CHILDREN BORN SMALL FOR GESTATIONAL AGE ARE NOT AT SPECIAL RISK FOR PRESCHOOL EMOTION AND BEHAVIOUR PROBLEMS

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ABSTRACT

Despite the wealth of literature examining long term outcomes of preterm low birthweight children, few studies have directly assessed the developmental impact of being born full term but small for gestational age (SGA). We aim to determine whether (i) being SGA increases preschool behavioural problems and (ii) other risk factors operate differently in SGA and appropriate for gestational age (AGA) controls. 550 New Zealand European mothers and their 3.5 year old children participated in this study. All children were born at full term (> 37 weeks' gestation) and approximately half were SGA ($\leq$ sex specific 10th percentile for gestation) the remainder were AGA controls. Extensive data were collected at the child's birth, 1 year and 3.5 years. Behavioural problems were measured when children were 3.5 years, using the Strengths and Difficulties Questionnaire (SDQ). Multiple regression analyses were used to examine the associations between risk factors and behavioural problems; statistical weighting was used for analyses of the total study group. There was no significant difference in behavioural problems between SGA and AGA groups. In the total sample the significant predictors of behavioural problems included: mothers' school leaving age; smoking during pregnancy; maternal alcohol use during pregnancy; and absence of the father. Predictors of behavioural problems were found to be the same for SGA and AGA groups. These results do not support the view that SGA is a risk for behavioural preschool difficulties or that SGA children are sensitized to risks known to be associated with such difficulties in the preschool years.

Key words: *preschool children, emotion, behaviour, risk factors, strengths and difficulties questionnaire, small for gestational age*

Abbreviations: SDQ (Strengths and Difficulties Questionnaire), SGA (small for gestational age), AGA (appropriate for gestational age), ABC (Auckland Birthweight Collaborative)

INTRODUCTION

Behaviour and emotional problems are of particular concern in the preschool age group as once established they often persist throughout childhood and adolescence and, in some cases, continue into young adulthood (1, 2). The prevalence rates of these problems vary between 10% and 20% (3, 4) with between study differences in estimates due to the limitations of diagnostic tools used to classify problem behaviour. Gathering prevalence estimates for this age group is complicated as the preschool developmental period is one of the most challenging in terms of defining and measuring psychiatric symptomatology. On the whole, the literature suggests that externalising problems (e.g., hyperactivity, impulsivity, and oppositional defiant symptoms) are more prevalent in males (5, 6), and internalising problems (e.g., phobias, anxiety and depression) are more prevalent in females (7).

Childhood emotional and behavioural problems, particularly in the preschool years, are multi-factorial in determination. Socio-demographic factors include young maternal age (8), limited parental education (9), low socio-economic status (4) and low levels of social support (10). Furthermore, perinatal exposures to nicotine (11), alcohol (12) and stress (13) are also important. Some of the strongest support is for prematurity (14-19) and/or being small for gestational age (SGA:14, 19, 20, 21) both of which have been related to the onset of childhood disorders such as ADHD (22, 23), anxiety and depression (24) and learning difficulties (25). Understanding these particular associations is complicated as both are linked to (i) a suboptimal prenatal environment (which is often as a result of foetal exposure to one or more of the factors previously described), and (ii) prematurity and SGA are often combined in studies of the risks associated with low birthweight. The hypothesis that SGA effects should operate independently of prematurity and therefore constitute a distinct risk factor is based on the concept of intra uterine growth restriction (IUGR). One possible cause

of IUGR is when a foetus does not receive enough oxygen or nutrients during pregnancy, and as a consequence their overall body and organ growth is limited. Many factors have been found to be associated with SGA and/or IUGR including; low maternal pre pregnancy weight (26, 27), maternal smoking during pregnancy (28, 29) and maternal hypertension (29). In addition, genetics have also been found to be involved in both IUGR and being born SGA (for a review see 30). It is unclear exactly how poor intra uterine growth affects the foetal brain to cause psychopathology. One possibility is that there is a restriction in blood flow through the placenta which results in IUGR. This in turn causes the foetus to receive less oxygen than would normally be expected, increasing the risks to the foetus during pregnancy, delivery, and afterwards. Furthermore, such foetal hypoxia can result in damage to catecholamine receptors in the brain (e.g., dopamine; 31, 32).

