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Longitudinal Bi-directional Effects of Disordered Eating, Depression and Anxiety

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Abstract

Objective: To explore the potentially longitudinal bi-directional effects of disordered eating (DE) symptoms with depression and anxiety.

Method: Participants were 189 (49.5% males) adolescents from Melbourne, Australia. DE, depressive, and anxiety symptoms were assessed at approximately 15, 16.5, and 18.5 years of age.

Results: Analysis of longitudinal bi-directional effects assessed via cross-lagged models indicated that DE symptoms of eating and shape/weight concerns were risk factors for anxiety. Results also showed that depression was a risk factor for eating concerns.

Conclusion: Our findings provide preliminary evidence that preventative measures designed to target concerns about eating and shape/weight might be most efficacious in reducing the transmission of effects between symptoms of DE, depression and anxiety.

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1. LONGITUDINAL BI-DIRECTIONAL EFFECTS OF DISORDERED EATING, DEPRESSION, AND ANXIETY

Disordered eating (DE) is a multidimensional construct that encompasses a range of maladaptive behaviours and cognitions, including dietary restraint (restricting caloric intake with the aim of losing weight to achieve a desired weight/shape) and concerns about eating, shape, and weight (Fairburn & Beglin, 1994). Estimates for the prevalence of DE symptoms in the general population are as high as 61% (Neumark-Sztainer, Wall, Larson, Eisenberg, & Loth, 2011), and research indicates that individuals with an eating disorder have an increased likelihood of reporting a mood (odds ratio = 5.20) and/or anxiety (odds ratio = 3.49) disorder (Johnson, Cohen, Kasen, & Brook, 2002).

Although the co-occurrence of DE and mood/anxiety disturbances is well established, the nature of this association remains unclear. As a result, the mechanisms that generate these comorbidities remain elusive which, in turn, limits insight into prevention and treatment efforts to reduce these high levels of comorbidity. Factors that have potentially limited our understanding regarding the mechanisms that underpin this comorbidity is that eating pathology consists of a pattern of symptoms which might be differentially related to mood/anxiety, and that researchers have tended to explore eating pathology symptoms at broad (at the level of disorder) as well as specific (at the level of symptoms) loci.

For a sub-group of adolescents, prevalence rates of DE increase (relative to pre-adolescence) and remain stable into adulthood (Neumark-Sztainer et al., 2011).

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Further, many cases of DE and mood/anxiety disturbances have onset in adolescence (Cyranowski, Frank, Young, & Shear, 2000; Kessler et al., 2005; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011). It is possible that these comorbid effects between DE and depression and anxiety symptoms (henceforth depression and anxiety) are transmitted via uni-directional associations where there is a continued effect of one variable on another over time, or a bi-directional association, whereby the two variables influence each other over time. The accumulated evidence of increases in prevalence of DE, depression and anxiety during adolescence suggests that the transmission of these constructs may typically take place during this developmental period and hence, assessing individuals during this age range to test causal/longitudinal association hypotheses is warranted. Therefore the aim of this study was to explore the potentially bi-directional longitudinal association between DE with depression and anxiety during adolescence.

1.1. Potential Reasons for the Link Between Disordered Eating with Depression and Anxiety

DE has been proposed to lead to depression (Tanofsky-Kraff et al., 2011) and anxiety (Pallister & Waller, 2008) via a failure to meet unrealistic body image standards due to dissatisfaction/concerns about one's perceived weight and/or shape status. Others have suggested that depression (Heatherton & Baumeister, 1991) and anxiety (Bulik, Sullivan, Fear, & Joyce, 1997) may be etiological to developing DE, as DE might be used as an attempt to reduce negative internal mood states and/or be sequelae to anxieties associated with sociocultural pressures to attain the thin ideal.

