and age of onset of eating disorder had no

significant impact on ToM deficits. Meta-regression analyses were not conducted for BN due to a smaller number of studies available.

Discussion

The current meta-analysis investigated ToM in AN and BN in comparison to healthy controls. Our findings suggested that ToM was impaired in both eating disorders but more severe deficits were found in acute AN. The impairment in ToM-PT was evident only in AN. Depressive symptoms and severity of weight loss contributed to ToM deficits in AN.

The effect sizes of ToM deficits in **acute** AN suggested a large-sized impairment in ToM-PT and medium-sized impairment in ToM-decoding. ToM deficits in recovered AN and BN were characterized by small effect sizes. BN was characterized by impairment in decoding but not reasoning about mental states. ToM, particularly ToM-PT, deficits seem to be specific **feature** of AN among eating disorders.

The relative severity of ToM-PT in AN and its relative specificity for this diagnosis among eating disorders can be important to understand the nature of poor insight in AN. Meta-cognitive abilities (57) and the ability to take other's view about self is an important aspect of awareness of having an illness and realizing its social consequences (58).

Previous studies have shown a relationship between ToM-PT and unawareness of illness in other psychiatric disorders (58-59), and same might be potentially true for AN.

However, it is important to note that poor insight has a complex nature in AN. Poor insight in AN does not always reflect true unawareness. Disagreement with the perspective of others also plays a role in poor insight in AN (60). Other than ToM-PT

deficits, factors such as executive dysfunction, interoceptive unawareness (59), psychological defensiveness and personality traits can contribute to poor insight in AN. On the other hand, both **ToM-PT** and **ToM-decoding** have been associated with poor social functioning in other psychiatric disorders (62-63) and these deficits can potentially contribute to interpersonal problems in AN. Unfortunately, most of the available studies have not explored the relationship between ToM deficits and poor insight or social functioning in AN.

Another consideration is whether ToM deficits in AN represent a trait or state related characteristics of the illness. The significant relationship between low BMI and ToM deficits and small effect sizes observed in recovered AN suggest that effects of starvation on social cognitive brain networks can be the main reason for observed deficits.

Starvation has been associated with weak central coherence and set-shifting deficits in healthy individuals (32) and can potentially have a similar effect on social cognitive abilities. Starvation-related difficulties in cognitive perspective taking can contribute to treatment resistance during acute illness. However, there was evidence for small impairment in recovered and nondepressed AN individuals who had normal BMI ratings. These findings can suggest that sizeable minority of patients with recovered AN can still have difficulties in ToM. This is an important finding as it supports the potential role of ToM deficits as a vulnerability factor for developing eating disorders. It is also important to consider the possibility that ToM deficits in recovered in AN can be a consequence of long-term effects of starvation, rather than related to vulnerability to AN as longer duration of illness was associated with more severe ToM impairment in AN.

BN was associated with only a small-sized impairment in ToM. This is in contrast to findings suggesting a similar level of impairment in set-shifting and central coherence abilities across different eating disorders (19-20). Unlike AN, there was no significant difference for ToM-PT between healthy controls and BN. The small effect size for healthy control-BN differences for ToM-PT in BN suggests that perspective taking abnormalities do not play a significant role in persistent overconcern with body shape and weight in this disorder. Moreover, it can be argued that small sized deficits in ToM-Decoding in BN might be secondary to effects of neurocognitive deficits. However, it was not possible to explore effects of neurocognition on ToM in BN in the current meta-analysis due to lack of sufficient information in studies included.

It is also important to consider the role of co-morbidity in ToM deficits in eating disorders. In the current meta-analysis, similar to studies in Major Depression (31), depressive symptoms seem to contribute to ToM impairment in acute illness. However, depressive symptoms cannot be the main factor underlying ToM deficits in eating disorders as ToM deficits were mild in individuals with BN who were as depressed as acute AN patients. Another potential consideration is the role of autistic features in ToM deficits in AN. One recent study suggested that ToM impairment might be only evident in recovered AN with co-morbid autism spectrum conditions (40). It remains to be further investigated whether subgroups of individuals with ToM deficits in recovery have more autistic traits than other individuals with AN.

