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The impact of maternal gestational stress on motor development in late childhood and adolescence: a longitudinal study

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Maternal Stress During Pregnancy Affects Long Term Offspring Motor Development

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Although the human brain and nervous system have shown a capacity for adaptivity, often referred to as plasticity, there is evidence that suggests insults to the developing central nervous system (CNS) in-utero can be long lasting and in some cases permanent (Pitcher, Henderson-Smart, & Robinson, 2006). Evidence of the impact the in-utero environment has on short and long term health outcomes are growing, evidenced by the rapidly growing field of research, the Developmental Origins of Health and Disease (DoHAD) (Barker, 2007). The development of the CNS is a complex process that begins at approximately 3 weeks gestation, however differentiation of embryo cells into specific tissues starts only a few days after fertilization (Brodal, 2010). Prenatally the process includes neural induction, proliferation, migration and differentiation. Pioneering work by Barker and colleagues (Barker, 2007) hypothesized that nutritional deficits in-utero led to structural and functional changes in the developing fetus, termed 'fetal programming', and coincide with an increased risk of disease in adult life. While this theory has been tested with outcomes such as coronary heart disease, stroke, hypertension and diabetes there is little research that has applied the hypothesis to outcomes of motor coordination.

Periods of critical importance during fetal development, have been previously reported (Barker, 1997; Nathanielsz, 1999). These windows of opportunity occur at times when cell proliferation and division in tissues, organs and systems occur at a rapid rate,

therefore different critical periods occur for different tissues. The timing of events which can influence fetal development are an important consideration in the study of in-utero environments, however there have been few longitudinal studies that have sought to pinpoint these critical windows of development in relation to motor development. Sensitive periods during gestation when the fetus may be more vulnerable to prenatal stress have been identified across most aspects of development (Ellman et al., 2008; Laplante et al., 2004; Van den Bergh & Marcoen, 2004). While the majority of these findings indicate stress in early pregnancy is of particular importance to offspring development some researchers (Huizink, Robles de Mina, Mulder, Visser, & Buitelaar, 2003; O'Connor, Heron, Golding, & Glover, 2003) have reported that stress in late pregnancy affects mental, emotional and behavioral development in infancy and early childhood. Pitcher, Henderson-Smart, and Robinson (Pitcher et al., 2006), reported that during the third trimester, the developing fetal brain may be more vulnerable to hypoxic and ischemic affronts. The cerebellar cortex, which develops mainly during late pregnancy, is important for the development of postural control, coordination, and motor skill function (Gramsbergen, 2003). While work with animal models has supported this role it is not fully understood how pregnancy stress may affect the developing human cerebellar cortex and whether the timing of this stress has long term neurological consequences. Long term functional deficits in motor development could also result from the increase in hormones such as cortisol (DiPietro, 2004), androgen (Kaiser & Sachser, 2009) or progesterone (Paris & Frye, 2011) which occur when the mother is stressed. Changes in these hormone levels are hypothesized to permanently affect the functioning of the hypothalamic-pituitary-adrenal (HPA) axis (Lazinski, Shea, & Steiner, 2008; Paris & Frye, 2011), limbic system, prefrontal cortex (Van den Bergh, Mulder, Mennes, & Glover, 2005) and Autonomic Nervous System (ANS) (Lazinski et al., 2008) in offspring. Although

not directly related to motor control, some of these, for example the limbic system which controls spatial memory and motivation, may affect motor functioning.

Changes in the structure and function of the developing fetal neurological system, due to maternal stress have been hypothesized to cause long term deficits in several developmental domains (Glover & O'Connor, 2006; 2003; Ruiz & Avant, 2005; Van den Bergh et al., 2005). Birth outcomes reportedly effected by maternal gestational stress include lower birth weight and gestational age, smaller head circumference and poorer neurological scores at birth (Glover & O'Connor, 2006). Previous research has revealed maternal gestation stress can also negatively impacts a range of health and developmental outcomes in infancy and early childhood (Monk, 2001; Ruiz & Avant, 2005; Talge, Neal, & Glover, 2007; Tegethoff, Greene, Olsen, Schaffner, & Meinlschmidt, 2011). These include cognitive (Buitelaar, Huizink, Mulder, de Medina, & Visser, 2003; Glover & O'Connor, 2006; Huizink et al., 2003; Laplante et al., 2004; Sandman, Davis, Buss, & Glynn, 2012), motor (Buitelaar et al., 2003; Huizink et al., 2003), language (Henrichs et al., 2011; Laplante et al., 2004), behavioral and emotional development (de Weerth, van Hees, & Buitelaar, 2003; Glover & O'Connor, 2006; O'Connor, Heron, Golding, Beveridge, & Glover, 2002; Robinson et al., 2008; Sandman et al., 2012) as well as physical and neuromuscular maturation (Ellman et al., 2008; Sandman et al., 2012). For example Buitelaar (2003) reported gestational stress to be predictive of lower motor development outcomes at 8 months and Huizink (2003) reported an average decline of 8 points on mental and motor development scales in infants born to mothers who recorded higher levels of the stress hormone cortisol.