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These two viewpoints propose that one construct confers risk to the other via a uni-directional association (e.g., that one construct is a risk factor for the other). However, as these viewpoints are not mutually exclusive, it is possible that the constructs are bi-directionally related (Boujut & Gana, 2014; Fairburn, Cooper, & Shafran, 2003), such that each construct confers risk to the other, and that both maintain each other in a feedback loop. This bi-directional theory proposes, for example, that if DE is used to reduce aversive internal mood states, it may unwittingly lead to further negative mood if the solution is ineffective, thereby creating a cycle of depression/anxiety→DE→ depression/anxiety. A fourth suggestion has been that the constructs are linked through shared risk factors (e.g., genetics; Munn-Chernoff et al., 2015). To date, it is unclear which aforementioned explanation(s) is most plausible.

1.2. Disordered Eating and Depression

Extant literature indicates a high level of comorbidity between eating difficulties and depression (e.g., up to approximately 50%; Swanson et al., 2011), and that the relationship between DE and depression might be bi-directional. In support of this, a recent meta-analysis on 30 longitudinal studies (Puccio, Fuller-Tyszkiewicz, Ong, & Krug, 2016) identified that symptoms of DE and depression were risk factors for each other, lending validity to the view that these symptoms are bi-directionally related. In their meta-analysis, Puccio et al. (2016) found that the effect of DE on depression and vice versa was differentially moderated by different operationalizations of DE; for example, the effect of DE on depression was stronger

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for overall DE symptoms relative to bulimic and binge-eating symptoms. By contrast, other recent studies have suggested either a uni-directional longitudinal association where binge-eating symptoms predicted depression (Sehm & Warschburger, 2016), or a non-significant relationship between dysfunctional food consumption or dieting with depression (De Caro & Di Blas, 2016). Thus, the longitudinal DE – depression relationship might be subserved by specific symptoms of DE, or at the very least, that distinct symptoms of DE confer increased risk for developing depression and vice versa.

1.3. Disordered Eating and Anxiety

Despite the high comorbidity rates between anxiety and eating difficulties (e.g., 65%; Swinbourne et al., 2012), very few longitudinal studies have assessed evidence for their direction of effects (Pallister & Waller, 2008). Two studies (Procopio, Holm-Denoma, Gordon, & Joiner, 2006; Zaider, Johnson, & Cockell, 2002) to our knowledge have examined evidence for a longitudinal bi-directional relationship between eating difficulties and anxiety. Zaider et al. (2002) found no longitudinal relationship between either a threshold or sub-threshold eating disorder (binge-eating disorder or bulimia nervosa) and an anxiety disorder (generalized anxiety disorder or panic disorder). Procopio et al. (2006) reported in a community sample of females (M = 45 years, N = 150) that baseline anxiety symptoms predicted an increase in bulimic symptoms 2.5 years later but not vice versa. It should be noted, however, that as the majority of research into DE has focused on adolescent/young

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adult females, the results of Procopio et al.'s (2006) study might not be representative of the transmission of these effects in adolescence.

Other longitudinal studies have shown significant effects of binge-eating symptoms on anxiety in girls (Tanofsky-Kraff et al., 2011). Similarly, an eating disorder diagnosis has been shown to predict an anxiety disorder in adolescent females (Johnson et al., 2002) and females and males (Micali et al., 2015). By contrast, anxiety symptoms have been shown to predict DE symptoms (Hautala et al., 2011), and an anxiety disorder diagnosis has been shown to predict bulimic symptoms, but not dietary restraint (Johnson et al., 2002). Other longitudinal studies have found that anxiety symptoms failed to predict overall DE symptoms (Le Grange et al., 2014), bulimic symptoms (Gilbert & Meyer, 2005), and an eating disorder diagnosis (Fichter, Quadflieg, & Rehm, 2003).

Thus to date, the transmission of effects between different DE symptoms with depression and anxiety might vary according to the respective symptom type that is assessed. Despite this possibility, no study has assessed whether different symptoms of DE are differentially linked to depression and anxiety in the same sample, and vice versa.

