Exposure to Ambient Particulate Matter and Depressive Symptoms during Pregnancy

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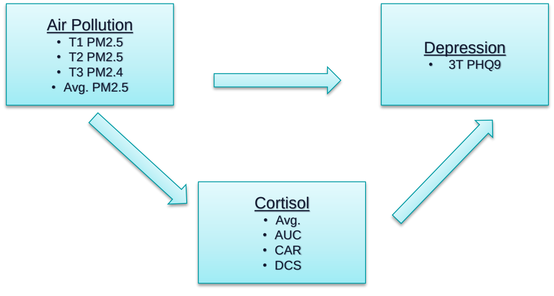
Background

An estimated 12% of women experience perinatal depression[1], with prevalence as high as 38% in certain high risk groups.[2] Appreciable evidence indicates that depression during this significant time period affects not only women's well-being but their children's health outcomes[3,4]. Despite its prevalence and pervasive effects, understanding of the biological basis of perinatal mood disorders is limited. Recent research has suggested the biological impact of air pollution exposure during pregnancy in the risk for postpartum depression[5,6]. However, the effect of pollution exposure on prenatal depression has not been examined; nor is there any information regarding differential effects of pollution by trimester of the pregnancy. In addition, potential mechanisms for the effects of pollution on depression are unknown. One proposed mechanism is that pollution may induce dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis which in turn mediates depressive symptoms and other CNS effects[7]. Studies have linked increased air pollution exposure to elevated serum levels of HPA axis hormones[8,9], implicating air pollutants as physiologic stressors that contribute to activation of the HPA axis. It is well documented that long-term activation of the stress response system increases the risk of depression[10,11].

In this study, we had 2 research aims:
1. To examine the association between pregnancy air pollution exposure and prenatal depression during the 3rd trimester
2. To determine if specific cortisol parameters mediate the relationship between air pollution exposure and maternal depression

Conceptual Framework



Methods & Materials

Women (n=50) were recruited in 2 obstetric clinics during their third trimester. They completed the Patient Health Questionnaire-9 to assess depression and provided salivary samples at 4 times during the day for 2 days. Four measures of cortisol were calculated from salivary assays: average cortisol levels, cortisol awakening response (CAR), diurnal cortisol slope (DCS), and area under the curve (AUC_G). We acquired data on particulate matter (PM2.5) within each woman's residential area from public records of the air quality control district. Structural equation modeling was used to analyze the aims.

Descriptive Results

Table 1. Characteristics of the Sample

Covariates	Unit or Category	Mean (IQR) or N (%)
Maternal age	years	33 (21-44)
Maternal education		
	Elementary school	3 (6%)
	High school or GED	8 (16%)
	Some college	10 (20%)
	2 year college degree	3 (6%)
	4 year college degree	9 (18%)
	Master's degree	9 (18%)
	Professional degree (e.g. MD)	5 (10%)
	PhD degree	3 (6%)
Maternal income		
	Less than $15,000	11(22%)
	$15,000-$20,999	5 (10%)
	$21,000-$30,999	6(12%)
	$31,000-$50,999	3 (6%)
	$51,000-$75,999	-
	$76,000-$100,999	-
	$101,000-$149,999	8(16%)
	$150,000+	18(34%)
Stress	Scores	7.3 (0-39)
Depression*	Scores	6.4 (1-20)
PM2.5 exposure		
1st trimester exposure	$\mu g/m^3$	8.8 (4.2-20.7)
2nd trimester exposure	$\mu g/m^3$	8.1 (4.2-21.1)
3rd trimester exposure	$\mu g/m^3$	7.4 (5-10.6)
Avg. pregnancy exposure	$\mu g/m^3$	8.1 (5.8-12.6)
Cortisol parameters		
2 day avg.		.26 (.03-.55)
CAR		.02 (-.3-.29)
AUC		184.66 (32.18-337.95)
DCS		.24 (-.28-.75)

Results Related to the Aims

Increased PM2.5 exposure during the 1st trimester was associated with more severe depressive symptoms (0.05, p=0.047), higher cortisol AUC_G (6.74, p=0.01), and higher average levels of cortisol (0.38, p=0.02) during the 3rd trimester. DCS was associated with increased 3rd trimester depressive symptoms in the 1st trimester exposure models (-.892, p=.046). The PM2.5 and DCS interaction term had a significant association to increased depressive symptoms (1T PM2.5: .195, p= .037) in regression adjusted models. The risk of depressive symptoms decreased when the DCS was stronger and more robust in the low exposure group (p=.002). Cortisol did not show a mediating relationship between PM2.5 exposure and depressive symptoms, but rather PM2.5 appeared to moderate the effect of cortisol on depression.