While longitudinal studies have shown that maternal pregnancy stress affects behavioral, mental and cognitive development in middle childhood (Rodriguez & Bohlin, 2005; Van den Bergh & Marcoen, 2004) and into adolescence (Mennes, Van den Bergh, Lagae, & Stiers, 2009; Robinson et al., 2011) few studies have investigated the consequences

on motor development. Earlier work using animal models revealed reduced motor skills and balance in infant monkeys after repeated maternal stress (Schneider & Coe, 1993). Hands and colleagues (Hands, Kendall, Larkin, & Parker, 2009) examined whether a range of perinatal factors influenced human motor development and found that a high level of post natal maternal stress was related to the presence of mild motor delay in males at 10 years. Gestational stress was not reported as a contributing factor, however the variables were dichotomized, with a stressful pregnancy defined by the presence of 3 or more stressful events. In light of other findings regarding timing and number of stressors being pertinent to the effect on developmental outcomes (Davis & Sandman, 2010; Ellman et al., 2008; Robinson et al., 2011) further investigation of the available gestational stress data is warranted. The current study will examine how stressful events during early and late gestation, as well as total number of stressful events throughout pregnancy affect motor development outcomes at 10, 14 and 17 years.

Low motor competence has previously been linked to decreased short and long term mental and physical health outcomes (Cantell, Smyth, & Ahonen, 1994; Fitzpatrick & Watkinson, 2003; Schoemaker & Kalverboer, 1994; Skinner & Piek, 2001). While the body of evidence regarding the negative effects of lowered motor competence is growing there remains a paucity of research involving early risk factors for suboptimal neurological development during the antenatal, perinatal and neonatal stages.

Events which are believed to cause stress such as marital problems, financial issues, loss of a close family member or the accumulation of smaller daily hassles are most often used as stress markers (Huizink et al., 2003; Robinson et al., 2011; Whitehouse et al., 2010). The purpose of this paper is to investigate whether the number and timing of stressors experienced during pregnancy impacted long term motor development at 10, 14 and 17 years. We hypothesize that the experience of stressful events during pregnancy would negatively

impact offspring motor development, with later pregnancy stress playing a more important role in motor outcomes than earlier stress.

Methods

Participants

Participants ($N=2900$) were from the Western Australian Pregnancy Cohort (Raine) Study. The cohort were primarily Caucasian, from European descent (88.2%), and included mothers who identified as Aboriginal (2.4%), Chinese (4.4%), Indian (2.6%), Polynesian (0.9%) and Vietnamese (0.3%). Recruitment criteria included gestational age between 16-18 weeks, adequate English language skills to comprehend the study requirements, expected delivery at King Edward Memorial Hospital and for ease of future follow up of children, the desire to remain living in Western Australia. Mothers were recruited between 16-20 weeks gestation ($M=18$ weeks) from May 1989 to November 1991 at a rate of approximately 100 per month. Full cohort details and enrolment criteria have been published previously (Newnham, Evans, Michael, Stanley, & Landau, 1993). In total 2868 live births were recorded (Table 1) and questionnaire data including socioeconomic status and maternal health and psychosocial characteristics were collected from the mothers at 18 and 34 weeks gestation, with obstetric data collected throughout the antenatal, perinatal and neonatal periods. Physical data were collected at 10 ($M = 10.54$, $SD = 2.27$), 14 ($M = 14.02$, $SD = 2.33$) and 17 ($M = 16.99$, $SD = 2.97$) years from the offspring. A total of 989 children completed motor development testing at all three data collection phases, while 395 completed one data collection phase and 533 participated in two of the three follow ups. The participation rates for the active cohort (Table 1) were good at each follow up phase; 10 ($n = 1622$, 79%), 14 ($n = 1584$, 85%), 17 ($n = 1221$, 69%). There were no statistical differences in motor development outcome between those participants who were assessed at 10 years only ($M = 94.72$, $SD = 14.38$) and those who participated in all three data collection phases ($M = 94.35$, $SD = 14.12$).

