

## Severity of insomnia, disordered eating symptoms, and depression in female university students

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**Key words**

depressive mood, direct relationship, eating habits, indirect relationship, insomnia.

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**Abstract**

**Background:** Insomnia is one of the most common sleep disorders, and it frequently co-occurs with several other psychiatric conditions. The relationship between insomnia and eating disorders is supported by clinical evidence indicating that patients with eating disorders experience poor sleep even if they rarely complain of it. Furthermore, indirect evidence comes from studies indicating that poor sleep predicts obesity and several studies also evidence that restrictive-type eating disorders are associated to objective reduction of sleep quality.

**Methods:** One thousand nineteen female university students volunteered for participating to the study. Valid and reliable questionnaires were used and the mediating role of depressive mood assessed.

**Results:** Evidence was found that increased severity of insomnia is associated with higher severity of disordered eating. Both insomnia and disordered eating symptoms were related to depression. The mediation analysis evidenced that both the direct path linking insomnia symptoms and eating disorder symptoms are significant and also the indirect paths related to the mediation of depression.

**Conclusions:** These findings support the existence of both a direct and an indirect relationship between insomnia symptoms and eating disorder symptoms.

**Key Points**

- 1 Patients with eating disorders frequently experience poor sleep even if they rarely complain of it; only few empirical data support the relationship between these disorders, so it could be spurious, i.e., because of the relationship with depression of both insomnia and eating disorders.
- 2 The present study assessed this relationship in a wide female non-clinical sample using valid instruments and evidencing that it stands still after removing the effect of depression.
- 3 The existence of both a direct and an indirect relationship between insomnia and eating disorders suggest that the use of CBT for insomnia, known to be effective for both primary and secondary insomnia, may be also useful in reducing eating disorder symptoms.

**Introduction**

Insomnia is one of the most common sleep disorders affecting 9–15% of the general population (Ancoli-Israel & Roth, 1999; Ohayon & Reynolds, 2009). It is characterised by poor sleep, difficulty in initiating, or maintaining sleep, non-restorative sleep, with daytime consequences persisting for at least 4 weeks (American

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Psychiatric Association, 2000; American Sleep Disorders Association, 2005). Insomnia commonly co-occurs with most psychiatric disorders, especially major depression (Riemann & Voderholzer, 2003; Tsuno, Berset, & Ritchie, 2005). In addition, patients with eating disorders frequently experience interrupted sleep and early morning awaking, although they rarely spontaneously complain of it (Crisp, 1967; Crisp, Stonehill, & Fenton, 1971). However, epidemiological evidence of the co-occurrence of anorexia nervosa (AN) or bulimia nervosa (BN), and insomnia is lacking.

Indirect evidence of the relationship between insomnia and eating disorders emerged from studies indicating that poor sleep (or sleep of short duration) predicts obesity in children (Marshall, Glogzerb, & Grunstein, 2008; Patel, 2009; Van Cauter & Knutson, 2008), in adults (Lauderdale *et al.*, 2007; Patel *et al.*, 2006) and in the elderly (Patel *et al.*, 2008; Van der Berg *et al.*, 2008). A second type of indirect evidence was also derived from laboratory studies demonstrating the sleep-disruptive effects of hunger. Hunger induced by food deprivation or administration of orexin produces increased nocturnal wakefulness and reduced slow-wave sleep (Ohno & Sakurai, 2008). This effect was consistent with the results of polysomnographic studies demonstrating reduced sleep efficiency, increased wake time, and/or increased stage 1 non rapid eye movement sleep in restrictive type AN compared with a control group; these results are summarised by Lauer and Krieg (2004).

Recently, Kim *et al.* (2010) reported that sleep disturbances were highly prevalent (50.3%) among 400 female patients diagnosed with AN, BN, or binge eating disorder (BED). In addition, the general symptomatological condition was more severe in patients who also had sleep disturbances compared with those who did not complain of them. This study provided evidence of an association between eating disorders and sleep disturbances in a broad clinical sample. However, the association could not be interpreted unambiguously because the more pronounced severity of eating disorder in patients presenting co-morbid sleep disorders could also be viewed as evidence of a more severe clinical condition in general, namely a condition characterised by the concomitant presence of different syndromes, including others not clearly indicated or even excluded in the study.

