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# ACT-i, an insomnia intervention for autistic adults: a pilot study

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## Abstract

**Background:** Insomnia and disturbed sleep are more common in autistic adults compared with non-autistic adults, contributing to significant social, psychological and health burdens. However, sleep intervention research for autistic adults is lacking.

**Aims:** The aim of the study was to implement an acceptance and commitment therapy group insomnia intervention (ACT-i) tailored for autistic adults to examine its impact on insomnia and co-occurring mental health symptoms.

**Method:** Eight individuals (6 male, 2 female) aged between 18 and 70 years, with a clinical diagnosis of autism spectrum disorder, and scores ranging from 9 to 26 on the Insomnia Severity Index (ISI) participated in the trial. Participants were assigned to one of two intervention groups (4 per group) within a multiple baseline over time design for group. Participants completed questionnaires pre-intervention, post-intervention, and at 2-month follow-up, actigraphy 1 week prior to intervention and 1 week post-intervention, and a daily sleep diary from baseline to 1 week post-intervention, and 1 week at follow-up.

**Results:** At a group level there were significant improvements in ISI ( $\lambda^2=10.17$ ,  $p=.006$ ) and HADS-A (anxiety) ( $\lambda^2=8.40$ ,  $p=.015$ ) scores across the three time points. Clinically reliable improvement occurred for ISI scores ( $n=5$ ) and HADS-A scores ( $n=4$ ) following intervention. Client satisfaction indicated that ACT-i was an acceptable intervention to the participants (median 4 out of 5).

**Conclusions:** This pilot study with eight autistic adults indicates that ACT-i is both an efficacious and acceptable intervention for reducing self-reported insomnia and anxiety symptoms in autistic adults.

**Keywords:** acceptance and commitment therapy; autistic adults; insomnia; sleep disturbance

## Introduction

Disturbed sleep is associated with poor mental and physical health outcomes (Chattu *et al.*, 2019). This has high salience and impact for adults with an autism spectrum disorder (autism) diagnosis who have increased mental and physical health problems compared with the general population (Croen *et al.*, 2015). Disturbed sleep is more common in autistic adults compared with non-autistic adults (Morgan *et al.*, 2020) and adds significantly to their social, psychological and health burdens. While autism affects at least 1% of adults (Brugha *et al.*, 2016) translational research about effective interventions for insomnia in autistic adults is lacking. This knowledge gap contributes to risk associated with no treatment or over-prescribing poorly

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chosen medications, which lack long-term efficacy data and may cause harm (Qaseem *et al.*, 2016).

Regardless of age, cognitive ability or core autistic traits, most autistic individuals have significant sleep disturbance, primarily insomnia symptoms including increased sleep onset latency (SoL) and wake after sleep onset (WASO), reduced sleep efficiency (SE; proportion of time spent in bed where one is asleep) and total sleep time (TST) (Schreck and Richdale, 2020). Sleep differences in autism emerge early in life (Humphreys *et al.*, 2014) occurring in children (Malow *et al.*, 2006; Richdale and Prior 1995), adolescents (Baker *et al.*, 2013; Goldman *et al.*, 2017) and adults (Baker and Richdale, 2015; Baker and Richdale, 2017; Croen *et al.*, 2015; Hohn *et al.*, 2019). In Australian and Irish samples, 64% and 89% of autistic adults, respectively, reported poor sleep quality as opposed to 46% of non-autistic Australian adults aged 15 to 80 years (Jovevska *et al.*, 2020; Leader *et al.*, 2021) while 28% of Australian and 55% of Dutch adults met insomnia criteria (Baker and Richdale, 2015; Hohn *et al.*, 2019) and 44% had a circadian sleep-wake disorder (Baker and Richdale, 2017). Thus, poor sleep is a significant issue for a large proportion of autistic adults.

Poor sleep in autistic adults also has a wider impact as it is associated with increased risk of daytime fatigue (Baker and Richdale, 2015; Baker *et al.*, 2013), reduced quality of life (Deserno *et al.*, 2019; Lawson *et al.*, 2020), unemployment (Baker *et al.*, 2019a), increased core autistic traits (Hohn *et al.*, 2019; Phung and Goldberg, 2017) and a mental health diagnosis (Jovevska *et al.*, 2020) or increased psychiatric symptoms (Gisbert Gustemps *et al.*, 2021). Poor mental health and hyper-arousal may play a significant role in the development and maintenance of insomnia symptoms in autistic individuals. American private healthcare provider data show that 29.1% of autistic adult members (18+ years) had an anxiety disorder (Croen *et al.*, 2015), while clinically significant anxiety and depression symptoms were found in a 25–40% of autistic individuals aged from late adolescence to old age (Hollocks *et al.*, 2019; Uljarević *et al.*, 2019). In young autistic adults, insomnia symptomatology has been related to both anxiety and depression (Tani *et al.*, 2003). Additionally, pre-sleep somatic arousal is reported as significantly associated with insomnia symptoms, which together with reduced pre-sleep cortisol suggested that autistic adults may be chronically hyper-aroused (Baker *et al.*, 2019b).

