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Research Article

A Multilevel Mediation Model to Investigate the Effect of Sleep Quality as a Mediation Factor on the Association between Secondhand Smoking and Depressive Symptoms

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Abstract

We investigate in this study the impact of sleep quality as a mediating factor in the association between second hand smoking and depressive symptoms. The study included 196 pregnant women interviewed during and after pregnancy. A multilevel mediation model was applied to the data. Our results suggested that exposure to second hand smoking (ETS) positively predicted sleep quality 0.314, 95% CI [0.124, 0.504], $\rho = 0.001$.

In turn, sleep quality was positively related to the depressive symptoms 0.551, 95% CI [0.122, 0.98], $\rho = 0.012$. As a result, the between-cluster indirect effect of ETS on depressive symptoms was 0.173 (= 0.314 × 0.551). The distribution of the product of the coefficients 95% CI for the indirect effect was [0.028, 0.375], which did not contain zero. This indicates that the indirect effect of ETS on depressive symptoms through sleep quality was significantly different from zero. Therefore, the results show that the association of ETS on depressive symptoms was completely mediated by sleep quality. These findings highlight the need to develop interventions for pregnant women who are exposed to low sleep quality. Reducing exposure to environmental tobacco smoke in the antepartum period may be an important step in mitigating the risk of peri-natal and post-patum depression among vulnerable populations. We think that improving sleep architecture may improve the quality of life for pregnant women and their infant.

Introduction

Poor sleep quality has been shown to be associated with adverse birth outcomes including low birth weight, small for gestational age, preeclampsia, and preterm delivery [1-3]. Also, studies have linked disturbed sleep to both depressed mood [4-6] and stress during pregnancy [5] and has also been shown by others to be associated with depressive mood postpartum [7-9]. A combination of chronic stress, sleep disturbance, and perinatal depression symptomatology has been established to jointly activate the hypothalamic-pituitary-adrenal (HPA) axis and progesterone (PROG)-derived gamma-amino-butyric acid (GABA)-ergic neurosteroids, each of which has been linked to reproductive mood disorders [10-13].

Depression affects approximately 10–15 % of women during the perinatal period [14] and has been linked to maternal functional impairment [15], poor birth outcomes [16], subsequent postpartum depressive symptoms and poor quality of life [17]. Approximately 40–50% of underprivileged women suffer from prenatal depressive symptoms [15]. These rates are especially greater among low income African American women, since they are less likely to receive mental health services than their pregnant counterparts [18]. Published literature has established an association between smoking and depressive symptoms [19] and this relationship persists among the pregnant population [20,21]. Despite the independent link between smoking and sleep on depressive symptoms, there is limited information in published reports regarding the influence of smoking as a mediating factor between sleep quality and depressive symptoms during pregnancy. Understanding this link will not only provide more insight into the deleterious effects of sleep quality on maternal health, it will also provide an avenue for understanding the influence of maternal smoking on sleep quality and depressive symptoms during pregnancy.

Two types of mediation models for hierarchical data have been proposed in the literature [22, 23]. These are (1) Upper level mediation in which the effect of a Level 2 predictor on a Level 1 outcome is mediated by another Level 2 predictor $(2 \to 2 \to 1 mediation)$ and (2) Lower level mediation in which the effect of a Level 2 predictor on a Level 1 outcome is mediated by another Level 1 predictor $(2 \to 2 \to 1 mediation)$ or the effect of a Level 1 predictor on a Level 1 outcome is mediated by another Level 1 predictor $(1 \to 1 \to 1 mediation)$. We will consider in this study, a mediation model that explicitly consider that the impact of ETS on pre- and post-partum depressive symptoms differ on sleep quality during pregnancy. We assume a multilevel model in which the outcome (depressive symptoms) is measured at level 1, whereas the exposure (ETS) and the mediator (sleep quality) were measured at level 2.

