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A comparative study of circadian rhythm functioning and sleep in people with Asperger syndrome

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ABSTRACT The circadian rhythm functioning and sleep patterns of 10 adults with Asperger syndrome were investigated using actigraphy. When compared with data from neurotypical adults, both statistical and clinically significant differences were found between the two groups, with the adults with Asperger syndrome showing marked abnormalities in both the quantity and the quality of sleep recorded. Examination of the actigraphic data indicated low sleep efficiency and high fragmentation as being characteristic of the sleep of participants with Asperger syndrome. These individuals also showed lower-amplitude circadian rhythms that were less strongly linked to environmental synchronizers, but no evidence of significant desynchronization of circadian rhythm. Possible mechanisms for these abnormalities and implications for clinical practice are discussed.

KEYWORDS *Asperger syndrome; circadian rhythms; sleep*

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Introduction

The sleep patterns and disturbances of sleep in children with autistic spectrum disorders (ASDs) have been investigated in a number of studies (Hering et al., 1999; Honomichl et al., 2002; Hoshino et al., 1984; Inuama, 1984; Johnson, 1996; Patzold et al., 1998; Richdale and Prior, 1995; Schreck and Mulick, 2000; Taira et al., 1998a; 1998b; Williams et al., 2004). The majority of these studies have utilized data obtained from record-keeping by parents and carers. However, there is a growing body of research using more objective measures of sleep disturbance in both

children and adults with autistic spectrum disorders. Hering et al. (1999) investigated the sleep of 22 children with autistic spectrum disorders, using actigraphy (actometry) to obtain objective data on sleep patterns independent of parental reports. This study indicated that parents tended to *overestimate* the frequency and number of sleep problems experienced by their children. More recently, Øyane and Bjorvatn (2005) investigated sleep disturbance in 15 young adults aged between 15 and 25 years with autistic spectrum disorders (including six with Asperger syndrome), also using actigraphy, and found evidence of *underestimation* of sleep disturbance by parents and carers.

The sleep functioning of adults with ASDs, including Asperger syndrome, has received less attention to date. Although there are anecdotal accounts of sleep disturbance in adolescents with Asperger syndrome, systematic investigations are currently limited to three studies. Tani et al. (2003) carried out a study using self-report measures to assess the quality of sleep in 20 adults with Asperger syndrome and reported a significant incidence of sleep disturbance, which primarily took the form of delayed sleep latency (i.e. problems in falling asleep) and impaired sleep efficiency (i.e. increased night-time waking). Objective studies of sleep functioning in adults with Asperger syndrome are limited to the studies by Godbout et al. (1998; 2000) and Øyane and Bjorvatn (2005). Godbout and colleagues (2000) investigated sleep in eight people with Asperger syndrome over two nights using polysomnography (PSG: multiple physiological measures taken during sleep, including electroencephalogram, electro-oculogram and electromyogram data) in a sleep laboratory setting. Comparison with a matched neurotypical control group revealed less overall sleep in the participants with Asperger syndrome, together with various abnormalities of REM sleep and a low incidence of sleep spindles. Godbout et al. note that the former has been associated with daytime cognitive difficulties and the latter with problems in selective attention. Although other aspects of sleep, including sleep latency and sleep efficiency, were found to be normal, Godbout et al. propose that *sleep control* deficits should be considered as part of the clinical presentation of Asperger syndrome. The studies by Tani et al. and Godbout et al. are important, as the sleep disturbances they identify have been associated with impairments in daytime cognitive functioning (Dinges and Kribbs, 1991), involving both explicit and implicit learning (Heuer et al., 1998). As noted above, Øyane and Bjorvatn (2005) included young adults with Asperger syndrome in their actigraphic studies. However, given the disparate living arrangements of the participants in their studies – some lived in institutional settings, some with families and some independently – it is difficult to differentiate between sleep disturbance related to intrinsic factors

specific to Asperger syndrome and that related to extrinsic factors in connection with current living arrangements (see Hare et al., in press). Moreover, only one study to date, that of Godbout and colleagues, has involved a direct comparison with typically developing participants.