Depression is among the syndromes known to be co-morbid with both eating disorders (Slane, Burt, & Klump, 2010) and sleep disorders, especially insomnia (Baglioni *et al.*, 2011). In the study by Kim *et al.* (2010), the concomitant presence of a depressive syndrome in patients with eating disorders was excluded on the basis of the Lauer and Krieg (2004) review reporting data derived from studies demonstrating that the sensitivity of

the central cholinergic system in eating disorder patients, either with or without a co-existing depressive disorder, did not differ from that in healthy subjects. This result, according to the authors (Lauer & Krieg, 2004), supports the existence of a neurobiological link between eating disorders and sleep disorders, and clearly contradicts the assumption that this link is mediated by the involvement of the affective system. However, in the study by Kim *et al.* (2010), the high prevalence of sleep disorders was also associated with increased severity of the global clinical condition. It is thus possible that there is both a direct relationship between sleep disorders and eating disorders, and an indirect relationship, mediated by depression. However, it is still unknown to what extent this relationship is direct and to what extent indirect.

In order to disentangle the direct and indirect link between insomnia and eating disorders, community studies are also needed together with the clinical studies to ascertain if the co-occurrence is present when a full-blown clinical syndrome is absent. To date, only a few community studies (Makino, Hashizume, Yasushi, Tsuboy, & Dennerstein, 2006; Soares *et al.*, 2011) have been conducted, and their results confirmed a positive association between self-reported insomnia symptoms and the severity of eating disorder symptoms. However, most of these studies did not use valid and reliable measures for assessing sleep problems, and none of them evaluated the possible mediating role of affective variables, such as depression. Similar to the clinical studies, the relationship between eating disorders and sleep disorder symptoms in community studies can be either spurious or indirect because of the effect of a third variable, i.e., depression.

The present study sought to replicate the results of community studies using valid and reliable instruments. We used insomnia severity as predictor of the severity of eating disorder symptoms because an increasing amount of evidence suggests that insomnia may be considered a transdiagnostic factor likely to increase the probability of occurrence of many other disorders (Baglioni *et al.*, 2011; Harvey, 2009). Furthermore, because both insomnia and eating disorders are related to depression, we also wanted to assess the mediating role of depression.

## Materials and Methods

### Participants and Procedure

Fifteen undergraduate students administered questionnaires to colleagues, friends, and acquaintances. Potential participants were approached at the Sapienza University of Rome, specifically in the library, around campus, and in lecture halls. Participants were first invited to read a description of the study. Only those who showed interest

were asked to sign a consent form and fill out a series of questionnaires administered in a counterbalanced order. A schedule was also included for sociodemographic data. The whole block of questionnaires required approximately 40 min to be completed.

All individuals who received the consent form filled out the block of questionnaires in the presence of the students charged with data collection. A total of 1020 packets were collected. One block was only partially filled out and thus rejected. The final sample consisted of 1019 female university students ranging from 18 to 48 years and with a mean age of 24.38 (standard deviation (*SD*) 4.12) years. The research was approved by the Ethical Committee of the Department of Psychology of the Sapienza University of Rome.

## Measures

### **The Disordered Eating Questionnaire (Lombardo, Russo, Lucidi, Iani, & Violani, 2004)**

Disordered Eating Questionnaire (DEQ) is a 24-item questionnaire that assesses the presence and intensity of eating disorder symptoms (e.g., limiting or controlling the amount of food or calories consumed, avoiding certain foods, dealing with intrusive thoughts about body weight and shape, etc.) according to Diagnostic and Statistic Manual IV version - Text Revised (DSM-IV-TR) diagnostic criteria. All questions have a time frame pertinent to the preceding 3 months. The validation study (Lombardo et al., 2001) showed a unifactorial structure including 20 items and excluding those items dealing with purging behaviours. The questionnaire also asks for information concerning weight and height. A recent validation study comparing a group of patients with eating disorders and a control sample (Lombardo, Cuzzolaro, Vetrone, Mallia, & Violani, 2011) found that a score of 30 is the best cut-off for maximising sensitivity (83%) and specificity (66%). Sensitivity and specificity scores are higher than those of other well-known instruments, including the Eating Attitude Test - 40 (EAT-40) items (Garner & Garfinkel, 1979) and the Eating Disorder Inventory - 2 (EDI-2) version (Garner, 1991), which assesses disordered eating through many more items than the DEQ (Vetrone, Cuzzolaro, Antonozzi, & Garfinkel, 2006).

### **The Sleep Disorders Questionnaire (Violani, Devoto, Lucidi, Lombardo, & Russo, 2004)**

Sleep Disorders Questionnaire (SDQ) is a brief self-report questionnaire that evaluates the presence of insomnia according to both DSM-IV-TR diagnostic criteria and the quantitative criteria indicated by a consensus report (Lichstein, Durrence, Taylor, Bush, & Riedel, 2003).