Materials & Methods

Participant Data

The data is a prospective study that included pregnant women recruited from an outpatient health clinic affiliated with Tampa General Hospital in Tampa, Florida between November 2009 and July 2011 [21]. Eligible participants were women between the ages of 18 and 44 years old, less than 20 weeks of gestation. At baseline, women were screened for tobacco exposure using questionnaires administered to all study participants. These instruments were validated using Salivary cotinine measurements obtained from NicAlertTM. This test strips display seven levels from 0 to 6, with each level representing a range of cotinine concentrations. Participants who reported no tobacco use from the questionnaire and received a score of zero from the test strip were classified as non-smokers. Those who reported they were non-smokers but received a score of 1 on the test strip were classified as passive smokers. Participants with a cotinine score of 2 or above were classified as smokers. Sleep quality was also assessed at baseline using PSQI questionnaire, an instrument that has been previously validated for use in assessing sleep quality [24]. PSQI assessed sleep quality during the previous month and is composed of 19 items covering seven major domains including subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications and daytime dysfunction. Each domain score ranges from 0 to 3 (no difficulty to severe difficulty). The Edinburgh Perinatal Depression Scale (EPDS) was used to measure depressive symptoms during pregnancy and the postpartum period. The EPDS is a ten-item self-report screening scale that assesses a woman's depressive symptoms during the previous 7 days. Study participants were assessed for depressive symptoms at 5 time points throughout the study (baseline, third trimester, 2 4 and 6 weeks after delivery). The EPDS indicators are scored 0 to 3, however, without loss of generality, the EPDS indicators were categorized as binary variables (1 = if EPDS score>0, 0 = Otherwise).

Research questions: For this analysis, the research question was whether the relationship between passive smoking and depressive symptoms was mediated by sleep. When data on individual items are available, it is preferable to use latent variables instead of average (composite) scores for each construct to account for measurement errors [25]. Consequently, Sleep quality and depressive symptoms were modeled as latent variables whereas passive smoking was modeled as categorical variable. We did not include covariates in the study as previous publication using this data [21] did not identify other variables that were associated with passive smoking. (Figure 1) shows a path diagram for the causal relationships between the three variables: smoking status (), PSQI (), and depressive symptoms (). The upper level unit are pregnant women and the lower level unit is time (baseline, third trimester, 2, 4 and 6 weeks post-delivery). For this study, we posit that the Level 1 variable is assumed to be caused by two Level 2 variables, and . From Figure 1, the variable is associated with , and the variable is associated with . The direct effect of on would be and the indirect effect would be . The total effect denoted by would be $c' + ab$

Methods: The hierarchical structure of this dataset invalidates the majority of proposed methods (e.g. linear regression and path analysis) used in evaluating mediation data due to multiple limitations [26-28]. Because observations for this study were collected repeatedly overtime on the same individual, the measurements are nested within individuals. Because the independent variable () and the mediator variable () in the 2-2-1 design vary strictly between clusters, the direct and indirect effects in such a design are between effects; that is, there is no mediation at level 1. A variety of multilevel mediation models have been proposed for example the 1–1–1 designs that permits random intercepts and fixed or random slopes [22, 29-31], as well as for the 2–1–1 design that permits random intercepts and fixed slopes [22,29]. The model we will apply to this data will use a 2-2-1 design that permits random intercepts and fixed slopes [22, 30] and we will additionally utilize MSEM approach proposed by others [32] as follows:

Level 1 within measurement model:

$$Y_{ij} = \Delta \eta_{ij}$$

Level 2 between structural model:

$$\eta_j = \mu + \beta \alpha_j + \xi_j$$

where indexes Level 1 units and indexes Level 2 units. $\xi_j \sim MVN(0, \psi)$ .The η_j vector can be observed to contain the random effects that potentially varies at the cluster level. Since there is only one variable (y_{ij}) with within variation, there is no within effect (no direct or indirect within effect). The vector η_j contains the random coefficients, here the random intercepts from α_j. The β matrix contains the path coefficients making up the between effects (direct and indirect), which are obtained by

multiplying the between effect of X_j on $M_j(\beta_{MX})$ by the between effect of M_j on $Y_{ij}(\beta_{YM})$ [33, 34].

The residual terms ξ_j are multivariate normally distributed and independent across equations.