Despite accumulating evidence that autistic adults have problematic sleep associated with a range of negative sequelae, sleep intervention research focusing on adults is lacking. Current research primarily focuses on behavioural interventions (Carnett *et al.*, 2020) or melatonin (Gringras *et al.*, 2017; Guérolé *et al.*, 2011) for autistic children and adolescents. Cognitive and behavioural therapy for insomnia (CBT-I) is the empirically supported intervention for non-autistic individuals with insomnia, including those with insomnia and co-occurring mental health conditions (Taylor and Pruiksma, 2014; van Straten *et al.*, 2018). In CBT-I, cognitive therapy (CT) addresses dysfunctional beliefs, and worry and thoughts about sleep, while behaviour therapy (BT) supports behaviour change in sleep practices (van Straten *et al.*, 2018). However, the core social-communicative and behavioural features of autism, including lack of cognitive and behavioural flexibility, and co-occurring executive function difficulties suggest that at least some autistic adults may have difficulty grasping and applying the cognitive concepts of traditional CBT (Kiep *et al.*, 2015). There is some evidence that adapted or augmented CBT-I may be effective for improving sleep among autistic children (McCrae *et al.*, 2020; McCrae *et al.*, 2021). Nevertheless, currently there is no CBT intervention specifically adapted for autistic adults and in one study 21% of autistic adults reported they would not participate in CBT-based interventions, and CBT was only ranked sixth among the approaches they would use (76%) (Benevides *et al.*, 2020). Thus, it is necessary to provide autistic individuals with a range of efficacious approaches to address insomnia and mental health difficulties.

An alternative psychological approach to insomnia treatment is acceptance and commitment therapy (ACT), a contextual CBT that promotes behavioural flexibility through the development

of acceptance of insomnia, orientation to personal values, and the use of mindfulness strategies (Hayes *et al.*, 2012). In a recent review it was concluded that ACT was an effective treatment for insomnia (Salari *et al.*, 2020). ACT has been shown as effective in improving sleep quality and quality of life in non-autistic, CBT-I non-responders (Dalrymple *et al.*, 2010; Hertenstein *et al.*, 2014). More recently ACT has demonstrated promise as a primary insomnia treatment for people with chronic pain (Zetterqvist *et al.*, 2018), chronic insomnia (Zakiei and Khazaie, 2019), and as a self-help, internet intervention for insomnia (Lappalainen *et al.*, 2019; Zakiei and Khazaie, 2019). Study outcomes have reported changes via sleep questionnaires (Lappalainen *et al.*, 2019; Zakiei and Khazaie, 2019; Zetterqvist *et al.*, 2018) and sleep diaries (Zakiei and Khazaie, 2019; Zetterqvist *et al.*, 2018), and included follow-up of between 2 and 6 months.

ACT presents a promising alternative to CBT for autistic individuals, as it addresses some of the difficulties associated with core symptoms of autism, including lack of cognitive flexibility (Geurts *et al.*, 2009) and perseveration or rumination (Gotham *et al.*, 2014; Patel *et al.*, 2017). The ACT model incorporates acceptance and mindfulness, with an orientation toward values-based action (Hayes *et al.*, 2012). It takes an experiential and pragmatic approach, ensuring that sessions are interactive, concrete and focused on behaviour change, which is an important feature for the adaptation of psychosocial interventions in the guideline for adults with autism (National Institute for Health and Care Excellence, 2016). In contrast to CT, ACT takes a behavioural approach to cognition where the function of thoughts, worries and urges is explored with reference to personal goals (Hayes, 2016).

Mindfulness-based interventions have been used to successfully treat mental health conditions in autistic individuals, including adults (Cachia *et al.*, 2016). Mindfulness-based cognitive therapy adapted for autistic adults ( $n=20$ ) effectively reduced a range of psychological symptoms compared with a treatment as usual group ( $n=21$ ) following a 9-week program (Spek *et al.*, 2013). Using the same adapted intervention, Kiep *et al.* (2015) found improvements in anxiety, depression, rumination, sleep problems and well-being in a sample of 50 autistic adults. Two studies have compared the effectiveness of mindfulness-based therapies and CBT and found that they were both equally effective in reducing anxiety (Gaigg *et al.*, 2020; Sizoo *et al.*, 2017) and depression (Sizoo *et al.*, 2017) among autistic adults.

A 6-week ACT intervention was successful in reducing stress, emotional problems and hyperactivity in 15 autistic individuals aged 13–21 years, compared with a no intervention control group of 12 similar age autistic youth, with maintenance at 2-month follow-up (Pahnke *et al.*, 2014). Most recently a 12-session ACT intervention, with modifications to increase structure, including opportunity for individuals to seek support and clarification post-session, adaptation of examples and colour-coded worksheets, was used in a feasibility study addressing psychological symptoms in 10 autistic adults (Pahnke *et al.*, 2019). Participants improved in: (1) stress and psychological flexibility at post-treatment; (2) functional impairment (social) and cognitive fusion at post-treatment, and 3-month follow-up; and (3) satisfaction with life and depressive symptoms at 3-month follow-up. Overall, participants found the intervention credible. Thus, psychological interventions that include ACT, together with BT, may be an efficacious and acceptable approach for treating insomnia in autistic adults.

The aim of this study was to implement an ACT/BT group insomnia intervention (ACT-i) tailored for autistic adults and to examine its impact on (1) insomnia and (2) co-occurring mental health symptoms (anxiety, depression, general psychological distress, and experiential avoidance). Based on limited existing literature for autism, we hypothesised that the intervention would lead to clinical improvement on insomnia symptoms as measured by the Insomnia Severity Index (ISI), and anxiety as measured by the Hospital Anxiety and Depression Scale-Anxiety (HADS-A), and both would be maintained at 2-month follow-up. No hypotheses concerning the other mental health measures were made, other than we expected that they would not worsen if insomnia on the ISI showed clinical improvement. We

also explored whether there was any change in sleep (SoL, TST, WASO, SE) on a sleep diary and objectively via actigraphy.

## Method

### Design

A concurrent, group (four individuals per group) multiple baseline design was used to implement treatment and follow-up. There were four phases: baseline (A), intervention (B), post-intervention (C), and 2-month follow-up (D).

### Participants

Twelve adults expressed interest in the study and completed the eligibility questionnaires. To meet eligibility criteria, participants had to be aged 18–70 years, have a clinical diagnosis of autism spectrum disorder and score  $\geq 8$  on the ISI (Bastien *et al.*, 2001) indicating at least subthreshold insomnia (Morin *et al.*, 2011). Of those expressing interest in the study, 11 met the screening criteria (one did not have an autism diagnosis), but only eight (6 male and 2 female) were able to attend the available session dates. Participants were assigned to one of two intervention groups (4 per group) within a multiple baseline over time design for group. One individual who began therapy disengaged before the end (103); they completed pre-/post-intervention questionnaires, but not follow-up.