Circadian rhythms (i.e. human behavioural, physiological or biochemical cycles with a period of approximately 24 hours) have an impact on normal sleep propensity and on the development and maintenance of forms of insomnia (see Lack and Bootzin, 2003). Disturbances in circadian functioning, as opposed to the sleep disorders they influence, have been discussed in connection with sleep disorders in children with ASD (Patzold et al., 1998; Stores and Wiggs, 1998), but there remains a paucity of empirical data. To date, there have been no studies of circadian rhythm functioning in adults with Asperger syndrome. However, the use of actigraphy enables the measurement of both sleep characteristics and the circadian sleep–wake cycle (Ancoli-Israel et al., 2003) to be objectively investigated in a non-invasive manner, via examination of activity levels over 24 hour periods. Actigraphy employs a miniature acceleration sensor (actigraph) to detect physical movement over an extended period (up to 3 weeks). The actigraph is usually worn on the non-dominant wrist, being of similar size and shape to a normal wristwatch, and places few if any restrictions on the wearer. Although REM and non-REM sleep cannot be readily distinguished using actigraphy data, such data correlate very highly with data from polysomnography and enable periods of sleep to be distinguished from periods of wakefulness (Sadeh et al., 1995). Therefore, the use of actigraphy both obviates the need for self- or third-party recording of sleep patterns and also in many instances can substitute for laboratory-based PSG, due to the high concordance between actigraphic and PSG analyses (Jean-Louis et al., 2001). To address the paucity of objective data on both sleep disturbances and the circadian sleep–wake cycles in adults with Asperger syndrome and methodological deficits in previous research, a naturalistic study of sleep patterns and circadian activity rhythm functioning in adults with Asperger syndrome was conducted as part of a larger study on circadian functioning in adults with autistic spectrum disorders.

Method

Participants

Adults (i.e. aged over 18 years) with verifiable diagnoses of Asperger syndrome according to ICD-10 or DSM-IV criteria were recruited via a specialist independent service for people with autistic spectrum disorders in the north-west of England. In total 10 people with Asperger syndrome took part in the study. As a formal diagnosis of Asperger syndrome was a

prerequisite to accessing this service, all diagnoses were verifiable and all potential participants approached agreed to take part in the study. The people with Asperger syndrome in the present study were not selected on the basis of having an extant sleep disorder.

In order to provide appropriate comparison data, an opportunity control group was created from participants in a parallel research study (Jones et al., 2005). This study utilized an identical methodology to collect data on sleep and circadian rhythm functioning in adults with no learning disabilities, developmental disabilities or mental health disorders. In total, data from 18 'neurotypical' adults were available for the present study. Although it was not possible to match the two groups on the basis of age, this was controlled for in the course of the data analysis. All participants in the study had English as their first language, as it was not possible to arrange for the necessary translation and validation of the measures used in the study.

Procedure

Demographic data were collected from the participants with Asperger syndrome on their age, gender and any current medication. Verbal comprehension ability was assessed using the British Picture Vocabulary Scale short form (BPVS) and the degree of autistic symptomatology was examined by the Autism Screening Questionnaire (ASQ). Each participant in the study wore a Cambridge Neurotechnology AW4 Actiwatch for 7 days while going about their normal activities. A 15 second sampling interval was utilized. Over the same period a basic sleep diary was compiled, noting times of going to bed and getting up. Data from the Actiwatch were then downloaded and analysed using Actiwatch sleep analysis software v1.19 (Cambridge Neurotechnology 2001) to provide the following data:

- *L5 and M10 onset*: indicate the average time of the start of the least active 5 hour (L5) and the most active 10 hour (M10) periods across a circadian cycle and indicate the degree to which a person's circadian cycle is in phase with a normal 24 hour cycle.
- *Relative amplitude*: indicates quantity of activity and is derived from the normalized difference between the most active 10 hour period and the least active 5 hour period in an average 24 hour pattern, with a range of 0–1, higher values indicating greater difference between the most and least active phases.
- *Intradaily variability*: indicates fragmentation of rhythm, by assessing the frequency and extent of transitions between rest and activity. This is derived as a ratio of the mean squares of the differences between all successive hours and the mean squares of the differences with the grand mean.