The above MSEM equations reduces to 2-2-1 mediation model with random intercepts and fixed slopes, of the form

$$Y_{ij} = \begin{bmatrix} M_j \\ Y_{ij} \end{bmatrix} = \Delta\eta_{ij} = \begin{bmatrix} 0 & 1 & 0 \\ 1 & 0 & 1 \end{bmatrix} \begin{bmatrix} \eta_{ij} \\ \eta_{Mj} \\ \eta_{Yj} \end{bmatrix}$$

$$\eta_j = \begin{bmatrix} \eta_{Mj} \\ \eta_{Yj} \end{bmatrix} = \begin{bmatrix} \mu_{\alpha\eta^X j} \\ \mu_{\alpha\eta^M j} \end{bmatrix} + \begin{bmatrix} \beta_{MX} & 0 \\ \beta_{YX} & \beta_{YM} \end{bmatrix} \begin{bmatrix} \alpha_n X_j \\ \alpha_n M_j \end{bmatrix} + \begin{bmatrix} \xi\alpha_n X_j \\ \xi\alpha_n M_j \end{bmatrix}$$

Model estimation: The random intercept model was fitted using restricted maximum likelihood estimation in MPLUS [34]. This method assumes normal distributions for error terms and random effects, to obtain estimators, we fixed the following with in cluster effects: the relationship between the outcome variable and the mediator (); the relationship between the outcome variable and the exposure (); and the relationship between the mediator variable and the exposure . However, the intercept was allowed to vary in the model. The between indirect effect was estimated using the product and . The confidence intervals of the indirect effects were obtained using the asymptotic normal theory. Even though this method is less efficient compared to the distribution of the product of coefficients method [34], M*plus*, does not incorporate the distribution of the product of the coefficient's method.

Results

A total of 236 pregnant women were enrolled into the study. However, 192 participants with complete data pre- and post-delivery were retained in this analysis. Table 1 presents demographic data of subjects in the study. As can be observed from this table, the data was collected on subjects in a high-risk area in Hillsborough County, Florida as majority of exposed subjects were black, obese and unmarried. Descriptive statistics for the nine EPDS indicators at Time 1 (during pregnancy) and Time 2 (after pregnancy) are displayed in (Table 1). With the exception of EPDS question 5 ("feeling that things have been getting on top of me"), more than 50% of study participants had a score of zero (EPDS score=0) for each of the EPDS indicators. Additionally, the proportion that had a score greater that zero were lower in time point 2 compared to time point 1 (Table 2).

Table 1: Distribution of baseline variables in the model.

Baseline Characteristics	Non-Smokers N=23	Second Hand Smokers N=106	Smokers N=107
Race			
white	7 (30.4)	19 (17.9)	27 (25.2)
black	7 (30.4)	51 (48.1)	38 (35.5)
Hispanics	7 (30.4)	34 (32.1)	38 (35.5)
others	2 (8.7)	2 (1.9)	4 (3.7)
Marital Status			
married	5 (21.7)	16 (15.1)	10 (9.4)
not married	18 (78.3)	88 (83.0)	94 (87.8)
not reported	0	2 (1.9)	3 (2.8)
Maternal Age			
< 30 years	15 (65.2)	85 (80.2)	88 (82.2)
≥ 30 years	8 (34.8)	21 (19.8)	19 (17.8)
Obesity Status			
obese	7 (30.4)	43 (40.6)	45 (42.1)
non-obese	8 (34.8)	26 (24.5)	28 (26.2)
not reported	8 (34.8)	37 (34.9)	34 (31.8)

Table 2: Frequency Distribution for the nine EPDS indicators at pre and post-delivery.

Indicator	Code	Label	During Pregnancy Frequency (%)	After Pregnancy Frequency (%)
[Not being] able to laugh and see the funny side of things	0	EPDS Score=0	140 (72.9)	163 (84.9)
	1	EPDS Score>0	52 (27.1)	29 (15.1)
[Not] have looked forward with enjoyment to things	0	EPDS Score=0	136 (70.8)	159 (82.8)
	1	EPDS Score>0	56 (29.2)	33 (17.2)
Blaming oneself unnecessarily when things went wrong	0	EPDS Score=0	108 (56.3)	113 (58.9)
	1	EPDS Score>0	84 (43.8)	79 (41.1)