To date, only one study has investigated the behavioural development of *full term* SGA (33). (The definition of *full term* is important as it rules out the possibility that the child's birthweight is a consequence of prematurity factors). Sommerfelt and colleagues (33) studied a relatively large group of SGA (n=318) and AGA (n=307) 5 year old children and found behavioural problems were not more common among the SGA children. Furthermore, being born SGA was not found to make an individual more sensitive to the negative impact of common risk factors (33). The present study extends this research in similar size sample of full term SGA and AGA preschool children in a study with a longitudinal cohort design. This has allowed for the collection of a number of risk factors at different points throughout the child's development. Here we aim to: (1) to determine whether SGA children are at increased risk for emotional and behavioural problems compared with AGA children; and (2) to examine whether the risk profile for SGA children is different to that of AGA children, and

if not we also aim to examine the prevalence and risk profile for the total study group using statistical weighting to allow for the complex weighting design.

METHODS

The Auckland Birthweight Collaborative (ABC) study is a longitudinal cohort with disproportionate sampling. The methodology for phases one to three of the ABC study has previously been described in detail (29). Participants are mothers and their singleton infants were born full term in the Auckland and Waitamata Healthcare regions, between 16 October 1995 and 30 November 1997. Approximately half of the infants were SGA with birthweights equal to or below the sex-specific tenth percentile for gestation, and the remainder were a random sample of infants born AGA (34). Infants were excluded from the study if they were born outside a designated study region, were from multiple births or had congenital abnormalities likely to affect growth or development.

Data collection has occurred in three phases: phase one, birth; phase two, 1 year; and phase three, 3.5 years. Extensive psychological, developmental, social and physical data were collected for children and their families at all phases. Due to the differential in response rates from ethnic groups at earlier phases, this study has been restricted to New Zealand European mothers and their infants. There were 871 New Zealand European mothers and infants enrolled at birth who were eligible to take part at 3.5 years; of these 550 (63%) were successfully followed up when children were aged 3.5 years. Complete SDQ scores were available for 538 children (SGA = 225, AGA = 313).

Factors associated with attrition at phase three have previously been reported in detail (35). In summary, respondents were more likely to be older, married, of high socio-economic status, have tertiary education, and be non-smokers. Respondents and non respondents did not differ in gestational age, birthweight, infant sex, parity and levels of family social support or maternal stress.

Outcome Measure

Child behaviour was measured at phase three using the parent format of the Strengths and Difficulties Questionnaire (SDQ; 36). SDQ is a 25 item scale which asks about both positive and negative emotional and behavioural attributes on 5 subscales - conduct, emotion, hyperactivity-inattention, peer group relationships and pro-social behaviour. Each item is scored on a 3 point Likert scale of 0 = '*not true*', 1 = '*somewhat true*' and 2 = '*certainly true*'. The total scores for each subscale are calculated by summing scores on items relevant to a particular problem, for example the hyperactivity-inattention subscale consists of items such as '*restless, overactive, cannot stay still for long*' (range = 0 – 10). The SDQ also provides a total difficulties score (range = 0 – 40) which is calculated by summing all subscale scores except pro-social (as the authors suggest the presence of pro-social behaviours is considered to be conceptually different to the absence of other behaviours). The SDQ has a good test-retest stability and the internal consistencies of the subscales range from 0.62 to 0.75 (37). Cut-off points used in this study to define 'normal', 'borderline' and 'abnormal' scores were taken from UK norms published on the SDQ website (www.sdqinfo.com). This study focused on the prediction of the total behavioural difficulties subscale because of the greater reliability of the total difficulties scale compared with the individual subscales (which are defined by 5 items). In this study children were defined as having an abnormal behavioural total difficulties score if they had a total difficulties score in the abnormal or borderline range. At phase three of the study SDQ data were collected for 538 children.

Measurement of risk factors

(i) Maternal Perceived Stress. This was measured at phases one and three using the 10-item Perceived Stress Scale (38). This scale was designed to measure the degree to which

situations in life are appraised as stressful. We created a categorical variable using a 75th percentile cut off, scores that fell above this were considered as high.

(ii) Social support. The Family Support Scale (39) measures different sources of support for families rearing a young children. This variable was examined categorically with low levels of social support defined by a total score that was less than or equal to the 25th percentile of scores in this study.