Current meta-analysis has several limitations. The most important limitation is the cross-sectional nature of available evidence. No sufficient information regarding a number of variables which can have an effect on ToM deficits (i.e subtypes of AN (restrictive type vs binge/purging type), co-morbid autistic traits, neurocognitive and IQ assessment, premorbid functioning) were available in the majority of the studies included in the current meta-analysis. Also, statistical comparisons of deficits in AN, recovered AN and BN were based on studies separately comparing these samples with controls. Indirect comparisons have limitations compared to head to head comparisons of samples and these findings should be considered preliminary. There is a need for further studies comparing social cognitive performances of different eating disorders. Another limitation was that a single pooled effect size for different tasks used to measure ToM-PT was used due to the absence of sufficient data to conduct individual task meta-analyses. It is important to use consistent ToM-PT measures in future studies to improve the comparability of different studies. The relationship between ToM deficits and clinical features including functional impairment, depression and insight have been rarely investigated. Also, it is not known whether ToM deficits observed in AN can be extended to male patients with eating disorders as only one study has investigated ToM-decoding in a mixed sample of eating disorders and found no significant difference with healthy controls (38). To our knowledge, no study has investigated ToM-PT in males with eating disorder.

As a conclusion, AN is associated with significant deficits in ToM. Social cognitive deficits in BN are modest. Deficits in ToM-PT can be a relatively specific feature of AN.

Current meta-analysis was helpful in overcoming limitation related to small sample sizes of individual studies. As a result, we were able to provide an estimate of ToM deficits in AN and BN. Current findings also supported a significant relationship between ToM deficits, depression, and BMI scores. Follow-up studies can reveal the acute and long-term effects of starvation on ToM in AN. Behavioral implications of ToM deficits and their relationship with autistic traits, social functioning and insight in AN should be clarified further.

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Table-1 Studies included in the meta-analysis

Study	Sample	Age	ToM measure	BMI	Characteristics	Criteria	Depressive symptoms	Outcome
Adenzato et al 2012 (29)	30 AN 32 HC	19.7	RMET	15.1	Duration=3.6 16 R, 14 BP	DSM-IV Structured interview	BDI=26.4	No difference
Calvo-Sagardoy et al 2014 (30)	41 HC		RMET			DSM-IV		
	39 AN			17.3	Subacute			No difference
	9 BN			21.8				No difference
Gal et al 2011 (42)	20 AN 20 HC		Faux pas	16.5	R type	DSM-IV		Impaired
Gillberg et al 2010 (6)	42 AN (Recovered) 46 HC		Happe cartoons		Not persisting in most (12 % active ED) Duration=18	DSM-IV		Impaired
Harrison et al 2009 (43)	20 AN 20 HC	26.3	RMET	15.8	Duration=7.2	DSM-IV Notes and ED interview	HADS=9.5	Impaired
Harrison et al 2010 (27, 44)	90 HC		RMET			DSM-IV Notes and ED interview		
	50 AN	26.7		15.4	Duration=9.2		DASS=18 (median)	Impaired
	35 AN (Recovered)	29.0		21.2	Duration=5.5		DASS=3 (median)	Impaired
	50 BN	27.5		20.1	Duration=8.4		DASS=12	No difference
Kenyon et al 2012 (45)	57 HC		RMET, RMF			DSM-IV Clinical and ED interview		
	48 BN	28.0		24.5	Duration=10.7		DASS=11.2	No difference
	34 EDNOS	27.6		25	Duration=9.9		DASS=11.2	No difference
Laghi et al 2014 (33)	16 HC		T.h.o.m.a.s			DSM-IV		
	16 BN	26.3		24.1	Duration=8.5			Impaired
	16 EDNOS -BN type	26.7		24.5	Duration=8.7			Impaired
Laghi et al	40 AN	14.9	RMET	15.8	Duration=1.3	DSM-IV		No