According to these criteria, the presence of syndromal chronic insomnia is indicated when symptoms are described as chronic for at least 1 month and occur at a frequency of at least three nights per week, and when clinically relevant, daily consequences (e.g., decreased functioning at work and/or in social and personal life) are reported.

The questionnaire allows the identification of three categories of sleep quality:

1. Good sleepers (GS), which characterises people who report no sleep problems.
2. Persistent syndromal insomnia (PI), which characterises people who report clinically significant symptoms of insomnia on the basis of diagnostic criteria.
3. Subthreshold insomnia (SI), which characterises people who report symptoms of insomnia with frequency, persistence, or consequences lower than those indicated by diagnostic criteria. The validity of this classification regarding sensitivity and specificity was reported in a previous study (Violani et al., 2004), which evidenced that the SDQ has a sensitivity of 95% and a specificity of 87%. Because the SDQ gives a categorical measure, a second instrument was added for having a parametric measure of insomnia severity.

### **The Insomnia Severity Index (Bastièn, Vallières, & Morin, 2001)**

Insomnia Severity Index (ISI) was chosen because it assesses the severity of insomnia during the preceding 2 weeks. A total score, ranging from 0 (insomnia absent) to 28 (very severe insomnia), is computed by summing the scores on the seven items. A score  $\leq 7$  is considered the cut-off for the absence of clinically significant insomnia.

### **The Depression Questionnaire**

Depression Questionnaire (DQ) included in the Cognitive Behavioral Assessment Battery 2.0 (Sanavio et al., 1997).

DQ is a 24-item questionnaire measuring the intensity of depressive states. Respondents are required to choose a yes/no response indicating if each item describes her/his current experience. A total score is calculated, which is a valid and reliable measure of the severity of depressive symptoms. In the present study, one item dealing with sleep problems was not considered for the total score in order to avoid that differences among groups were inflated by the presence of sleep problems in some of the participants.

### **Data Preparation and Statistical Analyses**

The sleep groups classified on the basis of the SDQ were compared in respect to body mass index, age, and depression using one-way analyses of variance (ANOVAs).

**Table 1** Distributions of participants (N = 1019) across the three groups obtained on the bases of the Sleep Disorders Questionnaires

Groups	Frequency	Percentage (%)	% of underweight (N = 147; 15.2%)	% of overweight (N = 91; 9.4%)	% of normal weight (N = 727; 75.3%)	DEQ > 30 (%)
Good sleepers	175	17.2	15.80	6.60	77.60	6.9
Subthreshold insomnia	680	66.7	15.10	9.6	75.20	24.2
Persistent insomnia	164	16.1	15.10	11.30	73.60	39.0

The percentage of people of normal weight, underweight, and overweight, and the percentage of people scoring above the cut-off score of 30 in the Disordered Eating Questionnaire (DEQ) within each group are also reported.

Significant results were analysed through Scheffé post-hoc tests. ISI scores were used as a continuous measure of insomnia severity in the correlation analyses. DEQ scores were used as a continuous dependent variable in an analysis of covariance (ANCOVA) comparing sleep groups defined on the basis of the SDQ. The covariate was the depression score. The DEQ scores were also used for dividing the sample into two subgroups on the basis of the clinical cut-off of 30.

The chi-square test was computed for evaluating the distribution of observed frequencies across sleep and disordered eating groups.

The relationship between DEQ and ISI scores was further evaluated using the SPSS macro that accompanies the article by Preacher and Hayes (2008) for testing a mediation model that considers the insomnia severity scores as predictor, DEQ scores as criterion, the depression scores as mediator and controlling for age. DEQ, ISI, and DQ scores were standardised before running PROCESS, as suggested by the authors. A bootstrapping procedure was used to obtain estimates and confidence intervals around the indirect effects.

All analyses were performed using SPSS 19 software for Windows.

## Results

### Descriptive Results

Self-reported height and weight were used to compute BMI (weight/height<sup>2</sup>). The BMI values for our sample

ranged from 14.84 to 34.89, with a mean value of 21.16 (SD 2.85). Table 1 provides the distribution of participants across groups, giving also information about the distribution of people classified as underweight, normal weight, and overweight according to World Health Organisation criteria.

BMI and age of the sleep groups (PI, SI, and GS) were compared using two one-way ANOVAs. As regards BMI, no difference was found, but the GS group was older than the other groups ( $P < 0.05$  for all post-hoc comparisons). Means and *SDs*, and the results of the ANOVAs are reported in Table 2.

### Differences in Depression and Disordered Eating

Depression scores were significantly different between the sleep groups (see Table 2 for the results) and Scheffé post-hoc tests evidenced that all groups differed from each other (all  $P < 0.001$ ). Means and *SDs* and the results of the ANOVAs are reported in Table 2.