(iii) Parental smoking and maternal alcohol use during pregnancy. Smoking data were collected at birth and coded as a dichotomous variable ('yes' or 'no') for mothers who reported having smoked the year before pregnancy and fathers who smoked up to the time of the child's birth. Maternal alcohol consumption during pregnancy was recorded at the time of the child's birth and coded as '*no alcohol*', '*1 - 7 units of alcohol per week*', and '*>7 units of alcohol per week*'.

(iv) Child activity level. At phase three, parents were asked if their child was more active, less active or had the same levels of activity compared with other children of the same age. The '*increased activity level*' and '*same activity level*' categories were combined as there were no significant differences in risk of emotional and behavioural difficulties between the groups.

(v) Demographic factors. Data on a child's sex, parental marital status, maternal school leaving age and maternal age at the child's birth was collected at the time of the child's birth (phase one).

Ethical approval was obtained from the North Health Regional Ethics Committee.

Statistical Analyses

The difference in prevalence of emotional and behavioural problems between SGA and AGA groups was examined using chi-square. The association of problems with potential risk factors was studied at all phases of the study using simple and multiple regression analyses. In order to examine whether the association between a particular risk factor and behavioural problems differed between SGA and AGA groups, an interaction term was included in the analyses. Evidence of a significant interaction was determined at a 5% level.

Odds ratios (OR) from simple and multiple logistic regressions are presented for each category of a variable in relation to a reference category. Chi-square values and *p*-values for the overall effect of variables in the model are also given.

Multiple regression analyses adjusted for the potentially confounding effects of a child's sex, parental marital status, maternal school leaving age and maternal age at the child's birth. These variables were selected as there were 'a priori' reasons to expect that they would be associated with the occurrence of emotional and/or behavioural problems. Variables identified from previous studies as likely to be associated with the occurrence of behavioural problems in children that were found to be significant at a 10% level in simple regression analyses, were added to this base model in groups according to the data collection phase. Firstly, variables measured at the time of the child's birth were added to the model; those that continued to be significant at a 5% level remained in the model, however, those that were no longer significant were removed from the model. Following this, we used the same approach when adding variables that were collected when children were age 1 and 3.5 years to the model. The final multiple regression model provides an odds ratio for each variable which adjusts for the effects of all other variables in the model. An overall *p*-value of 0.05 or less was considered to be evidence of statistical significance.

Where estimates have been calculated for the total group, weighted logistic regression was used. Analyses were calculated in SAS v9.1 (SAS 2004).

RESULTS

No significant difference in the rate of emotional and behavioural problems between SGA and AGA groups was found (SGA= 17.8%, AGA=17.9%; ($\chi^2(1) = 1.02, p=0.31$). In the total weighted sample, the prevalence of an abnormal total difficulties score was 18.9%.

Table 1 presents the unadjusted and adjusted regression results for the total weighted sample. Variables of interest that met the inclusion criteria ($p>0.10$) in unadjusted analyses and continued to be predictive ($p>0.05$) of behavioural problems in adjusted analyses included: current maternal stress (when the child was age 3.5 years), maternal smoking prior to pregnancy, paternal smoking at the time of pregnancy, maternal alcohol use in the first month of pregnancy, whether or not the child's biological father was currently living in the family home (when the child is age 3.5 years) and child activity level at age 3.5 years.

Table 2 reports the frequencies of abnormal SDQ scores for the SGA and AGA groups separately with interactions between the key risk variables and SGA/AGA status. Given that no significant interactions were present, (meaning that the association of risk factors with behavioural problems were not found to be different for the SGA and AGA groups), all subsequent analyses are presented for the total weighted sample.

Mothers who left school when they were younger than age 16 years were found to be over four times more likely to have a child with an abnormal total difficulties score at age 3.5 years (OR=4.42, 95% CI = 1.53 – 12.76) than those mothers who left school at 16 years of age or older. In an adjusted analysis, maternal stress when the child was aged 3.5 years continued to be predictive of abnormal total difficulties, yet maternal social support at 3.5 years no longer remained statistically significant. Maternal stress and social support measured at the time of the child's birth were not found to be significantly associated with an abnormal

total difficulties score ($p>0.05$), however maternal social support was found to be of borderline significance ($p=0.06$).