- *Interdaily stability*: indicates invariability of the 24 hour rhythm between days and is the 24 hour value from a chi-square periodogram, normalized for number of data points. This indicates the strength of the linkage of the circadian rhythm to *zeitgebers* (environmental synchronizers) such as amount of daylight, alarm clocks, sound levels and social interactions.
- *Periodicity*: the length of time or cycle length after which a definite cycle recurs.
- *Acrophase*: time of the peak of the 'best fit' circadian rhythm.

In addition to data on circadian functioning, data on important parameters of participants' sleep were also collated in order to investigate whether these had a significant association with daytime activity independently of circadian rhythm. The sleep parameters examined in the present study were: *sleep latency*, the time between going to bed and onset of sleep; *sleep duration*, the time actually asleep; *sleep efficiency*, the actual time asleep as a percentage of the time in bed; and *sleep fragmentation*, the percentage of immobile phases of 1 minute duration as a proportion of the total number of immobile phases. In addition to the mean scores *per se*, the mean standard deviation for each of these parameters was used as a measure of variability.

Results

The participants with Asperger syndrome had a mean age of 30.8 years (SD 6.91), which was significantly younger than that of the neurotypical group who had a mean age of 46.89 years (SD 14.82) ($t = -3.233$, $p = 0.003$). The Asperger syndrome mean BPVS score (189.30, SD 4.16) indicated that the verbal comprehension abilities of this group were age-equivalent for 15 years 9 months and could be taken as indicative of normal verbal abilities. A circadian rhythm analysis (van Someren et al., 1999) was performed for the available actigraph data from all of the participants. This analysis yielded mean values for each participant for measures of interdaily stability, intradaily variability, periodicity and relative amplitude over a 7 day period. Table 1 shows the mean scores and standard deviations of the circadian rhythm variables for both groups of participants, together with the median value for both groups combined. All the data were found to be normally distributed.

There were no statistically significant differences in the mean scores for periodicity or intradaily variability. However, the mean periodicity for the adults with Asperger syndrome indicated a slightly phased delayed circadian rhythm of 24½ hours, compared to the 24 hours indicated for the neurotypical group. There was a significant difference in the mean relative amplitude scores, with that of the participants with Asperger syndrome

Table 1 **Mean scores for circadian rhythm variables derived from actigraphic recording**

	Median value (*N = 29*)	*Adults with Asperger syndrome* (*N = 10*)	*Neurotypical adults* (*N = 19*)	*t* (*2-tailed*)	*p*
Interdaily stability	0.6130	0.5136 (0.120)	0.629 (0.0908)	–2.970	0.006
Intradaily variability	0.6510	0.754 (0.267)	0.671 (0.1233)	1.149	n.s.
Relative amplitude	0.9200	0.816 (0.099)	0.929 (0.0366)	–4.490	<0.000
Periodicity	1445.00	1447.00 (18.135)	1447.63 (16.614)	–0.094	n.s.

being significantly lower, indicating a lower level of overall activity with less differentiation between the most and least active phases over the circadian period. Similarly, there was a statistically significant difference between the two groups with regard to interdaily stability, with that for the participants with Asperger syndrome being lower, indicating that their circadian rhythms were less strongly linked to external *zeitgebers*.