Feeling sad or miserable	0	EPDS Score=0	106 (55.2)	112 (58.3)
	1	EPDS Score>0	86 (44.8)	80 (41.7)
Feeling that things have been getting on top of me	0	EPDS Score=0	76 (39.6)	81 (42.2)
	1	EPDS Score>0	116 (60.4)	111 (57.8)
Feeling unhappy that I have been crying	0	EPDS Score=0	107 (55.7)	109 (56.8)
	1	EPDS Score>0	85 (44.3)	83 (43.2)
Having been anxious or worried for no good reason	0	EPDS Score=0	100 (52.1)	110 (57.3)
	1	EPDS Score>0	92 (47.9)	82 (42.7)
Having been so unhappy that I have had difficulty sleeping	0	EPDS Score=0	134 (69.8)	136 (70.8)
	1	EPDS Score>0	58 (30.2)	56 (29.2)
Having felt scared or panicky for no very good reason	0	EPDS Score=0	122 (63.5)	126 (65.6)
	1	EPDS Score>0	70 (36.5)	66 (34.4)

Using the multilevel mediation model approach, we tested the hypothesis that sleep quality would affect depressive symptoms indirectly via exposure to ETS. Because sleep quality and exposure to ETS are level 2 measures and depressive symptoms is a level 1 measure, we fitted a a 2-2-1 design. We specified a random intercept and a fixed slope in the two-step approach. Overall, the model shows significant indirect effects, with a dose response relationship observed for smoking status as active smokers had the greatest indirect effect for the two time points considered in the study, followed by passive smoking mothers (Figure 1). This indicates that actively smoking mothers had the greatest effect on depressive symptoms followed by passive smoking mothers.

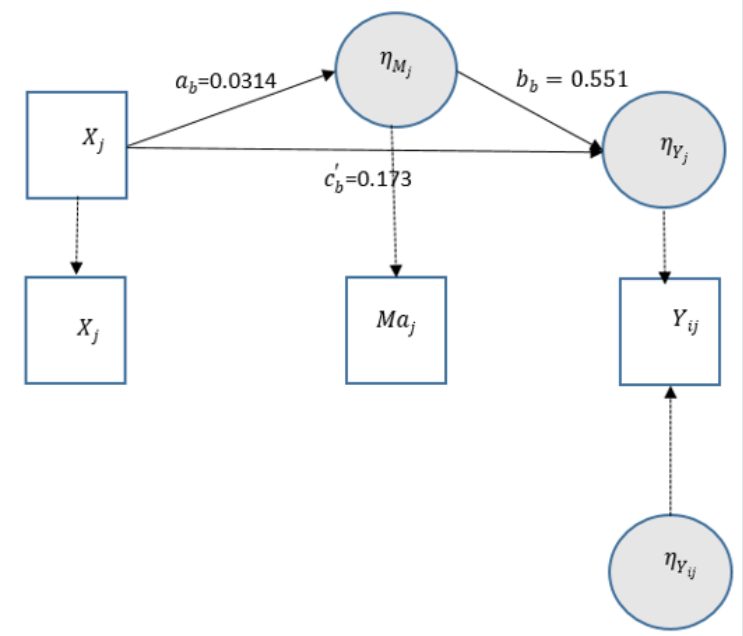

Figure 1: Upper and lower level mediation in a two-level model: Nested frames indicate levels of sampling, boxes indicate variables, arrows without circles represent fixed effects, arrows with circles represent random effects.

Our model also showed significant direct effects of exposure to ETS on depressive symptoms, sleep quality and exposure to ETS and sleep quality depressive symptoms. There was a dose response relationship in the direct effect of exposure to ETS and depressive symptoms with the greatest effect observed in actively smoking mothers. Also, the association in the direct effect between sleep quality and exposure to ETS was largest for mothers who smoked actively followed by mothers who were exposed to secondhand smoke.

In the between-cluster part of the model, ETS positively predicted sleep quality, $a_b = 0.314$, 95% CI [0.124, 0.504], $\rho = 0.001$. In turn, sleep quality was positively related to the depressive syptoms, $b_b = 0.551$, 95% CI [0.122, 0.98], $\rho = 0.012$. As a result, the between-cluster indirect effect of ETS on depressive symptoms was 0.173 (= 0.314 × 0.551). The distribution of the product of the coefficients 95% CI for the indirect effect was [0.028, 0.375], which did not contain zero. This indicates that the indirect

effect of ETS on depressive symptoms through sleep quality was significantly different from zero. Therefore, the results indicate that ETS on depressive symptoms was completely mediated through sleep quality.