Children of mothers who reported having smoked regularly prior to pregnancy were found to be twice as likely for their child to have an abnormal total difficulties score at age 3.5 years (OR=2.01, 95% CI = 1.06 – 4.15) than those mothers who did not smoke prior to pregnancy. Furthermore, children of fathers who smoked at the time of their birth were also found to be more than twice as likely to have an abnormal total difficulties score at age 3.5 years than children whose fathers did not smoke at this time (OR=2.21, 95% CI = 1.04 – 4.15). Mothers who consumed more than 7 units of alcohol per week in the first month of pregnancy were found to be over six times more likely to have a child with an abnormal total difficulties score 3.5 years later (OR=6.13, 95% CI = 1.73 – 21.65).

Children with high activity levels at age 3.5 years were over twice as likely to have an abnormal total difficulties score than children whose activity levels were normal to low (OR=2.13, 95% CI = 1.06 – 4.31). Finally, children who did not live with their biological father when they were aged 3.5 years were found to be three times more likely to have behavioural problems compared with children whose biological father lived in the family home (OR = 3.11, 95% CI = 1.09 – 8.89).

DISCUSSION

The current findings replicated the Sommerfelt et al., paper (33) in (1) finding no significant difference in emotional and behavioural problems between SGA (17.8%) and AGA (17.9%) children, suggesting that SGA is not associated with an increased risk of behavioural problems in the preschool years; and (2) the risk profile for SGA children was not found to be different to that of AGA children. Although we did establish independent associations for a range of prenatal biological factors and postnatal factors, these operated in a similar way in both groups – (i.e. SGA did not sensitize children to these risk factors). This suggests that previous findings showing a link between SGA and behavioural problems may be the result of prematurity or other factors with which SGA is confounded and which have not always been well controlled for statistically.

Studies that have highlighted differences in the rates of emotional and/or behavioural problems between SGA and AGA groups have focused mainly on infants (40) and adolescents (41), with few examining the developmental period between these age groups (i.e., preschool and school age). The developmental period from preschool through to school age is an important time as this is a time when there are a number of critical periods for the development of key skills and abilities, such as language, emotional regulation, self regulation, social adjustment and moral realism. Secondly, the majority of studies in this area have been limited by small sample sizes and the use of non standardised definitions for SGA status. Here, we employed a relatively large sample of SGA children defined as such using the tenth percentile of sex specific birthweight percentiles by gestational age based on the New Zealand population. Finally, there are few studies that employ population based designs.

The current study findings refute the idea that poor IUGR growth affects the foetal brain and in turn causes psychopathology up to the preschool years. It is possible that subtle pathophysiologic alterations during brain development may result from factors that affect growth in utero and as the brain develops and matures over the course of childhood, such alterations could affect child behaviour.

Of the factors shown to be important for the whole sample there were a number of striking findings. In line with other studies in the literature we found that children of mothers who had limited education were more likely to have behavioural difficulties (8, 9). Young mothers encounter greater risks themselves and for their children as they can be less affectionate and nurturing than older mothers and less likely to stimulate their children verbally and cognitively (8). Furthermore, these mothers are often of low socio-economic status and are therefore more likely to have poor nutrition and lack social support (42). Callaghan (1999) suggests that limited maternal education, regardless of age, is a risk factor for child behavioural problems, as it directly affects the quality of parenting. For example, maternal 'cognitive readiness' for parenting (defined as knowledge of child development, parenting style preferences and parenting attitudes) has been found to directly impact a child's tendency to have behavioural problems (43). Despite this, it has been suggested that maternal age and education level may not directly cause children's behavioural problems, as the disadvantage of children born to younger mothers has been shown to be reduced when the effects of maternal family background are controlled (44). Although measures of maternal family background were not explicitly collected in this study, the association between young maternal age and limited education on preschool behavioural difficulties remained, after controlling for a number of factors related to family socio-demography.

It is not surprising that mothers with high stress levels when their children were aged 3.5 years were found to be more likely for their child to have emotional and/or behavioural problems at this age - compared with mothers who had normal to low levels of stress at this time. The continued adverse effects of parental stress on child psychopathology have been documented previously in the literature (45-48). It is possible that other adverse circumstances that have been found to contribute to maternal stress (e.g., socioeconomic disadvantage (48), parental psychopathology (49), poor parent – child relationship (50) and family discord (51), can also contribute to child psychopathology: therefore determining the underlying cause can become difficult. Nonetheless, these results further support the notion that mothers who are stressed are at greater risk of having children who become stressed, discontent and insecure (45).