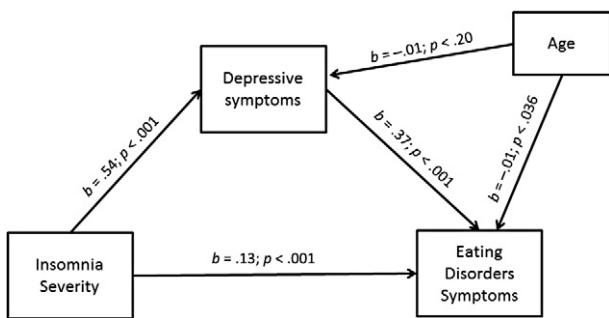
Results of the ANCOVA evidenced that the DEQ scores differed significantly across sleep groups. Scheffé post-hoc tests found that the PI and SI groups were not significantly different ( $P = 0.97$ ), whereas they both differed from the GS group ( $P < 0.001$ ). The covariate was significant ( $F_{(1,1014)} = 174.62$ ;  $P < 0.001$ ) and accounted for the 21% of the variance (adjusted  $R^2 = 0.208$ ). Means, *SDs*, and a summary of the results are provided in Table 2.

Two groups were obtained based on the DEQ scores using the cut-off of 30: the Eating Disorders Asymptomatic

**Table 2** Results of the ANOVAs comparing age, nutritional status (body mass index), disordered eating symptoms, and depressive symptoms across groups defined on the basis of the Sleep Disorders Questionnaire (Violani et al., 2004)

	Good sleepers, M (SD)	Subthreshold insomnia, M (SD)	Persistent insomnia, M (SD)	F <sub>(df)</sub>	P
Age	25.40 (4.64)	24.28 (4.05)	23.71 (3.62)	7.835 <sub>(2,1015)</sub>	0.001
Body mass index	20.92 (2.45)	21.18 (2.90)	21.32 (2.97)	0.794 <sub>(2,962)</sub>	0.452
Depression Questionnaire	1.30 (2.00)	3.31 (3.53)	6.40 (4.40)	92.871 <sub>(2,1015)</sub>	0.001
Disordered Eating Questionnaire	11.07 (11.41)	20.51 (16.90)	26.98 (18.99)	40.820 <sub>(2,1016)</sub>	0.001

Depression score was included as a covariate in the ANOVA that compared the Disordered Eating Questionnaire across sleep groups. ANOVA, analysis of variance; *M*, mean; *SD*, standard deviation.



**Figure 1** Results of the mediation analysis. Only significant direct and indirect paths, including  $b$  for each path, are reported in the figure.

group ( $N = 779$ , scores  $<30$ ) and the Eating Disorder Symptomatic Group ( $N = 240$ , scores  $\geq 30$ ). The distribution of these groups was significantly different across the sleep groups ( $\chi^2_{(2)} = 49.016$ ;  $P < 0.001$ ). The prevalence of individuals classified as symptomatic based on the DEQ was lower than expected within the GS (standard residuals  $-4.6$ ) as compared with the SI (std. residuals  $0.3$ ) and PI (std. residuals  $4.1$ ) groups.

### Assessing the Mediational Role of Depression on the Relationship Between Insomnia and Eating Disorder Symptoms

The mediation model tested the direct effect of insomnia severity on severity of the eating disorder symptoms and the indirect effect through the mediation of depression, using age as covariate. The total model is statistically significant ( $R^2 = 0.113$ ;  $F = 53.81$ ;  $P < 0.001$ ). The direct and indirect significant paths are reported in Fig. 1, which also includes the  $b$  for each path.

Depression results a significant mediator of the relationship between insomnia severity and severity of eating disorder symptoms, and the direct path is also significant.

### Discussion

The sleep problem considered in the present study is the most widely prevalent in community samples (Ohayon & Reynolds, 2009), namely insomnia, defined according to the combined criteria indicated by the DSM-IV and international consensus reports (Edinger et al., 2004). Insomnia symptoms were evaluated by two valid and reliable instruments, the SDQ (Violani et al., 2004) and ISI (Bastièn et al., 2001). Disordered eating was evaluated using a questionnaire whose validity has been demonstrated in both community (Lombardo et al., 2004) and clinical samples (Lombardo et al., 2011). In the present study, a measure of depression was also included. As the sample was non-clinical, a brief and reliable instrument

was selected, which offered a good level of sensitivity for depressive mood in the non-clinical range (Sanavio et al., 1997).