Ethics clearances were obtained from the Human Research Ethics Committee at King Edward Memorial Hospital and the Princess Margaret Hospital for Children, Perth, Western Australia. Informed consent was obtained at enrolment and at each follow up from parents and/or guardians.

Predictor Variable

Maternal stress data were collected at 18 and 34 weeks gestation from the mothers using a 10-item questionnaire based on the Tennant and Andrews (1977) Life Stress Inventory. A yes/no format was used to ask if the mothers had experienced any of the listed stressful events, such as pregnancy problems, death of a close relative, death of a close friend, separation or divorce, marital problems, problems with children, involuntary job loss, partner's job loss (involuntary), money problems and residential move (Table 2). Another item labeled 'other' was available if the mother had experienced stress from an unlisted event or circumstance. The first questionnaire at 18 weeks asked if the mothers had experienced any of the listed stressors since becoming pregnant, while the questionnaire at 34 weeks asked if they had experienced the listed stressors in the last four months. This ensured stressors that occurred during the first questionnaire were not counted in the second questionnaire unless they were still occurring. For example moving house which is a one off event would only need to be included in one questionnaire while marital or financial problems which can be ongoing may have been included in both. To explore the impact of early and late stress two continuous variables were created to reflect the number of stressors experienced at both time points. Three groups were then created which categorized stress severity. This allowed for comparison to other published works which used similar methodology (O'Connor et al., 2003), including previous research using the Raine Study cohort (Robinson et al., 2011; Whitehouse et al., 2010). Each stressful event was weighted

equally and mothers were categorized as experiencing either no stress (NS), low stress (LS; <3 stressors) or high stress (HS; ≥ 3 stressors) throughout pregnancy.

Outcome measure

At 10, 14, and 17 years, offspring motor outcome was measured by the McCarron Assessment of Neuromuscular Development (MAND) (McCarron, 1997). The MAND comprises a battery of 10 items including a) hand strength b) finger-nose-finger placement c) jumping d) heel-toe walk e) standing on one foot f) beads in a box g) beads on a rod h) finger tapping i) nut and bolt j) rod slide. Raw scores are converted to scaled scores ($M = 10$, $SD = 3$). The scaled scores are summed and the total normalized to form a composite score, the Neuromuscular Development Index (NDI) ($M=100$, $SD=15$). The NDI can be used as a continuous outcome measure (Table 3), or a cutoff of <85 can be used to determine the presence of mild motor delay (Table 4) (Hands et al., 2009; McCarron, 1997). Test-retest reliability coefficients of the MAND tasks are reported by McCarron (McCarron, 1997) at 0.99 overall. A comparison of the MAND to two other highly utilized motor coordination tests revealed the MAND to be superior in detecting motor development problems in Australian children (Tan, Parker, & Larkin, 2001).

Covariates

The statistical models controlled for other variables known to influence motor development. These included maternal age, maternal smoking and alcohol consumption, percentage of expected birth weight, parity, child's sex, gestational age and family income. A categorical variable for maternal smoking was created with three groups; non-smokers, ≤ 10 cigarettes a day, and > 10 cigarettes a day. Maternal alcohol intake was classified as daily, several times a week, once a week or less, or never. Family income was dichotomised to

reflect a minimum income level ($< \$24000$ p.a. or $\geq \$24000$ p.a.) according to Australian Government guidelines at the time.

Statistical Analyses

Maternal and child variables that were related to motor development at 10, 14 and 17 years were identified using cross sectional analyses including chi-square tests, t-tests and univariate ANOVA models (general linear model - GLM) with Bonferroni post hoc correction. No interactions were found between child's sex and maternal stress group or any of the control variables, so results were not stratified by sex.

Linear mixed models were used to examine the effect of stress on motor development, accounting for the unbalanced nature of longitudinal data with repeated measures. The first model examined the severity of pregnancy stress on offspring motor development throughout the entire pregnancy, using the categorical variables of no stress, low stress (< 3 stressful events) and high stress (≥ 3 stressful events). The second model explored the difference in early and late pregnancy stress on motor development using continuous variables of stress calculated at 18 and 34 weeks gestation. Covariates that were not significantly related to motor development were not included in the final models.