Here, we also report an association between child behavioural problems and parental marital status. Children who were born to single mothers were more likely to have behavioural problems at 3.5 years of age than children of married parents. This association did not remain statistically significant after controlling for the effects of additional socio-demographic factors (e.g., maternal age and education). Nonetheless, this finding supports the view that single mothers are likely to also experience financial difficulties, a lack of self esteem and low levels of social support and that such difficulties can lead to a less conducive environment for their children to develop (10).

The findings of this study emphasize the importance of paternal factors on preschool behavioural development; in particular the absence of a child's biological father from the home environment in the preschool years. Paternal absence has long been associated with adverse child behavioural outcomes, and factors such as economic stressors related to single parenthood, perceptions of abandonment or guilt by the child, and continuing marital conflict

have been argued to be contributory factors (52). A recent study, however, reported that the effects of being raised by biological parents, particularly fathers, depend upon the *quality* of care parents can provide, and in situations where fathers demonstrated antisocial behaviour, it was more beneficial to the child's behavioural development to live in single-parent families than to live with both parents (53).

We have found that children born to mothers who reported consuming more than seven units of alcohol per week in the first month of pregnancy were more likely to have behavioural difficulties at age 3.5 years than mothers who consumed less than seven units of alcohol. This finding remained statistically significant after controlling for the effects of confounding variables (including maternal smoking) in the final regression model. Children who are prenatally exposed to the effects of alcohol are at an increased risk for psychiatric disorders in later childhood (12) and are more likely to be rated as hyperactive, disruptive, impulsive or delinquent (54). Adverse effects of prenatal alcohol exposure on child behaviour in the school aged years have been reported to be evident even at low levels of exposure (i.e., one alcoholic beverage a week), and have showed dose-response effects (12). A number of physiological, environmental, and genetic pathways that link maternal substance use to children's behavioural outcomes have been put forward (55). Firstly, maternal substance use during pregnancy may affect a developing child's emotional and behavioural outcomes through teratogenic processes. Secondly, it is possible that maternal substance use could disrupt the family home environment or parenting in a way that leads to the development of a child's behavioural problems. Finally, mothers and their children may share a genetic vulnerability to substance use and other types of problem behaviours. These three pathways are not mutually exclusive; they are likely to overlap (55). Establishing a causal relationship between factors such as maternal alcohol use or smoking during pregnancy and later

behavioural problems in offspring is not a straightforward task. An observed positive association may not be causal as it is possible that unobserved factors that are correlated with maternal alcohol use and smoking and a child's emotional or behavioural outcomes are present. For example, if a mother suffers from chronic anxiety or depression, this may lead both to maternal substance use and to emotional or behavioural problems in her child. It is therefore important to interpret these results with caution, as this study did not control for factors such as maternal psychopathology.

This study also found an association between paternal smoking and child behavioural problems. This association remained strong after controlling for the effects of covariates such as maternal smoking and other socio demographic factors; however, it remains difficult to untangle the possible biological and socio-demographic mechanisms that might explain this relationship. As Weissman and colleagues suggest, if parental smoking was a marker for genetically driven behavioural problems in children, studies would have found that both maternal and paternal smoking contribute equally to adverse outcomes in children and this has not been the case (11). The majority of studies have found that maternal smoking is more strongly associated with adverse developmental outcomes than paternal smoking with the exception of one study, which has recently reported that paternal smoking (and not maternal smoking) had a negative effect on attentional control in children with ADHD. This effect appeared to be mediated by genetic risk factors (56). It is possible that the significant effects of paternal smoking at the time of the child's birth reported here are related to either personality or psychosocial factors associated with fathers / men who smoke. For example, there is evidence that cigarette smoking is over-represented in men who are arrested for domestic violence, and that violent men who smoke cigarettes are more likely to abuse other substances than those who do not smoke (57). This group of men are also more likely to

experience psychopathology, impulsivity, anger and criminality (58). It may be that nicotine dependence can serve as a marker for these other characteristics and that the significant association between paternal smoking and child emotional and behavioural problems found in this study provides evidence of a genetic transmission of psychopathology.

Findings from this study support previous evidence that sex differences in problem behaviours are not as marked in the preschool years as they are reported to be for children of older ages (59), despite evidence to suggest that sex differences in problem behaviour in the preschool years are apparent (60).