1.4. Limitations of Past Studies

Although comorbidity between disordered eating, mood and anxiety disturbances has been established, it is unclear which direction (if not both) the effects flow between. Furthermore, while many explanations for the link between the constructs focus on

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specific aspects of disordered eating as mechanisms linking to depression/anxiety, many prior studies (e.g., (Johnson et al., 2002; Zaider et al., 2002) instead focus at global, diagnosis level effects, and as such, fail to test these linkages directly. Of further concern is that an individual – particularly one in the subclinical range – may have severe scores on a particular symptom or subset of disordered eating symptoms that adversely affect their lives, but these individuals might be excluded in studies that assess individuals who meet criteria (across all symptoms) and/or may be under-represented. Hence, we urge focus on symptom severity to: (1) not neglect the fact that an individual can have severe symptoms in one domain without showing (or yet expressing) symptoms in another, and (2) identify mechanisms linking disordered eating to depression/anxiety.

1.5. The Current Study

The sample of this study comprised individuals participating in the Orygen *Adolescent Development Study* (ADS), a longitudinal study conducted in Melbourne, Australia. Given the above-mentioned limitations, and that past research has yet to identify which symptoms of DE, depression and anxiety might drive their transmission of effects, our study sought to overcome these shortcomings by testing whether different facets of DE related differentially to depression and anxiety in a sample of individuals whose age range fell within the peak onset for each of these disturbances. We assessed an Australian adolescent sample at three time points to investigate if DE symptoms of dietary restraint, eating concerns and shape/weight

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concerns were bi-directionally related with either depression or anxiety. The EDE-Q is a commonly used measure of DE that assesses these aforementioned aspects of DE, and was thus utilized to examine these relationships. As the literature on the longitudinal effects between depression and restraint is inconclusive, and because no study has assessed the potentially bi-directional effects between the eating and shape/weight scales of the EDE-Q separately with depression and anxiety, the present study made no hypotheses regarding the expected direction of effects.

2. METHOD

2.1. Participants and Procedure

Within this larger study, 2,479 Grade 6 students (aged 10-13 years) from schools across metropolitan Melbourne were initially screened at their school using the Early Adolescent Temperament Questionnaire- Revised (EATQ-R; Ellis & Rothbart, 2001). Four hundred and fourteen participants were then selected and invited to participate in the intensive assessment phases based on their scores on the Negative Emotionality and Effortful Control dimensions of the EATQ-R, which were used as a marker for being either low or high risk for developing emotional and behavioral problems. Of this group, hundred and twelve individuals agreed to participate.

Participants were selected so that equal numbers of males and females were recruited across the range of 0–1, 1–2, 2–2.5, and greater than 2.5 standard deviations above and below the mean on each subscale. Participants for the current study

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initially included 214 individuals who had been selected to participate in the study based on the screening procedure (49.5% males). Participants (N = 25) were excluded from the study if they did not provide data for at least 2 waves, resulting in a total sample of 189 individuals (46.8% males). This sample size was comparable to other studies (Gilbert & Meyer, 2005; Tanofsky-Kraff et al., 2011) that have also examined the longitudinal transmission of effects between mood and DE symptoms.

Participants were asked to complete questionnaires at 3 time points, on average two years apart. Participant ages ranged from T1: 13.74 years to 16.18 years, M = 15.03, SD = .43; Time 2: age range: 15.42 years to 18.11 years, M = 16.60, SD = .57; and Time 3: age range: 17.30 years – 19.96 years, M = 18.90 years, SD = .46. Informed consent was obtained from one parent and the adolescent when the adolescent was under the age of 18, and from just the adolescent when they were over the age of 18. Of the 189 individuals in the study, the majority of participants' parents were from Australia (74.1%), UK (7.5%), and New Zealand (0.9%). This study was approved by a human research ethics committee at a university in Australia.