The data on the L5 and M10 parameters for the participants with Asperger syndrome indicated wide variability in the onset of both phases. The modal L5 onset time was 1 a.m., with a range from 10 p.m. to 4 a.m., and the modal M10 onset time was 11 a.m., with a range from 6 a.m. to 2 p.m. Taken together, these data indicate one instance of significant phase advancement in the sleep–wake cycle (L5 = 10 p.m., M10 = 6 p.m.) and one instance of phase delay (L5 = 4 a.m., M10 = 2 p.m.), both of which can be considered pathological. No such presentations were found in the neurotypical group. Further examination of the circadian rhythm indicated that the mean acrophase did not differ between the two groups (Asperger syndrome mean 15.16 (SD 1.83), neurotypical mean 15.05 (SD 0.32)). The mean values of the sleep parameters (sleep onset (sleep latency), efficiency, duration and activity during sleep (fragmentation)) were calculated for the equivalent 7 day period and are shown in Table 2.

In all aspects bar mean actual sleep time, the participants with Asperger syndrome differed from the neurotypical participants with regard to the quality of their sleep, with longer sleep latency, lower sleep efficiency and more fragmented sleep. In addition, the range of variability in sleep quality, as indicated by the mean standard deviations, was greater for the participants with Asperger syndrome. In terms of comparing the results to normative data on adult sleep patterns, the mean latency period for the participants with Asperger syndrome was slightly higher than would be expected, while mean actual sleep and mean sleep fragmentation were lower than would be expected, i.e. mean efficiency < 85 percent (Lacks and Morin, 1992). Given the significant difference in age between the two

Table 2 **Mean and standard deviation scores for sleep parameters: time of sleep onset (sleep latency), efficiency, duration and activity during sleep (fragmentation) derived from actigraphic recording**

	Adults with Asperger syndrome (N = 10)	*Neurotypical adults (N = 19)*	*t (2-tailed)*	*p*
Mean latency (min)	32.55 (12.75)	17.07 (14.11)	2.896	0.007
Mean SD latency	34.61 (17.59)	21.18 (16.98)	1.998	0.056
Mean actual sleep (min)	415.51 (71.34)	446.78 (30.08)	–1.669	n.s.
Mean SD actual sleep	90.29 (27.67)	58.92 (33.30)	2.546	0.017
Mean sleep efficiency	74.27 (4.99)	86.43 (4.20)	–6.946	<0.001
Mean SD sleep efficiency	10.75 (4.21)	4.89 (2.82)	4.477	<0.001
Mean fragmentation	64.33 (21.28)	43.76 (14.87)	3.047	0.005
Mean SD fragmentation	20.79 (7.89)	11.51 (6.21)	3.481	0.002

groups, a series of linear regression analyses were performed using age and group as independent variables with the sleep and circadian rhythm variables as the dependent variables. Age was not found to be a significant predictor except for SD fragmentation (age $p = 0.017$, group $p = <0.001$), suggesting that the difference in mean age between the two groups was not a confounding factor with respect to either sleep or circadian rhythms.

Discussion

The results of the present study appear to indicate that not only is the sleep of people with Asperger syndrome significantly different from that of people with typical development, but also that these differences constitute a degree of abnormality. This is shown by the increased onset latency and fragmentation of sleep and the reduced sleep efficiency. In addition, the sleep of the people with Asperger syndrome exhibited greater variability in both quantity and quality (i.e. efficiency and fragmentation). As such, these findings, although derived by different means, are in accordance with the previous findings of Tani et al. (2003) and also the findings of Øyane and Bjorvatn (2005), but apparently conflict with the PSG studies conducted by Godbout et al. (2000). This discrepancy may be due to the differences in the data obtained by actigraphy and PSG. However, the findings of increased sleep fragmentation lend support to Godbout et al.'s contention that sleep control deficits are inherent in Asperger syndrome.