The present study has a number of strengths. It contains a relatively large number of participants in the total study population, including a good number of SGA children. This has allowed for investigations of the differences in risk factors between the two groups. The ABC study dataset also includes a wide range of variables (including key biological and socio-demographic determinants) that can be examined in relation to the occurrence of emotional and behavioural problems in childhood. Despite this, a potential limitation of this study is that the sample consisted predominantly of well-educated, middle and high socioeconomic status, New Zealand European mothers and their children. Therefore, the results may not be generalisable to non-European ethnic groups. Furthermore, the SDQ cannot be considered a full assessment of a child's emotional and behavioural difficulties in the preschool years. Future research should include multiple methods of assessment (such as observational assessments of the child and the parent–child relationship) to ensure that accurate estimates of early problems are obtained.

These results support the hypothesis that there are diverse pathways through which factors such as young parenthood, limited maternal education and single parenthood

influence subsequent child behaviour (61). We found that factors associated with preschool behavioural problems are related more to the social and lifestyle context of the family and less to characteristics that relate directly to the child, such as birthweight. It has not been possible to attribute complete causality to one environmental risk factor or another from the current findings, nor are we able to explain why some children appear to be vulnerable to adversity while others are resilient. One possible method of investigating these issues further is by looking at the role of gene – environment interactions, which would allow the examination of effects of specific prenatal influences (such as birthweight) together with genetic and/or postnatal environmental factors on the development of behavioural problems later in life (23).

Clinical Implications

It is important to understand the factors that place children at risk of significant mental health problems when planning prevention and early intervention strategies. One of the most significant protective factors in preventing the development of a mental health problem in childhood is socioeconomic security. Despite evidence suggesting that reducing socioeconomic disadvantage has no effect on child psychiatric morbidity (62), this study recommends that continued efforts to reduce socio-economic disadvantage could potentially provide the right ‘social context’ for the development of policies and programs that are targeted towards reducing psychiatric disorders in young children. The findings of this study highlight the need for routine screening in paediatric primary care settings to identify preschool children and families who may benefit from early intervention services and to prevent continued difficulties in later childhood. Finally, it is critical to increase awareness about early-emerging psychopathology among professionals who are working with parents of very young children in order to promote early identification.

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Table I. Frequencies, simple and adjusted odds ratios for variables measured at a child's birth and age 3.5 years associated with preschool behavioural problems in the total study group.

	Abnormal		Normal		Odds Ratio		(95% CI)		† Adjusted	(95% CI)
	N = 96		N = 442						Odds Ratio	
A Child's Sex (missing = 0)					$\chi^2_{(1)} = 1.17, p=0.28$			$\chi^2_{(1)} = 2.48, p=0.11$		
Female	43	(15%)	239	(85%)	1.00				1.00	
Male	53	(21%)	203	(79%)	1.38		(0.77 – 2.46)		1.70 (0.88 – 3.29)	
Maternal School Leaving Age (missing = 15)					$\chi^2_{(2)} = 16.07, p=0.0003$			$\chi^2_{(2)} = 7.73, p=0.02$		
< 16 years	19	(36%)	27	(64%)	6.05		(2.38 – 15.35)		4.42 (1.53 – 12.76)	
= 16 years	34	(22%)	115	(77%)	2.20		(1.15 – 4.22)		1.74 (0.82 – 3.72)	
> 16 years	42	(12%)	298	(88%)	1.00				1.00	
Maternal Age at Birth of Child (missing = 19)					$\chi^2_{(3)} = 19.07, p=0.0003$			$\chi^2_{(3)} = 4.72, p=0.19$		
<20 years	15	(24%)	30	(77%)	7.03		(2.42 – 20.38)		3.83 (0.88 – 16.56)	
20 – 24 years	29	(23%)	97	(77%)	2.21		(0.96 – 5.08)		1.70 (0.69 – 4.14)	
25 – 29 years	33	(15%)	188	(85%)	0.97		(0.43 – 2.18)		1.02 (0.39 – 2.61)	
30+ years	17	(12%)	122	(88%)	1.00				1.00	
Marital Status (missing = 12)					$\chi^2_{(2)} = 13.50, p=0.001$			$\chi^2_{(2)} = 2.02, p=0.36$		
Married	57	(14%)	359	(86%)	1.00				1.00	
De Facto	27	(29%)	66	(71%)	2.69		(1.37 – 5.31)		0.54 (0.11 – 2.76)	
Single	12	(41%)	17	(59%)	5.55		(1.62 – 19.04)		1.50 (0.72 – 3.13)	
Maternal Stress at Birth (missing = 15)					$\chi^2_{(1)} = 7.35, p=0.007$			$\chi^2_{(1)} = 0.38, p=0.53$		
Normal – Low	58	(14%)	360	(86%)	1.00				1.00	
High	36	(33%)	73	(67%)	2.50		(1.29 – 4.86)		1.31 (0.56 – 3.04)	