The multilevel mediation model approach provides fit indices for the mediation model, and these indices showed satisfactory model fit (comparative fit index .94, Tucker–Lewis index .92, RMSEA .047, SRMRB .08, where SRMRB is the standardized root mean square residuals for the Between model). These indices indicate that mothers with lower sleep quality were more likely to smoke actively or exposed to secondhand smoking during pregnancy. The results of this study is unique as it presents methods that target specific kinds of clustered data within the context of multi-level modelling.

Discussion

In this study, we examined the relationship between trouble sleeping, exposure to ETS and depressive symptoms in a sample of pregnant women in Hillsborough County, Tampa, Florida. As expected, troubled sleeping was found to occur commonly in women who were exposed to secondhand smoke and was associated with significantly increased depressive symptoms during and after pregnancy. While this is not surprising, systematic measurement of sleep parameters has often been neglected in studies of women during and after pregnancy. Even though studies have shown smoking to affect sleep quality [5], none has been able to fit a model that includes sleep, mediating the association between secondhand smoking and depressive symptoms in a longitudinal setting. Poor maternal health can lead to adverse birth outcomes and impaired fetal health [6-10]. Our study shows an association between exposure to ETS and insufficient sleep patterns as well as an association between insufficient sleep patterns and depressive symptoms pre and post-delivery after accounting for exposure to ETS. These observations have been independently corroborated by numerous studies that have associated passive smoking with poor sleep patterns among pregnant women [5]. Ohidra et al discovered that pregnant non-smoking women who are exposed to ETS were likely to suffer from insufficient sleep, difficulty in initiating sleep, short sleep duration, and snoring loudly or breathing uncomfortably [5]. Other investigators have also shown that sleep deprivation is related to depressive symptoms, particularly in the postpartum period [35]. This collection of reports supports the hypothesis that impaired maternal health and poor sleep hygiene caused by passive smoking can lead to maternal antenatal and post-partum depression. Our result is unique compared to others because it investigates a three-way association in one model.

The findings from this work suggest that exposure to environmental smoke is a precursor for post-partum depression. Even though it is well established by us and others [21] that exposure to ETS is associated with antenatal and postpartum depression, this study has shown that the association is not only attributed mainly to tobacco smoke during pregnancy, but also to poor sleep quality. None of these studies have established sleep quality as a mediating factor in the association between second hand smoking and depressive symptoms during and after pregnancy. Including sleep quality as a mediating factor in a repeated measurements setting has helped in delineating the relationship between sleep quality, ETS exposure and maternal depressive symptoms during and after pregnancy. This methodological approach is vital because it has helped in establishing the trajectory of depressive symptoms in association with ETS and sleep quality, information that could provide potential time points for effective intervention.

A limitation of this study is its posited sample size, especially in the statistical analyses utilized. It should be noted that, sample sizes in structural equation modeling is a complex topic on which there is no general consensus. Others have proposed a minimum sample size ranging from 50 to 500 subjects [39]. To minimize the effect of sample size, we fit and tested a relatively simple model (2-2-1 mediation model) which lead to convergence and therefore reliable measures. This simple model was also limited in the sense that we could not get the within direct effect as variability of the data was constrained only at Level 2. Other models that could have been considered are Latent growth modeling, a SEM extension for longitudinal data that can flexibly evaluate mediating relationships between multiple time-varying measures, and the autoregressive and multilevel models that have been used by others for longitudinal mediation analyses with SEM [36]. The strength to this study is its longitudinal design that helps capture both within individual dynamics and between individual differences over time (pre- and post-delivery). The repeated measures design also offers a cost-effective approach to answering important questions regarding the effects of passive smoking and sleep quality on maternal health as it allows for a smaller sample size. Also, the design of the study has enabled us to observe the importance of time in assessing whether the mediator is more likely to be influenced by changes in the outcome, presenting more accurate representations of the temporal order of change over time that lead to more accurate conclusions about mediation[37].

In this article, we discussed how to conduct and interpret mediation analysis in hierarchical data sets to answer important research questions on depression symptoms during and after pregnancy. This allows researchers to estimate the structural relationships among constructs at between and within-cluster levels as well as modeling the measurement error and thus obtaining more reliable estimates of important epidemiological constructs. In summary, mediation analysis is a popular technique as it addresses important questions about how an independent variable impacts epidemiological outcomes by changing one or more intervening variables in a causal process. In epidemiologic research, mediation analysis can particularly help researchers

study causal effects and investigate how and why changes occur in human activities by identifying and targeting important mediators as shown in this study [38].

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