2.2. Measures

Scale reliabilities are presented in Table 1.

2.2.1. Socio-economic status.

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Socio-economic status (SES) was calculated using the Australian National University-4 (ANU4) scale for occupations (Jones & McMillan, 2001), which provides a score between 0 and 100, representing participants' social stratification. For parents that had missing data, number of years of education, scaled to reflect ANU4 codes, was used as a substitute (Marks, McMillan, Jones, & Ainley, 2000). The mean levels of parents' SES scores for males (M = 59.24, SD = 20.61) and females (M = 56.74, SD = 20.87) in this sample were slightly higher than average.

2.2.2. Depression.

Adolescent depressive symptomatology was measured using the Center for Epidemiological Studies—Depression Scale, Revised (CES-D; Radloff, 1977). The CES-D is a 20-item self-report measure that assesses levels of depressive symptoms over the past week, and has been found to be valid and reliable for adolescents (Radloff, 1977). Questions pertain to cognitive, behavioral and interpersonal aspects of depression that occurred during the past week. Items are rated on a 4-point Likert-type scale with responses ranging from 0 = Rarely or None of the Time (Less than one day) to 3 = Most or All of the Time (5–7 days).

2.2.3. Anxiety.

The Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) was used to assess anxiety symptoms. The BAI is a 21-item self-report measure that assesses cognitive and physiological symptoms of anxiety over the past week. Participants rate items according to the degree they endorse a particular symptom.

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Each item is rated on a four-point scale ranging from 0 (not at all) to 3 (severely, I could barely stand it).

2.2.4. Disordered eating.

The Eating Disorders Examination-Questionnaire (EDE-Q 6.0; Fairburn & Beglin, 1994) was used to assess the following symptoms of DE; dietary restraint, eating concerns, shape/weight concerns, and an overall total score. This three-factor structure (i.e., dietary restraint, eating concerns, and shape/weight concerns) was utilized based on findings from recent research (e.g., White, Haycraft, Goodwin, & Meyer, 2014). These scale scores assess the frequency and severity of DE cognitions and behaviours over the past 28 days. Items are rated on a 7-point Likert-type scale with responses ranging from 0 = No days/6 = Everyday and "not at all"/"markedly".

2.3. Statistical Analyses

All analyses were performed using SPSS version 23 and Mplus version 7.2. Path analysis with continuous manifest variables was utilised to test the specified bi-directional models. The direction of effects between DE symptoms with depression and anxiety were tested by fitting a manifest variable autoregessive cross-lagged model. This approach has been considered a valid approach for examining reciprocal feedback loops using cross-lagged modelling (Martens & Haase, 2006). Variance in the outcome variable (e.g., DE, depression and anxiety) at T2 and T3 was predicted from three main sources; the effect of the same construct at T1 and T2, respectively

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(autoregressive path), the effect of the covariate at T1 and T2 (i.e., anxiety and depression as a covariate for the models assessing a bi-directional relationship between DE with depression and anxiety respectively), and the effect of the predictor at T1 and T2 (bi-directional paths).

The following cutoffs for standard fit indices were used in the present study: chi square is non-significant (p>.05), CFI and TLI >.95 for excellent fit, and >.90 for adequate fit; RMSEA <.06 for acceptable fit, and >.10 for poor fit (Marsh, Hau, & Wen, 2004). Given the high degree of comorbidity between depression and anxiety (Cummings, Caporino, & Kendall, 2014) each construct was controlled for in the relevant bi-directional models. Each model also controlled for participant sex.

Missing data were handled using the maximum likelihood estimator in MPlus. Upper bound rates of missing data were: T1 = 4%, T2 = 8%, and 11% at T3. Little's missing completely at random (MCAR) analysis indicated data were consistent with a MCAR pattern of missingness; $\chi^2(12371) = 10734.56$, $p = 1.000$. Absolute skew values indicated that several variables (T1 and T2 dietary restraint and eating concerns at T1, T2 and T3) exceeded 2, suggesting that the assumption for univariate normality was not satisfied (Kline, 2015). To address the issue of skewed variables, we used the MLR estimator in Mplus which is a maximum likelihood estimator with robust standard errors to account for non-normal data (Muthén & Muthén, 1998).