### Primary outcome measure

*Insomnia Severity Index* (Bastien *et al.*, 2001; Morin *et al.*, 2011)

The ISI has seven self-report items related to insomnia over the past 2 weeks and is answered on a 5-point Likert scale. A score  $\geq 8$  (99.4% sensitivity, 91.8% specificity in a clinical sample; 95.8% sensitivity, 78.3% specificity in a community sample) on the ISI is indicative of subclinical insomnia; scores  $\geq 15$  indicate moderate to severe insomnia.

### Secondary outcome measures

*Hospital Anxiety and Depression Scale* (HADS; Zigmond and Snaith, 1983)

The HADS measures self-reported anxiety and depression with seven items answered on a 4-point scale on each subscale. It assesses changes in anxiety (HADS-A) and depression (HADS-D) symptomatology in response to intervention. Scores  $\geq 8$  on either subscale indicates elevated symptoms, with scores  $> 10$  indicating clinical level of symptoms. Factorisation of the HADS on 151 adolescents and young adults with ASD confirmed an identical two-factor structure to the original HADS. Internal consistency was good for the anxiety scale ( $\alpha=.82-.84$ ) and acceptable for depression ( $\alpha=.62-.72$ ) (Uljarević *et al.*, 2018).

### Actigraphy

Participants completed 7 days of actigraphy 7 days prior to the first interventions session, 7 days immediately following the last intervention session, and for 7 days two months after the intervention. Actigraphy data was digitised in 1 min epochs using a sensitivity of 0.025g and a bandwidth of 0.35–7.5Hz and analysed with Respironics Actiware 6 software. TST, SoL, WASO and SE were evaluated (Baker and Richdale, 2015).

*Sleep diaries*

Participants completed an online sleep diary every day beginning at baseline 7 days (group 1) and 21 days (group 2) prior to their first group session until 7 days after the final intervention session. Participants also completed a 7-day sleep diary at the 2-month follow-up. In line with actigraphy data, TST, SoL, WASO and SE were calculated.

*Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989)*

The PSQI is an 18-item self-report questionnaire assessing overall sleep quality and problems related to poor sleep in the past month (total score  $\alpha=.83$ ). A global score  $>5$  indicates poor sleep quality. Seven component scores can also be calculated: sleep quality, latency, duration, disturbances, medication, habitual sleep efficiency, and daytime dysfunction. Internal consistency for the total score in autistic adults ( $\alpha=.68$ ) was acceptable (Baker and Richdale, 2015).

*CORE-10 (Barkham et al., 2013)*

The CORE-10 measures common psychological distress in primary care mental health settings and can be used to assess change in response to intervention. There are 10 items rated on a 5-point Likert scale. Higher scores indicate greater psychological distress. It has excellent internal reliability ( $\alpha=.90$ ) with a clinical cut-off of 11 for general psychological distress.

*Brief Experiential Avoidance Questionnaire (BEAQ; Gámez et al., 2014)*

The BEAQ has 15 items measuring unwillingness to remain in contact with distressing emotions, thoughts, urges and physical sensations even when doing so creates long-term harm. The BEAQ can be used to assess response to ACT components of intervention. Items are rated on a 6-point Likert scale, and it is validated in psychiatric and community samples with good internal consistency (Gámez et al., 2014; Kirk et al., 2019).

*Sleep Anticipatory Anxiety Questionnaire (SAAQ; Bootzin et al., 1994)*

The SAAQ measures anxiety related to pre-sleep arousal. Ten items are answered on a 4-point Likert scale with two subscales examining cognitive and somatic arousal. Higher scores indicate greater arousal, and it has excellent internal consistency in autistic adolescents ( $\alpha=.92$ ) and non-autistic adolescents ( $\alpha=.87$ ) (Richdale et al., 2014).

*Flinders Fatigue Scale (FFS; Gradisar et al., 2007)*

The FFS measures daytime fatigue associated with insomnia. It has seven items; six are rated on a 5-point Likert scale and one item asks the time of day fatigue is experienced (multiple responses can be given and the score for this item is the sum of checked times). Higher scores indicate greater fatigue, and internal consistency in autistic and non-autistic adults is good ( $\alpha=.84$ ) (Baker and Richdale, 2015).

*Social validity*

Social validity of the intervention was measured using a client satisfaction questionnaire with five items answered on a 5-point Likert scale, where higher scores indicate greater satisfaction. The items measure understanding of information and strategies presented, helpfulness of intervention in improving sleep, ability to use strategies in the future, approval of procedures used in intervention, and how likely they are to recommend the intervention to a friend. Participants were also asked three open-ended questions asking what was the most useful

about the intervention, what could be done to improve the intervention, and if they had any other comments.