Results

Group characteristics are reported in table 5. Mothers who experienced high stress throughout pregnancy (≥ 3 stressful events) were younger than those in either the low stress (< 3 stressful events) or no stress groups ($p = < 0.001$). More women in the high stress group were classified as having a low income ($p = < 0.001$) and they were more likely to smoke ($p = < 0.001$). Infants born to mothers in the high stress group had a lower gestation age ($p = < 0.001$). Money problems were the most commonly reported stressor with 28.1% of

participants at 18 weeks and 26.1% at 34 weeks stating they had experienced financial stress (Table 2). Pregnancy problems were the next most common stressor, followed by residential moves and marital issues. Problems with children were the fifth most common stressor, while other stressors were reported by between 1.7 – 5.4% of participants.

The first linear mixed model, adjusting for sex, gestation age, percentage of expected birth weight, maternal age, parity, maternal alcohol and smoking and family income revealed that number of stressful events and mean NDI were negatively related ($\beta = -1.197$, $p = 0.001$). The overall adjusted mean NDI for the no stress groups was significantly larger than the high stress group. Pairwise comparison revealed a significant difference between the no stress (98.91) and high stress (97.16) ($p = 0.017$) groups.

Of the potential confounding factors included in the analyses sex ($p = <0.001$), gestational age ($p = 0.001$), parity ($p = 0.040$), family income ($p = <0.001$) and maternal alcohol consumption ($p = 0.003$) were related to motor development. Males overall had higher NDI scores, while offspring with lower gestational ages had poorer NDI scores compared to their peers. First born children and those from families with incomes under the Australian Government threshold had lower motor development scores. Alcohol intake for those who were grouped as daily drinkers was negatively related to motor development.

The second model, investigating early and late pregnancy stress revealed that stressful events experienced in late pregnancy were negatively related with offspring motor development ($\beta = -0.541$, $p = 0.050$) while earlier stressful events had no significant impact. Covariates related to motor development scores in the second model included sex ($p = <0.001$), gestation age ($p = 0.001$), percentage of expected birth weight ($p = 0.042$), parity ($p = 0.020$), family income ($p = <0.001$) and maternal alcohol consumption ($p = 0.042$).

Cross sectional analyses revealed there were group differences in mean NDI at 10 years ($p = 0.034$), with Bonferroni post hoc results showing difference between the no stress and high stress groups ($p = 0.050$). At 14 years there was also a group difference ($p = 0.011$), with post hoc analyses showing difference between the no stress and high stress groups ($p = 0.009$). The highest group difference was seen at 17 years ($p = 0.001$) with post hoc results revealing differences between no stress and high stress ($p = 0.003$) and also low stress and high stress ($p = 0.010$) groups. The high stress group comprised more individuals whose NDI fell under the cutoff for mild motor delay at each year (Table 4). This difference was significant at 17 years ($p = 0.029$).

Discussion

The first linear mixed model, examining the impact of stressful events throughout pregnancy revealed support for the hypothesis that pregnancy stress would result in lower motor development scores in offspring. This was shown at ages 10, 14 and 17. The greatest difference in mean NDI was found between the no stress and high stress groups. Mothers who experienced three or more stressful events throughout their pregnancy had offspring with a lower motor competence than those who experienced none or less than three events. This may suggest an accumulative effect of stress on the developing fetal motor system, with small amounts of stress having a negligible effect and greater amounts having a negative effect. In contrast to our findings DiPietro and colleagues (DiPietro, Novak, Costigan, Atella, & Reusing, 2006) reported that non-specific maternal stress did not have a negative relation with overall child development at the age of two and motor development at this age was found to be positively impacted by higher levels of maternal stress. The smaller sample size (185) and restriction to low-risk, nonsmoking women over the age of 20 years may explain the difference in findings. Further to this the children measured in the previous study were much younger than the current study. Gramsbergen (2003) suggests that the underlying

neurobiological processes that contribute to motor development, including neurophysiological factors such as motor programming and sensory processing, continue to develop during a child's first 10 years. It is possible that the effects of maternal gestational stress on these processes may not be fully manifested until after these systems have fully developed.