Maternal Stress at 3.5 Years (missing = 14)					$\chi^2_{(1)} = 14.02, p=0.0002$		$\chi^2_{(1)} = 5.85, p=0.02$	
Normal - Low	56	(87%)	372	(13%)	1.00		1.00	
High	39	(37%)	66	(63%)	3.41 (1.79 – 6.47)		2.48 (1.19 – 5.17)	
Maternal Social Support at Birth (missing = 12)					$\chi^2_{(1)} = 4.13, p=0.04$		$\chi^2_{(1)} = 3.42, p=0.06$	
Normal - High	65	(16%)	328	(84%)	1.00		1.00	
Low	31	(21%)	114	(79%)	1.88 (1.02 – 3.47)		1.98 (0.96 – 4.09)	
Maternal Social Support at 3.5 Years (missing = 12)					$\chi^2_{(1)} = 4.77, p=0.03$		$\chi^2_{(1)} = 1.83, p=0.17$	
Normal - High	52	(15%)	294	(85%)	1.00		1.00	
Low	44	(23%)	148	(77%)	1.93 (1.07 – 3.48)		1.65 (0.79 – 3.41)	
Maternal Smoking prior to pregnancy (missing = 15)					$\chi^2_{(1)} = 18.36, p<0.0001$		$\chi^2_{(1)} = 4.51, p=0.03$	
No	34	(12%)	253	(88%)	1.00		1.00	
Yes	62	(25%)	186	(75%)	3.69 (2.03 – 6.71)		2.01 (1.06– 4.15)	
Paternal Smoking (missing = 17)					$\chi^2_{(1)} = 16.98, p<0.0001$		$\chi^2_{(1)} = 4.27, p=0.04$	
No	48	(12%)	343	(88%)	1.00		1.00	
Yes	48	(34%)	94	(66%)	3.64 (2.00 – 6.73)		2.21 (1.04 – 4.68)	
Maternal Alcohol Use in Pregnancy (missing = 15)					$\chi^2_{(2)} = 6.68, p=0.04$		$\chi^2_{(2)} = 7.97, p=0.02$	
0 units/week	34	(14%)	202	(86%)	1.00		1.00	
1-7 units/week	53	(19%)	221	(81%)	1.32 (0.72 – 2.45)		1.51 (0.75 – 3.08)	
> 7 units/week	9	(36%)	16	(64%)	4.52 (1.44 – 14.22)		6.13 (1.73 – 21.65)	
Biological Father living at Home at 3.5 Years (missing = 14)					$\chi^2_{(1)} = 19.72, p<0.0001$		$\chi^2_{(1)} = 4.49, p=0.03$	
Yes	69	(15%)	407	(85%)	1.00		1.00	
No	27	(45%)	33	(55%)	6.76 (2.91 – 15.70)		3.11 (1.09 – 8.89)	
Child Activity Level at 3.5 Years (missing = 15)					$\chi^2_{(1)} = 4.39, p=0.04$		$\chi^2_{(1)} = 4.76, p=0.03$	
More Active	38	(21%)	140	(79%)	1.88 (1.04 – 3.39)		2.13 (1.06 – 4.31)	
Same / Less Active	58	(16%)	299	(87%)	1.00		1.00	

† Multiple regression odds ratio reported adjusts for the effects of child's sex; maternal age at the child's birth; maternal school leaving age; parental marital status at birth; maternal smoking prior to pregnancy; maternal alcohol use during pregnancy; paternal smoking; living with the child's biological father at 3.5 years and child physical activity levels at age 3.5 years.

Table II. Frequencies and interaction p values for variables measured at a child's birth and age 3.5 years associated with preschool behavioural problems in the SGA and AGA groups.