### **Intervention**

Our group intervention (4×1.5 h, facilitator-led sessions delivered over 5 weeks; 3–4 participants/group) combined two broad components – behaviour therapy (BT) for sleep disturbance and mindfulness and values (ACT) (see Supplementary Table 1 for intervention details). The intervention was developed with consultation from three autistic adults for suitability, language use, and presentation of materials. It was based on current models of incorporating mindfulness/acceptance within BT for insomnia (Ong *et al.*, 2012), drawing on the psychological flexibility model (Hayes *et al.*, 2012), and using material from the Sleep School self-help materials (Meadows, 2014). The intervention aims were to decrease cognitive, emotional and somatic arousal, alter unhealthy sleep-related behaviours, and change sleep misperceptions. The ACT component was designed to promote flexibility in behaviour around sleep (Hayes *et al.*, 2006; Hayes *et al.*, 2012; Lundh, 2005; Ong *et al.*, 2012). It involved education about effects of control and suppression on arousal, rebound effects with thoughts, images and emotions; undermining over-control of sleep routine; introduction of mindfulness and acceptance for arousal reduction and emotion regulation; and practice of non-judgemental awareness toward inner experiences. Flexibility in responding to unwanted feelings, thoughts and images; and undermining unhelpful control (where it influences sleep hygiene) and pragmatism were promoted through experiential exercises and metaphor. Persistence in engaging in effective sleep hygiene and restriction was promoted by linking changes in sleep patterns to personal values (chosen life directions). Based on CBT-I community education programs (Swift *et al.*, 2012), the BT component included psychoeducation about sleep, functional analysis of efforts to control sleep, sleep hygiene education, stimulus control of bedroom, worry reduction, and developing a plan for sleep routine changes.

### **Procedure**

The study was advertised through Olga Tennison Autism Research Centre (OTARC) participant registry, La Trobe University Psychology Clinic, local autism associations and support networks (e.g. Amaze, Asperger Victoria) and via online media and community outlets. Interested participants contacted author L.P.L. and were sent a link to a Qualtrics survey, containing the Participant Information Statement and consent. Those consenting answered screening questionnaires including demographics and the ISI. Those meeting inclusion criteria were invited to attend an individual session at La Trobe University Psychology Clinic with a registered provisional psychologist or psychologist. At the clinic visit, participants were screened for serious mental health difficulties, sleep apnoea and risk of harm to self/others. The project was then explained in detail and participants were provided with information regarding questionnaires, sleep diary, and actigraphy. Figure 1 outlines the study procedure.

### **Data analysis**

Within-group comparisons for the questionnaire measures were made across the three time points (pre-intervention, post-intervention, and 2-month follow-up) using one-way, repeated measures Friedman tests. Any significant tests were followed up using the Wilcoxon signed ranks test, with alpha adjusted for three comparisons ( $\alpha=.017$ ), with effect size ( $r$ ) calculated (Pallant, 2011). Wilcoxon signed ranks tests were also used to determine if there were any

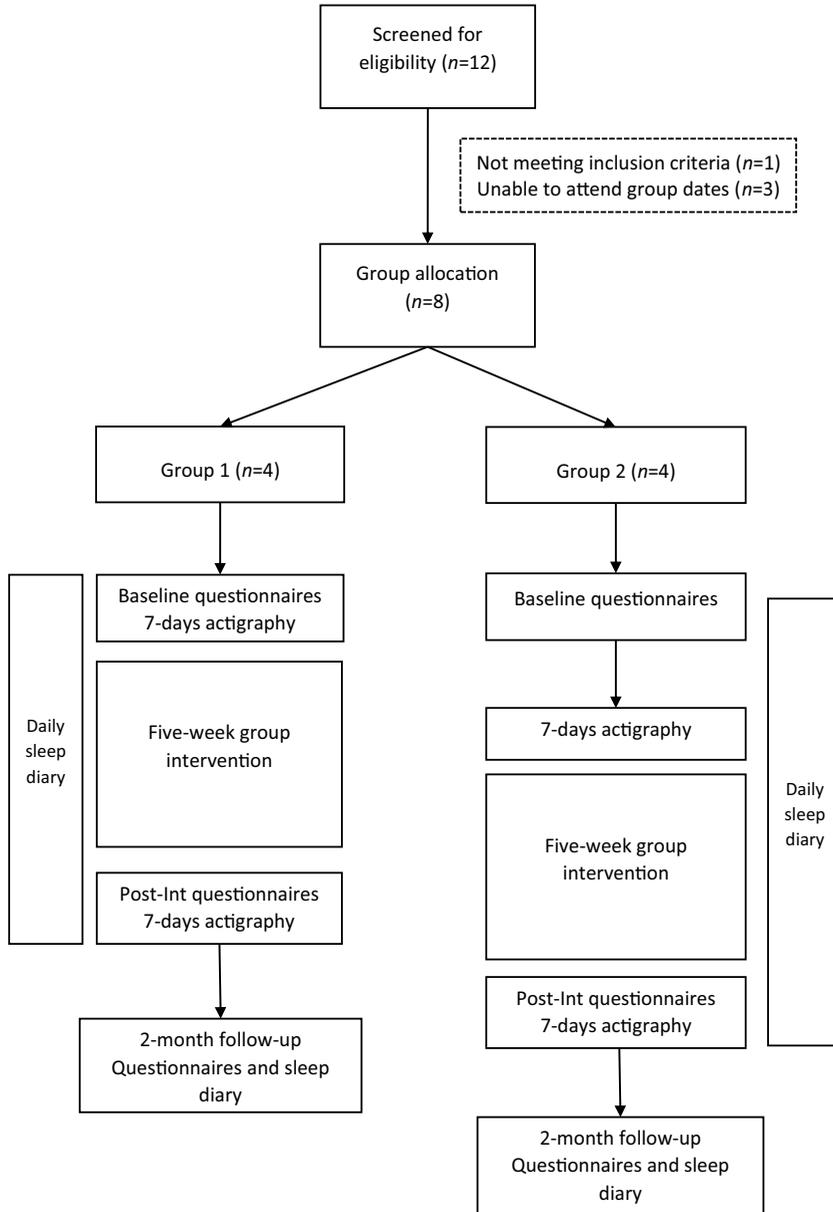


Figure 1. Trial procedure.

significant changes within participants from pre- to post-intervention for the actigraphy measures SoL, TST, SE and WASO.