The second linear mixed model, investigating the impact of early versus late stressful events confirmed that late pregnancy stress had a greater influence on motor development during late childhood and into adolescence than early pregnancy stress. The human neural system is one of the first systems to develop in utero, however disturbances of the developing cerebellar cortex, which occurs late in neuro-ontogeny, may be the key etiological factor for motor programming (Gramsbergen, 2003; Ivry, 2003). Growth of the cerebellar cortex occurs during the third trimester and includes a rapid increase in granule cells and the creation of neural pathways, which will eventually assist in adjustments to muscle tone, control of movement and posture and the learning of physical tasks and motor skills (Gramsbergen, 2003). While several previous studies highlight the importance of early pregnancy stress on cognitive, (Davis & Sandman, 2010; Laplante et al., 2004; Sandman et al., 2012) language (Laplante et al., 2004) and mental and behavioural (Van den Bergh & Marcoen, 2004) development, other researchers have found mid to late pregnancy stress affected early motor development (Huizink et al., 2003) and behavioral / emotional problems (O'Connor et al., 2003). Our findings support the theory of later pregnancy stress having a greater influence specifically on long term motor development and further research into the impact of this on the developing cerebellar cortex may help to further our understanding of how this occurs.

Alternatively the effect of maternal gestational stress on other areas of neurological development may account for the lower motor development scores. Changes in levels of hormones such as cortisol (DiPietro, 2004), androgen (Kaiser & Sachser, 2009) or

progesterone (Paris & Frye, 2011) are hypothesized to permanently affect the functioning of the limbic system (Murmu et al., 2006). Changes in neuron development within the limbic system due to maternal gestational stress have been observed in rat models (Murmu et al., 2006) however whether these changes affect motor development in humans is unknown.

Cross sectional analyses showed group differences at 10 ($p = 0.034$), 14 ($p = 0.011$) and 17 ($p = 0.001$) years. This finding was unexpected as no previous research has reported that the negative relationship between maternal gestational stress and offspring motor development becomes stronger with age, however the continued growth of the neurological systems throughout the first decade (Gramsbergen, 2003) may explain why the full impact on these systems is not evident until after puberty.

Strengths

A large population based sample and the collection of various potential confounders allowed for a stringent and robust analysis of the effect of pregnancy stress on motor development in late childhood and adolescence. As previously reported (Robinson et al., 2011), the inclusion of pregnancy concerns in the questionnaire allowed the mothers to include stressors that may have otherwise been overlooked, as pregnancy and impending birth, as well as related health problems, can be a cause of stress themselves. The use of two questionnaires to collect stress data allowed earlier and later stressors to be compared and the impact of timing to be analyzed. The MAND (McCarron, 1997) is a reliable and accurate measure of motor development among Australian children (Tan et al., 2001).

Limitations

While stressful events are commonly used as a measure of stress we acknowledge that this does not consider an individual's resilience which can ameliorate the level at which stressful events may impact them psychologically. The longitudinal nature of the study did

not allow for further measures regarding maternal resilience or perceived severity of stress. Other environmental and lifestyle factors have previously been linked to motor development throughout infancy and childhood however controlling for these factors was not within the scope of this study. The extensive and high quality antenatal data available allowed thorough and robust analyses of the factors contributing to motor development from this time period.

Conclusion

We found support for the hypothesis that stress during pregnancy contributed to a poorer motor development outcome in the long term. Glover (2014) has recently stated the emotional care of pregnant women is an often overlooked aspect of obstetric practice. With evidence for the importance of maternal emotional and mental health on a wide range of developmental and health outcomes for both mother and child, future programs aimed at early detection and reduction of maternal stress, may help improve offspring outcomes. Currently screening for post natal depression with user-friendly questionnaires occurs in most antenatal clinics in Australia. This cost effective model could be used to screen for maternal stress throughout pregnancy as part of regular clinic visits. Previous research has highlighted the importance of exercise in the reduction of stress, improvement of mood and enhanced mental health outcomes (Fox, Boutcher, Faulkner, & Biddle, 2000). Da Costa and colleagues (2003) reported women who exercised during pregnancy had significantly better mental health markers, including less state anxiety and less pregnancy-specific stress. Exercise presents a low-cost yet effective method of ensuring healthy women experience optimal mental health during pregnancy. Antenatal clinics provide an ideal arena for pregnant women to be informed of the benefits of exercise, in particular if they are experiencing a stressful pregnancy.