Tables and Figures

SEM Models

Table 2: SEM adjusted model of 1st trimester PM2.5 induced 3T depressive symptoms mediated by AUC cortisol

3T AUC Cortisol		
Model covariates	**Coef (95% CI)**	**P-value**
1st trimester PM2.5	6.737 (1.579 – 11.896)	.010
Stressors	-2.787 (-5.633 - .059)	.055
Age	-.341 (-4.2370 – 3.555)	.864

Depressive Symptoms		
Model Covariates	**Coef (95% CI)**	**P-value**
3T AUC Cortisol	-.001 (-.004 - .001)	.387
1st trimester PM2.5	.050 (.001 - .010)	.047
Stressors	.032 (005 - .059)	.018
Age	-.044 (-.080 - -.009)	.013

Table 3: SEM adjusted model of 1st trimester PM2.5 induced 3T depressive symptoms mediated by Avg. cortisol

Effects 3T avg. cortisol		
Model covariates	**Coef (95% CI)**	**P-value**
1st trimester PM2.5	.008 (.000 – .015)	.030
Stressors	-.002 (-.007 – .001)	.170
Age	.001 (-.004 – .007)	.658

Effects on depressive symptoms		
Model Covariates	**Coef (95% CI)**	**P-value**
3T Avg. Cortisol	-.880 (-2.658 - .898)	.332
1st trimester PM2.5	.050 (.001 - .099)	.045
Stressors	.033 (.007 - .059)	.014
Age	-.043 (-.078- -.008)	.016

Table 3: SEM adjusted model of 1st trimester PM2.5 induced 3T depressive symptoms mediated by DCS cortisol

3T Diurnal Slope Cortisol		
Model covariates	**Coef (95% CI)**	**P-value**
1st trimester PM2.5	.009 (-.006 – .024)	.259
Stressors	-.004 (-.012 – -.004)	.330

Depressive Symptoms		
Model Covariates	**Coef (95% CI)**	**P-value**
3T Avg. Cortisol	-.892 (-1.767 –-.018)	.046
1st trimester PM2.5	.052 (.003 - .100)	.037
Stressors	.042 (.017 - .067)	.001

Effect of Diurnal Slope on Depression for Women Exposed to High Versus How Levels of Pollution

Table 4: Depression adjusted regression model with DCS*1TPM2.5 Interaction

Effects on 3T depressive symptoms		
Model covariates	**Coef (95% CI)**	**P-value**
T1 PM2.5*DCS	.195 (.012 – .379)	.037
T1 PM2.5	-.000 (-.070 - .070)	.998
DCS	-2.921 (-5.028 - .813)	.008
Stressors	.034 (.006 – -.061)	.015

Figure 1: DCS and depression bivariate correlation model between high and low PM2.5 exposure groups

Pearson's Correlation

Group 1 (<8.8ug/m3), n=34 DCS & depression: r = -.528, p = .002

Group 2 (>=8.8ug/m3), n=16 DCS & depression: r = .041, p =.881

Conclusions

Exposure to pollution during the first trimester appears to have a significant impact on depression in the 3rd trimester, suggesting that effects of pollution on the brain may take time to develop or that women are particularly vulnerable to mood-related effects of pollution earlier in their pregnancy. Pregnant women with lower exposure to air pollution are more likely to have a 'normal' cortisol decline over the day (DCS) and a lower risk of developing depressive symptoms versus mothers with higher exposure to air pollution during the prenatal period. Further, air pollution may be contributing to blunted or dampened diurnal slopes in the higher PM2.5 exposed group. It has been proposed that the cortisol slope is controlled by our circadian system, suggesting potential dysregulation in circadian rhythms as well as the HPA axis itself as a result of pollution exposure. Our findings indicate that air pollution may be a modifiable risk factor for depression and HPA axis dysregulation. Policies and interventions to reduce pollution exposure could decrease women's vulnerability to depression during pregnancy. In addition, early depression assessment appears warranted for pregnant women in regions known for high pollution.

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