	SGA		SGA		AGA		AGA		Interaction
	Abnormal		Normal		Abnormal		Normal		(SGA/AGA status * risk factor)
	N = 40		N = 185		N = 56		N = 257		
A Child’s Sex									
Female	18	(15%)	104	(85%)	31	(20%)	122	(80%)	$\chi^2_{(1)} = 0.24, p=0.62$
Male	22	(21%)	81	(79%)	25	(16%)	135	(84%)	
Maternal School Leaving Age									
< 16 years	9	(36%)	16	(64%)	10	(47%)	11	(52%)	$\chi^2_{(2)} = 0.44, p=0.80$
= 16 years	12	(18%)	53	(82%)	22	(26%)	62	(74%)	
> 16 years	18	(13%)	116	(87%)	24	(12%)	182	(88%)	
Maternal Age at Birth of Child									
<20 years	6	(23%)	20	(77%)	9	(47%)	10	(53%)	$\chi^2_{(3)} = 4.87, p=0.18$
20 – 24 years	11	(22%)	40	(78%)	18	(24%)	57	(76%)	
25 – 29 years	15	(16%)	77	(84%)	18	(14%)	111	(86%)	
30+ years	6	(11%)	47	(89%)	11	(13%)	75	(87%)	
Marital Status									
Married	22	(13%)	145	(87%)	35	(14%)	214	(86%)	$\chi^2_{(2)} = 1.19, p=0.55$
De Facto	10	(26%)	28	(74%)	17	(31%)	38	(79%)	
Single	8	(40%)	12	(60%)	4	(44%)	5	(56%)	
Maternal Stress at Birth									
Normal – Low	20	(12%)	148	(88%)	38	(15%)	212	(85%)	$\chi^2_{(1)} = 1.79, p=0.18$
High	20	(37%)	34	(63%)	16	(29%)	39	(71%)	

Maternal Stress at 3.5 years

<i>Normal – Low</i>	20	(11%)	157	(88%)	36	(14%)	215	(86%)	$\chi^2_{(1)} = 2.23, p=0.13$
<i>High</i>	19	(41%)	27	(58%)	20	(34%)	39	(66%)	

Maternal Smoking Prior to Pregnancy

<i>No</i>	13	(13%)	86	(87%)	21	(11%)	167	(89%)	$\chi^2_{(1)} = 2.95, p=0.09$
<i>Yes</i>	27	(22%)	97	(78%)	35	(28%)	89	(72%)	

Paternal Smoking

<i>No</i>	16	(11%)	132	(89%)	32	(13%)	211	(87%)	$\chi^2_{(1)} = 0.31, p=0.58$
<i>Yes</i>	21	(32%)	50	(68%)	24	(35%)	44	(65%)	

Maternal Alcohol Use in Pregnancy

<i>0 units/week</i>	13	(14%)	82	(86%)	21	(15%)	120	(85%)	$\chi^2_{(2)} = 4.26, p=0.12$
<i>1-7 units/week</i>	24	(21%)	93	(79%)	29	(18%)	128	(81%)	
<i>> 7 units/week</i>	3	(27%)	8	(73%)	6	(43%)	8	(57%)	

Biological Father living at Home at 3.5 Years

<i>Yes</i>	24	(13%)	161	(87%)	45	(15%)	246	(85%)	$\chi^2_{(1)} = 1.17, p=0.28$
<i>No</i>	16	(41%)	23	(59%)	11	(52%)	10	(48%)	

Child Activity Level at 3.5 Years

<i>More Active</i>	17	(20%)	66	(80%)	17	(20%)	66	(80%)	$\chi^2_{(1)} = 0.55, p=0.46$
<i>Same / Less Active</i>	23	(16%)	117	(84%)	23	(16%)	117	(84%)	

Maternal Social Support at Birth

<i>Normal - High</i>	31	(19%)	135	(81%)	34	(15%)	193	(85%)	$\chi^2_{(1)} = 1.59, p=0.21$
<i>Low</i>	9	(15%)	50	(85%)	22	(26%)	64	(74%)	

Maternal Social Support at 3.5 Years

<i>Normal - High</i>	20	(15%)	115	(85%)	32	(15%)	179	(85%)	$\chi^2_{(1)} = 0.11, p=0.74$
<i>Low</i>	20	(22%)	70	(8%)	24	(24%)	78	(76%)	

† Interaction between SGA/AGA status and risk factor examined in multiple regression model including a child's sex; maternal age at the child's birth; maternal school leaving age; parental marital status at birth; maternal smoking prior to pregnancy; maternal alcohol use during pregnancy; paternal smoking; maternal stress at age 3.5 years; living with the child's biological father at 3.5 years and child physical activity levels at age 3.5 years