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Table 1.
Available Data from each follow up of the Raine Study

Year	Active	MAND	Deferred	Lost	Withdrawn	Deceased	Total
Birth	2868		-	-	-	-	2868
10	2047	1622	281	162	348	30	2868
14	1860	1584	357	207	412	32	2868
16	1754	1221	414	184	480	36	2868

Table 2
Type and frequency of stressful events

Stressor	18 Weeks (N = 2804)		34 Weeks (N = 2580)	
	n	%	n	%
Money problems	789	28.1	665	25.7
Pregnancy problems	733	26.1	511	19.8
Residential move	455	16.2	466	18
Marital Problems	247	8.8	184	7.1
Problems with your children	177	6.3	164	6.3
Relationship problems	151	5.4	140	5.4
Death of a relative	149	5.3	138	5.3
Your partners job loss (not voluntary)	136	4.8	136	5.3
Separation or divorce	114	4	77	2.9
Your own job loss (not voluntary)	85	3	36	1.4
Death of a close friend	56	1.9	43	1.6

Table 3

MAND scores at 10, 14 and 17 years according to pregnancy groups

NDI	N	No Stress 0 Stressors		N	Low Stress < 3 Stressors		N	High Stress ≥ 3 Stressors		Group Difference
		M	SD		M	SD		M	SD	
10yrs	352	95.36 ^a	13.55	616	94.74	14.21	542	93.09 ^a	13.81	0.034*
14yrs	336	101.19 ^a	18.32	612	99.48	17.62	524	97.54 ^a	17.21	0.011*
17yrs	260	98.60 ^a	17.51	466	97.53 ^b	17.36	423	94.13 ^{ab}	16.52	0.001*

* = significant group difference at the $p = 0.05$ level

Table 4

Prevalence of mild motor delay at 10, 14 and 17 years according to pregnancy groups

NDI		No Stress 0 Stress Events		Low Stress < 3 Stress Events		High Stress ≥ 3 Stress Events		Group Difference
		>85	≤85	>85	≤85	>85	≤85	
10yrs	N	272 (77.3%)	80 (22.7%)	467 (75.8%)	149 (24.2%)	386 (71.2%)	156 (28.8%)	0.082
14yrs	N	265 (78.9%)	71 (21.1%)	474 (77.5%)	138 (22.5%)	391 (74.6%)	133 (25.4%)	0.314
17yrs	N	192 (73.8%)	68 (26.2%)	341 (73.2%)	125 (26.8%)	279 (66.0%)	144 (34.0%)	0.029*

* = significant group difference at the $p = 0.05$ level

Table 5

Descriptive statistics of cohort according to pregnancy groups

	No Stress			Low Stress			High Stress			Group Diff
	0 Stress Events			<3 Stress Events			≥3 Stress Events			
Continuous Variables	N	M	(sd)	N	M	(sd)	N	M	(sd)	
Maternal Age (yrs)	567	28.67	(5.80)	1035	28.27	(5.82)	1014	26.48	(5.90)	<0.001
% Expected Birth Wt	567	97.48	(12.33)	1032	97.63	(12.44)	1007	96.63	(13.24)	0.173
Gestational Age (wks)	567	39.06	(1.70)	1034	38.91	(1.78)	1010	38.66	(2.21)	<0.001
Categorical Variables	N	n	(%)	N	n	%	N	N	(%)	
Smoking	567			1035			1014			<0.001
None		456	(80.4)		795	(76.8)		671	(66.2)	
≤10/day		67	(11.8)		156	(15.1)		172	(17.0)	
>10/day		44	(7.8)		84	(8.1)		171	(16.9)	
Alcohol	567			1034			1012			.661
None		309	(54.5)		576	(55.7)		533	(52.7)	
Once a wk or less		217	(38.3)		402	(38.9)		414	(40.9)	
Several times a wk		36	(6.3)		48	(4.6)		58	(5.7)	
Daily		5	(0.9)		8	(0.8)		7	(0.7)	
Sex	567			1035			1014			0.792
Males		295	(52.0)		520	(50.2)		520	(50.8)	
Females		272	(48.0)		515	(49.8)		499	(49.2)	
Low Income	528			976			947			<0.001
No		414	(78.4)		710	(72.7)		551	(58.2)	
Yes		114	(21.6)		266	(27.3)		396	(41.8)	
Parity	567			1033			1008			0.686
0		266	(46.9)		505	(48.9)		477	(47.3)	
1+		301	(53.1)		528	(51.1)		531	(52.7)	

Note. *p* values are for comparison between three groups according to ANOVA (continuous variables) and chi-squared analyses (categorical variables).