Within-participant analyses of sleep diary data were conducted across the study phases (A–E), using a measure of non-overlap for single case data, the Tau-U (Parker *et al.*, 2011). Consistent with the recommendations of Fingerhut *et al.* (2021), we calculated  $TauU_{adj}$  scores. This involved comparisons of between-pairs of consecutive phases for each participant, for the degree of non-overlap of data, and determining whether there is baseline trend to control for (Tarlow, 2017). Analyses were conducted for SoL, SE, TST and WASO. Within-participant comparisons using reliable change indices (Jacobson and Truax, 1992) were also made across the three time

**Table 1.** Group means and standard deviations for standardised outcomes measures at the three time points

	Baseline		Post-intervention		Follow-up	
	<i>n</i>	Mean ( <i>SD</i> )	<i>n</i>	Mean ( <i>SD</i> )	<i>n</i>	Mean ( <i>SD</i> )
ISI	8	17.88 (6.1)	8	12.63 (4.2)	6	10.8 (5.8)
HADS Anxiety	8	13.00 (4.8)	8	9.63 (3.7)	6	10.33 (4.2)
HADS Depression	8	9.75 (5.2)	8	7.63 (4.1)	6	6.50 (3.1)
PSQI	8	10.88 (4.2)	7	8.43 (4.2)	6	7.50 (3.3)
CORE-10	8	20.25 (7.1)	8	12.50 (4.8)	6	15.17 (2.3)
BEAQ	8	59.75 (15.8)	8	53.50 (12.5)	6	55.00 (10.4)
SAAQ	8	28.50 (4.1)	8	23.75 (4.1)	6	23.00 (7.0)
FFS	8	16.75 (9.7)	8	16.50 (6.2)	6	13.83 (7.4)

points (pre-intervention, post-intervention, and 2-month follow-up) for the questionnaire measures.

## Results

All participants completed the baseline and post-intervention questionnaires, but one person incorrectly filled out the PSQI at post-intervention and a total score could not be calculated. Two participants did not complete the 2-month follow-up surveys. The means and standard deviations for each measure at the three time points are provided in Table 1. Six of the eight participants had moderate to severe insomnia ( $ISI \geq 15$ ) on entry. All participants' ISI scores reduced following intervention and of the six participants completing follow-up, ISI scores reduced further in four participants, and two individuals no longer had insomnia (Supplementary Table 2). Four of the eight participants recorded taking regular medication throughout the trial including SSRIs and stimulants. One participant reported taking a stimulant at the beginning of the trial but did not complete the remaining diaries. One participant reported intermittently using medication for sleep and/or pain including benzodiazepine, oxycodone and temazepam. Full details of medication use for each participant is summarised in Supplementary Figure 1.

### Primary outcome measure: group level analyses

There was a significant change in ISI scores over the three time points,  $\lambda^2=10.17$ ,  $p=.006$ . Wilcoxon signed ranks identified that there was a significant reduction, with large effect size, in ISI scores from baseline to post-intervention ( $Z=-2.53$ ,  $p=.011$ ,  $r=-.632$ ) but, while the ISI scores fell from baseline to follow-up, this comparison ( $Z=-2.21$ ,  $p=.027$ ,  $r=-.591$ ) did not reach adjusted clinical significance, although the effect size was large. The drop in ISI score from post-intervention to follow-up ( $Z=-1.52$ ,  $p=.129$ ,  $r=-.406$ ) showed a moderate effect size but was not significant.

### Secondary outcome measures: group level analyses

Six participants exceeded the clinical threshold for anxiety on the HADS-A at baseline; HADS anxiety scores changed significantly across the intervention period,  $\lambda^2=8.40$ ,  $p=.015$ . Despite a reduction in scores, follow-up Wilcoxon signed rank tests did not meet adjusted statistical significance for baseline to post-intervention ( $Z=-2.21$ ,  $p=.027$ ,  $r=-.553$ ) or baseline to follow-up ( $Z=-2.02$ ,  $p=.043$ ,  $r=-.540$ ) although the effect size was large for both comparisons. There was no change from post-intervention to follow-up ( $Z=-.96$ ,  $p=.336$ ,  $r=-.257$ ) with small effect size. The Friedman test was non-significant for HADS depression

sores,  $\lambda^2=4.80$ ,  $p=.091$ . Four participants exceeded the HADS-D clinical threshold for depression at baseline. However, while there was a reduction in depression symptomatology across the three time points, this did not reach statistical significance.

PSQI scores significantly changed over the three time points,  $\lambda^2=6.78$ ,  $p=.034$ . Examining the means shows a reduction in scores over time, but the follow-up tests did not reach adjusted statistical significance for baseline to post-intervention ( $Z=-2.13$ ,  $p=.033$ ,  $r=-.550$ ) or baseline to follow-up ( $Z=2.02$ ,  $p=.043$ ,  $r=-.540$ ) but both effect sizes were large. There was no change from post-intervention to follow-up ( $Z=-.18$ ,  $p=.854$ ,  $r=-.050$ ).

There was also a significant change in BEAQ scores,  $\lambda^2=6.33$ ,  $p=.042$ , but the follow-up tests did not reach adjusted statistical significant for baseline to post-intervention ( $Z=-2.37$ ,  $p=.018$ ,  $r=-.593$ ) or baseline to follow-up ( $Z=-1.99$ ,  $p=.046$ ,  $r=-.532$ ) but effect sizes were large. There was no change from post-intervention to follow-up ( $Z=-.21$ ,  $p=.883$ ,  $r=-.056$ ). A significant change in SAAQ scores was found across the three time points,  $\lambda^2=8.45$ ,  $p=.015$ . However, the follow-up analyses did not reach adjusted statistical significance for baseline to post-intervention ( $Z=-2.37$ ,  $p=.018$ ,  $r=-.593$ ) or baseline to follow-up ( $Z=-2.20$ ,  $p=.028$ ,  $r=-.588$ ) but effect sizes were large. There was no change from post-intervention to follow-up ( $Z=-.13$ ,  $p=.892$ ,  $r=-.035$ ). The Friedman test was non-significant for both the CORE-10,  $\lambda^2=3.00$ ,  $p=.223$ , and FFS,  $\lambda^2=2.70$ ,  $p=.260$ .