3. RESULTS

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Tables 1 presents the descriptive statistics, scale reliabilities, and percentages of participants scoring in clinical range for each scale across respective waves across each wave. Table 2 presents Pearson's correlations for all variables for males and females separately. Table 3 summarizes the model fit statistics for the bi-directional relationships between DE with depression and anxiety. Results indicated that all models fit the data well.

----Insert Table 1 about here----

----Insert Table 2 about here----

----Insert Table 3 about here----

Figures 1a-c present the results for the bi-directional effects between DE with depression and with anxiety. Results showed that T2 eating and shape/weight concerns sub-scales predicted T3 anxiety. Results also illustrated that T2 depression predicted T3 eating concerns.

----Insert Figure 1a-c about here----

4. DISCUSSION

The aim of this study was to assess whether different DE symptoms and depression and anxiety were bi-directionally related. To our knowledge, this is the

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first study to assess these relationships congruently.

4.1. Disordered Eating and Depression Bi-Directional Models

The present findings highlight the importance of disambiguating the respective facets of DE when examining their influence on depression, and vice versa, as only one of the three DE scales (T2 depression predicting T3 eating concerns) showed a significant longitudinal relationship with depression.

The eating concerns subscale of the EDE-Q assesses negative cognitions linked to the act of eating. Past researchers (Heatherton & Baumeister, 1991) have proposed that DE is a compensatory mechanism to reduce negative mood states. Our finding that T2 depression predicted T3 eating concerns supports this view, suggesting that depression not only leads to actual eating *behaviours* (e.g., binge-eating; Puccio et al., 2016), but that it also confers risk to *cognitions/rumination* that are focused on eating.

Our results are therefore consistent with a recent meta-analysis (Puccio et al., 2016), which indicated that DE and depression are risk factors for each other. It should be noted, however, that unlike Puccio et al.'s (2016) review, which showed that DE and depression were concurrent risk factors, the present study found that the transmission of effects occurred during different periods in adolescence for different DE subscales. It is unclear why the results of this study showed that the effect of DE on depression changed as a function of age, and hence, future research would benefit from examining these longitudinal links.

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Regarding the possibility that DE and depression are generated by shared risk factors, a past review (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008) has shown that rumination, defined as the cognitive process of repetitively and passively focusing on distressing thoughts, is a risk factor for both depression and DE. The results of this study lend support to the idea that depression and DE might be linked via a tendency to ruminate on distressing aspects related to the self. For example, rumination over a perceived sense of worthlessness may be etiological to developing/maintaining depression, and rumination over a perceived sense of shame regarding one's shape and/or eating patterns may be etiological to developing/maintaining DE.

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4.2. Disordered Eating and Anxiety Bi-Directional Models

To our knowledge, this is the first study to assess the bi-directional relationship between different symptoms of DE with anxiety. Results indicated that DE was a risk factor for anxiety as eating concerns and shape/weight concerns at T2, predicted anxiety at T3. The dietary restraint scale was not significantly related to anxiety. Thus, results show that the transmission of these effects varied as a function of both age and symptom type as all observed significant effects occurred from T2 to T3. To date it remains unclear which, if any, factors influence these possible developmental differences and thus further research is required to investigate the

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etiology of these linkages.