In addition to examining sleep, the present study examined circadian functioning, and again some statistically significant differences were observed compared to neurotypical adults. The finding of such abnormalities in

adults with Asperger syndrome is in line with previous research into circadian function in children with autistic spectrum disorders (Patzold et al., 1998; Stores and Wiggs, 1998). In the present study, the participants with Asperger syndrome showed a lower relative amplitude over the 7 day period of data gathering, which indicated that there was less difference in activity levels between their most and least active periods than is usually observed in neurotypical adults. In addition, the participants with Asperger syndrome exhibited greater instability in their circadian rhythms over this same period, indicating that these rhythms were not as strongly linked to *zeitgebers* as would be expected. At this stage in the research, it is not possible to say which *zeitgebers* may be involved, but both social interaction and amount of daylight are plausible factors for the regulation of circadian rhythm function in Asperger syndrome. The latter plays an important role, via intermediate enzyme production, in the production of the hormone melatonin, which determines the quality of sleep (Arendt, 1996). Melatonin production increases in early evening and reaches a peak between 2 a.m. and 4 a.m. (i.e. corresponding to the least active 5 hour period in the circadian cycle). However, there was no evidence from the present study to indicate that the circadian rhythms of the participants with Asperger syndrome were significantly desynchronized *per se*, as their mean acrophases did not differ from those of the neurotypical participants which were in themselves normal.

This may account for the differences between the findings of the present study and those of Godbout et al.'s (2000) study, both of which used objective measures to measure sleep quantity and quality. The present study used actigraphy to measure sleep quantity and quality in a naturalistic context, whereas Godbout and colleagues measured sleep in the artificial setting of a sleep laboratory. It can be argued that the results of the present study are a more ecologically valid description of sleep quantity and quality in people with Asperger syndrome, as participants were exposed to the usual *zeitgebers* over the 7 day period in which the actigraph data were collected.

Taken together, the findings of reduced relative amplitude and reduced stability of circadian rhythms together with abnormal onset and sleep maintenance difficulties may be indicative of impaired melatonin production, as has been postulated with regard to autistic spectrum disorders (e.g. Nir, 1995). This is not to rule out an important role for other important *zeitgebers*, such as social interaction and social rhythms *per se*. Further research combining actigraphic data with the measurement of social rhythms, as has been carried out for other populations (e.g. Jones et al., 2005), would be useful in this regard.

A limitation of the present study is that the participants with Asperger syndrome were recruited from a relatively small geographical area via a

specialist service for people with ASD and it is not possible to make any statements regarding the representativeness of this group with respect to people with Asperger syndrome as a whole. However, the current findings appear to be robust in terms of both the methods of data collection and the high ecological validity of the data. Furthermore, the current study has obviated the limitations of previous research by having a diagnostically homogeneous sample and by the inclusion of a typically developed comparison group. Although age difference between the two groups in the study was controlled for in a *post hoc* manner and no significant effects were observed, future research would benefit from more closely matched participants.

The findings of the present study have a number of important clinical implications. Sleep disturbance *per se* has a deleterious impact on individual social and psychological functioning, the latter including impaired specific emotional and cognitive functions (Heuer et al., 1998; Wagner et al., 2001). Being out of phase with typical social rhythms can increase a person's sense of social isolation and difference (see Abell and Hare, 2005), which can lead to the development and maintenance of delusional beliefs (Johnson, 1988). Abnormal circadian functioning has also been implicated in the development of bipolar and other psychiatric disorders (Ashman et al., 1999; Jones, 2001). In practice, it will be more difficult for people with disturbed circadian rhythms to readily engage in culturally normative and valued activities such as education and work. Therefore, it is strongly recommended that when undertaking assessments of clinical and social need for people with Asperger syndrome, both the sleep–wake cycle and sleep quality be specifically assessed.

In conclusion, the present study has demonstrated the utility of actigraphy in the assessment of sleep patterns and circadian rhythm in people with Asperger syndrome in a way that is both acceptable to participants and yields high-quality ecologically valid data. The findings of the present study add to the growing literature indicating sleep disturbance in people with Asperger syndrome and also further specify the nature of this disturbance.

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