The results of our study confirm that insomnia symptoms and eating disorder symptoms covary systematically. We found that higher severity of insomnia symptoms is associated with higher severity of disordered eating, and scores above the clinical cut-off in the DEQ were more frequent than expected in groups with clinically severe insomnia symptoms. BMI was not significantly related to severity of insomnia symptoms. This lack of correlation may be due to the fact that poor sleep and insomnia are related to both the underweight and the overweight. However, it is also possible that it is due to the use of self-reported height and weight for assessing BMI, which is usually considered non-reliable. Future studies will address this issue. However, we are confident that this alternative explanation is not highly probable for two reasons. First of all, a previous study (Lombardo et al., 2011) evidenced that the correlation between the BMI computed on the bases of self-reported and objectively obtained height and weight was 0.95. Furthermore, the lack of relationship between BMI and sleep disorders in a non-clinical sample as in our study, is consistent with the results of previous community studies (Soares et al., 2011).

Both insomnia and disordered eating were shown to be related to depression. Their relationship is still present even after partialing out the effect of the depressive mood through the ANCOVA analysis. The correlation between ISI and the DEQ scores was significant and quite relevant, as it explained approximately 11% of the variance. The mediating role of depression in this relationship was also assessed. Results evidenced that even after controlling for age, both the direct and the indirect paths are significant.

In conclusion, the results summarised earlier are all consistent with previous evidence indicating that sleep and eating disorders share a common variance (Soares et al., 2011) and that both share a common variance with depression (Daga et al., 2011; Staner, 2010). Furthermore, the results of the present study indicate that both a direct and indirect relationship exists between eating and insomnia symptoms. The direct relationship appears lower than the indirect, at least in this non-clinical sample. However, it is plausible that both direct and indirect influence is relevant for determining the severity of the clinical condition, and the probability of developing a sleep or an eating disorder when a depressive mood is also present. It is also possible that sleep problems, when present, increase the probability of developing more severe depression and eating disorders, as a recent meta-analysis (Baglioni et al., 2011) seems to suggest at least for depression.

Perhaps the complex relationship between sleep, eating, and depressive symptoms would be better disentangled by considering the role of depression in clinical samples, including longitudinal designs, and assessing the mediational effect of depression through multidimensional measures and multivariate statistical analyses. Furthermore, the role of other variables known to modulate the relationship between mood and psychopathology should be considered, namely measures of life stress events, coping, and strategies for regulating emotion.

The link between sleep, eating, and depressive symptoms should also be evaluated considering possible clinical implications. A recent work by Borsboom, Cramer, Schmittmann, Epskamp, and Waldorp (2011) has identified a network model of psychiatric symptoms based on the evidence that comorbidity is the rule rather than the exception. Considering the DSM-IV, the authors found that symptoms of insomnia were connected with the largest number of other psychiatric symptoms. As a consequence, including treatment components directed to reduce sleep difficulties and related cognitive and emotional aspects into the routine psychological therapy for eating disorders and depression could increase effectiveness of the primary intervention and obtain a better outcome. Regarding depression, evidence supporting this hypothesis has already been published (Manber et al., 2008, 2011). On the other hand, the treatment of insomnia might benefit from the inclusion of strategies directed to related symptoms, as for example, the cognitive and emotional aspects of the daytime consequences of insomnia, as the results of a study by Harvey, Sharpley, Ree, Stinson, and Clark (2007) seem to confirm.

Before concluding, several limitations of the present study should be recognised. First of all, the correlations found in the present study, even though significant, were small, and this is probably due to the non-clinical sample examined. Future studies considering clinical samples are needed before generalising the relationships found. Second, our results evidenced that insomnia severity predicts eating disorder symptoms directly and through the mediation of depression; however, the cross-sectional design limits this conclusions and needs to be replicated with a longitudinal design. Actually, previous evidence derived from longitudinal studies confirms that insomnia predicts depression. However, the longitudinal prediction of disordered eating needs to be confirmed. Finally, the instrument used for assessing eating disorder symptoms is not commonly used in eating disorder studies, and this may limit the generalisability of the results. However, we are confident at least on the validity of this measure because previous studies using the DEQ (Lombardo et al., 2011) evidenced that its correlations with the Eating Disorder Examination clinical interview (e.g., Fairburn &

Cooper, 1993) are even bigger than those obtained with the Eating Disorder Examination Questionnaire (Mond, Hay, Rodgers, Owen, & Beumont, 2004); furthermore, sensitivity and specificity of the DEQ are comparable with that of the EAT-40 and greater than that of the EDI-2 (e.g., Lombardo et al., 2011).

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