### **Actigraphy**

Table 2 presents the data from the actigraphy measured at one week pre-intervention and one week post-intervention. Within-subject data and comparisons are presented. None of the participants had a significant change in their actigraphy data from pre- to post-intervention.

### **Single case analyses**

#### *Reliable change indices for questionnaire measures*

Table 3 presents the reliable changes indices for each of the study measures, comparing post-intervention (phase C) scores with the baseline (phase A). Scores had to exceed the  $\pm 1.96SD$  criterion to be considered a reliable change; for the purpose of reporting, positive indices indicate improvement and negative indices indicate deterioration. Five participants had a reliable clinical improvement in insomnia severity, one improved on sleep quality, four improved on psychological distress, four had improved anxiety symptoms, three had improved on depression symptoms, and two showed reliable clinical improvement in experiential avoidance. One participant had a clinically reliably deterioration on the depression scale, but it is important to note that this participant did not attend all intervention sessions.

Table 3 also shows the reliable change indices comparing phase D (study follow-up) with phase A (the baseline). Of the six people that completed the follow-up questionnaires, four showed clinical improvement in insomnia severity, two improved on their sleep quality, four improved on psychological distress, three showed improvements in anxiety and depression, and two showed improvement in experiential avoidance. One participant had a clinically significant deterioration on their psychological distress score from baseline to follow-up.

#### *Sleep diaries*

The Tau-U phase comparisons for each participant on SE, TST, WASO and SoL measured from the sleep diaries are presented in Table 4. Overall, few significant changes were found based on the sleep diary results. On SE one person worsened from phase A to B; one person improved on TST from phase C to D. Significant improvements were seen in WASO for one person from phase B to

**Table 2.** Means, standard deviations and Wilcoxon signed rank tests for actigraphy data for each participant

ID	Sleep onset latency (min)			Total sleep time (h)			Sleep efficiency (%)			Wake after sleep onset (min)		
	Pre Mean (SD)	Post Mean (SD)	Comparison	Pre Mean (SD)	Post Mean (SD)	Comparison	Pre Mean (SD)	Post Mean (SD)	Comparison	Pre Mean (SD)	Post Mean (SD)	Comparison
101 (M19)	127.7 (54.1)	154.5 (89.6)	Z=-1.15, p=.249, r=-.332	7.1 (1.5)	6.1 (2.4)	Z=-.94, p=.345, r=-.271	66.4 (6.7)	61.3 (15.3)	Z=-.734, p=.463, r=-.212	79.9 (18.0)	49.6 (29.6)	Z=-1.37, p=.172, r=-.395
102 (M54)	9.2 (11.5)	7.6 (9.3)	Z=-.10, p=.916, r=-.028	7.6 (1.7)	6.5 (.7)	Z=-1.57, p=.116, r=-.435	89.1 (3.0)	91.7 (3.2)	Z=-1.15, p=.249, r=-.319	29.2 (19.5)	19.5 (6.9)	Z=-.74, p=.463, r=-.205
103 (M27)	84.0 (101.4)	—	—	6.3 (1.8)	—	—	75.4 (11.8)	—	—	49.7 (22.7)	—	—
104 (M32)	36.5 (23.0)	34.3 (31.8)	Z=-.943, p=.345, r=-.272	7.9 (1.5)	7.9 (1.5)	Z=0.0, p=1.0, r=0.0	83.4 (6.7)	82.4 (2.3)	Z=-.105, p=.917, r=-.030	41.0 (21.1)	91.5 (63.8)	Z=-1.57, p=.116, r=.453
201 (M68)	9.1 (9.1)	8.5 (12.3)	Z=-.34, p=.735, r=-.091	6.5 (.7)	6.7 (.5)	Z=-.169, p=.866, r=-.045	83.7 (3.4)	87.1 (4.2)	Z=-1.69, p=.091, r=-.452	47.7 (6.5)	38.3 (7.4)	Z=-1.69, p=.091, r=.452
202 (M37)	8.2 (8.2)	27.4 (28.3)	Z=-1.78, p=.075, r=-.514	6.6 (.7)	5.6 (.8)	Z=-1.68, p=.093, r=-.485	81.4 (6.1)	73.4 (5.4)	Z=-1.78, p=.075, r=-.514	61.3 (12.6)	59.7 (12.7)	Z=-.10, p=.917, r=-.029
203 (F60)	14.8 (14.0)	27.2 (20.6)	Z=-1.57, p=.116, r=-.453	7.4 (1.9)	6.1 (.7)	Z=-.943, p=.345, r=-.272	77.0 (5.5)	75.5 (6.7)	Z=-.524, p=.600, r=-.151	104.0 (16.0)	94.3 (34.0)	Z=-.52, p=.600, r=-.150
204 (F50)	26.2 (61.7)	23.2 (22.7)	Z=-.67, p=.500, r=-.193	7.2 (1.2)	6.8 (1.2)	Z=-.943, p=.345, r=-.272	86.9 (7.2)	89.2 (5.3)	Z=-.314, p=.753, r=-.091	36.0 (19.3)	27.8 (12.8)	Z=-1.15, p=.249, r=-.332