In addition to the eating concerns subscales illustrated above, the shape/weight concerns subscale assesses concerns with one's perceived shape/weight status. The shape/weight concerns subscale of the EDE-Q assesses concerns/rumination relating to one's perceived body shape and weight status. Our results indicate that these negative cognitions around one's perceived shape/weight during early adolescence are a risk factor for anxiety. It is possible that a focus on one's perceived body shape/weight might highlight that one's perceived shape falls short of the thin-ideal. This, in turn, might lead to increased body dissatisfaction, and thereby predispose individuals towards symptoms of anxiety. Our results indicate that concerns focusing on one's body dissatisfaction (e.g., shape and weight concerns) and eating-patterns, likely drive the risk that DE confers to anxiety. This finding is novel. Further, the majority of these effects were observed during later adolescence, with participants' mean age at T3 being approximately 19 years. In this context, our results suggest that the risk of DE on anxiety is also influenced by age, as well as symptom type. However, because different types of anxiety disorders have been shown to peak at different ages during adulthood and adolescence (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012), future research would benefit from examining whether these effects replicate across a range of anxiety disorders.

While preliminary in nature, the present results are consistent with past research (Johnson et al., 2002; Tanofsky-Kraff et al., 2011) in supporting the view that DE generates anxiety, likely via the negative impact of a failure to meet

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unrealistic body image standards. For example, dissatisfaction/concerns about one's perceived weight, shape, and/or eating status might generate anxiety as these concerns 'shift' individuals further away from sociocultural norms of thinness (Pallister & Waller, 2008). As well as this, these results also highlight the important role that DE-related concerns/rumination play in the etiology of anxiety.

The results of this study are in conflict with past research (Hautala et al., 2011; Procopio et al., 2006) which has shown an effect of anxiety on DE/an eating disorder diagnosis. However, our findings are congruent with other studies that reported a non-significant relationship (Fichter et al., 2003; Le Grange et al., 2014; Zaider et al., 2002). As identified previously, it is possible that these differences in results be attributed, in part, to different conceptualizations of eating difficulties and anxiety. Due to the novel nature of this study, further research is required to replicate these findings.

4.3. Limitations

Given the self-report methodology of this study, participants may have overestimated/underestimated their level of symptomatology. Thus, future designs would benefit from utilizing structured clinical interviews. In addition, the null relationships between many of the paths tested in this study may be due to low power, however it should be noted that the sample size of this study was comparable to past research (Gilbert & Meyer, 2005; Tanofsky-Kraff et al., 2011; Zaider et al., 2002). Despite some pathways having non-significant unique effects on the dependent

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variables, the combined effects of the predictor variables for the various dependent variables seem to be meaningful and sizable (as per guidelines regarding acceptable minimum threshold R2 values; see Ferguson, 2009). When viewed with the pattern of bivariate correlations observed, the non-significance of some pathways in the model may reflect that they overlap with other predictors in the model, and hence have limited unique variance to explain in the dependent variable rather than being entirely unrelated to the dependent variable. Finally, participants of this study were selected to reflect a risk-enhanced sample as they were over-selected for both risk and resilience for emotional problems. Our sample might therefore not be representative of the general population, and hence, future research that assesses the relationships examined in this study utilizing other community and clinical samples is required.

4.4. Clinical Implications

The results of this study highlight that efforts to target specific facets of DE, specifically concerns about body dissatisfaction (e.g., shape and weight concerns) and eating concerns, might be most efficacious in reducing the influence of DE symptoms on depression and/or anxiety, and vice versa. Our findings also raise the possibility that the comorbidity between DE with depression and anxiety might be generated and/or maintained, in part, by a negative thinking style typified by worry and/or rumination.

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4.5. Conclusions

Results indicated that the longitudinal transmission of effects between symptoms of DE with depression and anxiety vary as a function of symptom type and age. Our findings illustrate the importance of disambiguating the respective facets of DE symptoms to understand the relative risk that each facet confers to depression and anxiety, and vice versa. The results of this study suggest that future models that examine the bi-directional relationship between DE symptoms and depression should also consider examining the influence of anxiety, and vice versa.

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Disclosure of Conflicts of Interest

Conflicts of interest to disclose: Nil.

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