2015 (31)	40 HC					ED interview		difference
Medina-Pradas et al 2012 (32)	39 HC		RMET			DSM-IV Structured interview		
	44 AN	26.8		15.8	Duration=9.9		BDI=21.2	No difference after correction for BMI, education and age
	30 BN	26.8		20.9	Duration=10.3		BDI=25.3	Impaired
	23 EDNOS	26.0	Exclude	25.8	Duration=7.3		BDI=28.0	Impaired
Oldershaw et al 2010 (46)	47 HC		RMET RMF RMV					
	40 AN			16.0	Duration=7.4	DSM IV BMI <18.5	HADS=9.1	Impaired
	24 AN (recovered)			20.8	Duration=5.6	DSM IV BMI >19.0, no symptoms for a year	HADS=3.2	No difference
Russell et al 2009 (47)	22 AN 22 HC	26.7	RMET Happe cartoon	15.3	Duration=9.5 17 R, 5 BP	DSM IV		Impaired
Tapajoz et al 2013 (28)	24 HC		RMET Faux pas			DSM IV		
	24 AN	24.5		18.1	Duration=7.8 Subacute		BDI=21.0	Impaired
	24 BN	24.4		25.0	Duration=7.7		BDI=21.9	No difference
Tchanturia et al 2004 (48)	20 AN 20 HC	27.4	Happe cartoon Happe stories	15.8	10 R, 10 BP	DSM IV	HADS=10.7	Impaired

AN=Anorexia Nervosa, BN= Bulimia Nervosa, EDNOS=Eating disorder-not otherwise specified
 HC=Healthy controls, R=Restrictive subtype; BP=Binge-Purge subtype,
 RMET=Reading the mind from the eyes test, BDI=Beck Depression Inventory, HADS=Hospital anxiety and depression scale, DASS=Depression anxiety stress scale

Table-2 mean weighted effect sizes for face emotion recognition between individuals with eating disorder and controls

Test	Sample	ED	HC	d	95 % CI	Z	P	Q-test p	τ^2	Bias
ToM	22	677	514	0.50	0.33-0.68	5.76	<0.001	<0.001	0.10	
<i>AN</i>	14	450	441	0.59	0.37-0.81	5.24	<0.001	<0.001	0.11	
-Recovered	3	101	183	0.35	0-0.70	1.95	0.05	0.14	0.05	
-Acute	11	349	395	0.67	0.40-0.94	4.91	<0.001	0.001	0.13	
<i>BN</i>	8	227	267	0.34	0.09-0.58	2.68	0.008	0.08	0.05	
RMET^{decode}	17	563	412	0.47	0.29-0.65	5.22	<0.001	0.001	0.08	
<i>AN</i>	11	368	355	0.55	0.31-0.79	4.49	<0.001	<0.001	0.10	0.11
-Recovered	2	77	136	0.32	-0.20-0.83	1.20	0.13	0.01	0.09	
-Acute	9	291	355	0.61	0.33-0.89	4.26	<0.001	0.002	0.12	
<i>BN</i>	6	195	251	0.33	0.09-0.57	2.72	0.007	0.001	0.03	0.27
ToM-PT	12	330	252	0.55	0.23-0.87	3.39	<0.001	<0.001	0.24	
<i>AN</i>	7	192	179	0.75	0.33-1.17	3.50	0.0005	<0.001	0.24	0.19
-Recovered	2	66	93	0.22	-0.29-0.73	0.87	0.39	0.12	0.08	
-Acute	5	126	133	0.99	0.53-1.44	4.25	<0.001	0.02	0.18	
<i>BN</i>	5	138	97	0.23	-0.15-0.61	1.19	0.23	0.04	0.11	0.006

D=Effect size of between-group difference, Bias= p value of the Egger's test,, HC=Healthy controls, d=Cohen d, decode= ToM-decoding

Figure-1 Flow Diagram for meta-analysis of ToM in AN and BN

Accepted Article

Figure 2 Forest plot of ToM in acute AN, recovered AN and BN

Accepted Article

Figure 3 Forest plot of RMET in acute AN, recovered AN and BN

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Figure 4 Forest plot of ToM-PT in acute AN, recovered AN and BN

Accepted Article

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Statement of interest: None to declare

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Figure-1 Flow Diagram for meta-analysis of ToM in AN and BN

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Figure 2 Forest plot of ToM in acute AN, recovered AN and BN
269x208mm (144 x 144 DPI)

Accep

Figure 3 Forest plot of RMET in acute AN, recovered AN and BN
269x182mm (144 x 144 DPI)

Accepted

Figure 4 Forest plot of ToM-PT in acute AN, recovered AN and BN
269x157mm (144 x 144 DPI)

Accepted

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