**Table 3.** Reliable change indices for study measures – difference scores phase a–phase c and phase a–phase d

Participant	Insomnia severity		Sleep quality		CORE-10		HADS-Anxiety		HADS-Depression		BEAQ	
	A-C	A-D	A-C	A-D	A-C	A-D	A-C	A-D	A-C	A-D	A-C	A-D
101 (M19)	0.95	2.87*	0.95	0.95	1.44	-1.92*‡	1.25	0.63	2.04*	-0.68	1.93	3.14*
102 (M54)	2.87*	4.78*	0.95	0	-0.48	-0.96	0	0	0	0	0.72	3.62*
103 (M27)	3.83*	n/a	-0.048	n/a	0.48	n/a	0	n/a	-4.08*‡	n/a	0	n/a
104 (M32)	2.87*	2.87*	0.48	1.90	1.44	2.41*	1.88	1.25	0.68	1.36	0.48	0.97
201 (M68)	0.48	1.43	1.90	2.38*	6.73*	5.29*	2.51*	3.76*	1.36	5.43*	0.24	-0.24
202 (M37)	1.91	0.96	0.95	0.48	3.37*	4.33*	3.13*	2.51*	0.68	2.71*	3.62*	1.93
203 (F60)	2.87*	n/a	2.38*	n/a	7.21*	n/a	3.13*	n/a	2.72*	n/a	1.93	n/a
204 (F50)	4.30*	5.26*	n/a	2.85*	9.62*	7.21*	5.01*	3.13*	8.15*	6.79*	3.14*	2.17

\* $p < .05$ . All improvements, except for values marked with ‡ (reliable deterioration).

C and for four participants from phase C to D. Finally, one person deteriorated on SoL from phase A to B. Supplementary Figures 2 to 6 show the sleep diary data across the entire trial for each participant on SE, TST, WASO and SoL.

#### Client satisfaction

The median for all five items was 4. For items assessing understanding of information and strategies, approval of the procedures, and recommendation of the intervention the range of responses was 4 to 5. The range for the other two items assessing helpfulness in improving sleep and ability to use strategies in the future was 3 to 5. Participants had mostly positive attitudes and reported that the mindfulness exercises and learning not to worry or battle with sleep were the most useful aspects of the intervention: (1) *Giving us permission not to battle with sleep. Providing strategies to help*; (2) *The scarf demonstration in the first session [Tug of War metaphor]*; (3) *The guided meditation, dried grape mindfulness and body scan exercises. Learning about mindfulness techniques and how they can help to improve my sleep.* Suggestions for improvement included expanding on information around anxiety-specific challenges for autistic adults, providing a workbook for participants, and improvement of slide and hand-out quality. For example, (1) *Maybe expand on what people on the spectrum face with anxiety and how this effects sleep*; (2) *Making them more interactive and structured. Giving participants a workbook with hints, tips and summary notes.*

#### Discussion

The primary aim of this study was to examine the impact of a novel ACT/BT group insomnia intervention (ACT-i) on insomnia in autistic adults; our secondary aim was to examine any concurrent impact on their mental health symptoms, particularly anxiety. Consistent with our hypothesis, at a group level there were significant improvements in self-reported insomnia and anxiety symptoms across the three time points. Importantly reductions in insomnia severity were seen at the individual level for all participants at post-intervention with gains being maintained or improving for five of the six participants completing follow-up. Clinically reliable change in insomnia occurred for majority of participants following intervention. Furthermore, client satisfaction indicated that ACT-i was an acceptable intervention to our participants. The improvements in ISI insomnia symptoms are consistent with those reported in a meta-analysis of CBT-I in non-autistic populations (Geiger-Brown *et al.*, 2015). Change in self-reported insomnia symptoms following the intervention are also consistent with studies in non-autistic adults which showed improvements in sleep in response to ACT interventions (Zakie and Khazaie, 2019; Zetterqvist *et al.*, 2018). In conjunction with reports from the

**Table 4.** Tau-U analyses across phases for each participant measured from sleep diaries

Participant	Sleep efficiency			Total sleep time			Wake after sleep onset (WASO)			Sleep onset latency		
	A-B	B-C	C-D	A-B	B-C	C-D	A-B	B-C	C-D	A-B	B-C	C-D
101 (M19)	0.23	-0.13	-0.08	0.21	-0.16	0.10	0.18	-.01	0.34	-0.24*	0.03	0.35
102 (M54)	0.24*	0.18	n/a	-0.09	0.06	n/a	0.08	0.00	N/A	0.01	0.28*	n/a
103 (M27)	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
104 (M32)	0.06	-0.06	0.45*	0.06	-0.09	0.20	0.20	0.07	-0.67**	-0.18	0.18	-0.11
201 (M68)	-0.40**†	0.26*	0.25	0.11	0.00	0.59**	0.00	0.01	-0.60**	-0.13	-0.05	0.00
202 (M37)	0.13	-0.08	-0.10	-0.06	-0.06	-0.04	-0.09	0.34**	-0.59**	0.09	0.15	-0.29
203 (F60)	-0.03	0.07	n/a	-0.06	-0.02	n/a	-0.05	-0.26*	n/a	0.35**†	0.12	n/a
204 (F50)	0.14	0.10	0.14	-0.04	-0.08	0.07	-0.13	0.01	-0.53**	0.01	-0.07	-0.27

\* $p < .10$  (trend), \*\* $p < .05$ . All improvements, except for values marked with † (deterioration). Tau-U<sub>adj</sub> scores reported.

non-autistic literature that ACT is effective in improving sleep quality in CBT-I non-responders (Dalrymple *et al.*, 2010; Hertenstein *et al.*, 2014), our intervention results provide some support the use of ACT-based intervention for insomnia in autistic adults.

The significant reduction in anxiety symptoms and pre-sleep arousal in response to ACT-i supports the identified relationship between insomnia, anxiety or poor mental health, and pre-sleep arousal in autistic populations (Baker *et al.*, 2019b; Gilbert Gustemp *et al.*, 2021; Richdale *et al.*, 2014; Tani *et al.*, 2003) and also provides support for a hyperarousal theory of insomnia in autistic adults (Baker *et al.*, 2019b). This hypothesis suggests that both cognitive (stress, worry and rumination about sleep) and somatic (hormone/neurotransmitter changes) arousal contribute to sleep behaviour changes resulting in a perpetuating cycle of sleep difficulties which leads to subsequent poor mental health (Riemann *et al.*, 2010). Overall, our intervention data and previous literature provide further support for insomnia as a transdiagnostic process associated with the onset and maintenance of mental health difficulties (Dolsen *et al.*, 2014). Therefore, treating insomnia in autistic adults using ACT-i may provide long-term benefits for both insomnia and co-occurring mental health difficulties.

There was no significant reduction in depressive symptoms, fatigue or general psychological distress. Overall, our participants showed a small, non-significant reduction in depression scores across the trial. Recent reviews suggest that treating insomnia in those with both depression and insomnia may lead to better long-term clinical outcomes (Bei *et al.*, 2018; Gee *et al.*, 2019). Only four of our participants had clinical levels of depression at baseline and three showed clinical improvement following intervention; one declined, but the latter individual did not attend all intervention sessions. Thus, further investigation of the impact of ACT-i on depression may be an important future avenue to explore in larger samples of individuals with both poor sleep and depression as high rates of depression are reported in autistic adults (Hollocks *et al.*, 2019). Furthermore, fatigue is a symptom of both insomnia and depression which may partially account for the lack of change in this variable (American Psychiatric Association, 2013). While fatigue may be increased in autistic adults (Baker and Richdale, 2015) little is known about its associations with mental health and sleep in autistic adults.

We also found a reduction in self-reported experiential avoidance over the course of the trial. Due to their high vulnerability to stressors, autistic individuals tend to engage in experiential avoidance (Pahnke *et al.*, 2014; Pahnke *et al.*, 2019), where an individual is unwilling to remain in contact with distressing emotions, thoughts and sensations in ways that cause harm in the long run. The aim of ACT-based intervention is to reduce experiential avoidance by increasing openness to experience and values-based behaviours (Hayes *et al.*, 2012). Therefore, the observed reduction in experiential avoidance suggests that ACT-i functions via the same mechanisms in autistic adults as non-autistic adults, providing further evidence for the usefulness of ACT-based intervention for autistic adults.

Despite the significant changes found in the self-report questionnaires, there were no significant changes in actigraphy data. Examining individual cases, sleep patterns across participants were highly variable; high variability in SoL and SE on actigraphy has been reported previously in autistic adults (Baker and Richdale, 2015). Therefore, given the small sample size it may not be surprising that significant group differences were not observed. The pilot nature of this study limited our ability to collect continuous actigraphy data and we were not able to collect actigraphy data at 2-month follow-up. It is possible that improvements in the objective actigraphy measurements may occur by follow-up as participants were able to consolidate the skills learnt in the intervention. Similar to the actigraphy data, the analysis of the sleep diary data revealed highly variable sleep patterns across the sample. However, the single case analyses revealed that for four of the seven participants who completed sleep diaries there were significant improvements in TST and/or WASO after the group intervention.

This pilot study had some limitations. First, our sample size was small, with only eight participants, and most questionnaire changes having a moderate to large effect size. The

restricted timing of our funding did not allow us to recruit a larger sample or offer additional time slots to accommodate the three additional, interested adults. Pahnke *et al.* (2019) reported that ACT was efficacious and acceptable in treating mental health concerns in a similar sample size of 10 autistic adults, supporting that ACT is promising intervention approach for autistic adults. Nevertheless, our findings require replication with much larger samples.

A second potential issue is that our ACT-i intervention, like CBT-I, incorporated a strictly behavioural component to address some sleep issues and it is possible that these alone are sufficient to treat presenting sleep concerns. Sleep literature suggests that to address sleep concerns it is necessary to maintain regular bed and wake times and to have comfortable physical conditions that promote sleep. However, insomnia is frequently associated with cognitive (Kalmbach *et al.*, 2020; Vargas *et al.*, 2020) and somatic hyperarousal (Vargas *et al.*, 2020), including in autism (Baker *et al.*, 2019b; Richdale *et al.*, 2014). Thus, ACT, which is itself based on behavioural principles, can address the psychological components of poor sleep by improving psychological flexibility, and as suggested by feedback received (e.g. *Giving us permission not to battle with sleep* – see Results) these components were perceived as important. However, it would be useful to examine which components of our intervention are most efficacious, or whether all components are necessary for optimal treatment outcomes. Finally, while the ISI is a widely used outcome measure for examining the efficacy of insomnia interventions, we found few significant changes in the self-reported sleep diaries and there were none on our objective measure of sleep, actigraphy. This may be due to the inherent variability in sleep parameters previously noted in autism (Baker and Richdale, 2015) combined with a small sample size. Furthermore, sleep diaries can lead to over-estimation of total sleep and sleep efficiency (Deitch *et al.*, 2021); it nevertheless introduces a note of caution to our findings.

In conclusion, this pilot study with eight autistic adults indicates that ACT-i is both an efficacious and acceptable intervention for reducing self-reported insomnia and anxiety symptoms in autistic adults. The study also indicates that using a multi-modal approach to measurement (i.e. sleep diaries, actigraphy, self-report questionnaires) is feasible in sleep intervention trials with autistic adults. Like the recent pilot study by Pahnke *et al.* (2019), our results support the efficacy of ACT-based interventions for autistic adults. Nevertheless, future research is required to assess the efficacy of ACT-i in addressing insomnia using a large sample, randomised control trial which compares ACT-i with a non-intervention group. In addition, a longer follow-up period could be beneficial to determining whether ACT-i has a longer-term impact on insomnia, and mental health, fatigue or psychological distress.

**Supplementary material.** To view supplementary material for this article, please visit: <https://doi.org/10.1017/S1352465822000571>

**Data availability statement.** The data that support the findings of this study are available on request from the corresponding author (E.M.J.M.). The data are not publicly available due to the small nature of the study and containing information that could compromise the privacy of research participants.

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