Risk factors for Aboriginal low birthweight, intrauterine growth retardation and preterm birth in the Darwin Health Region

Susan Sayers and Jennifer Powers
Menzies School of Health Research, Darwin

Abstract: Risk factors for Aboriginal low birthweight (<2500 g), preterm birth (<37 weeks' gestation) and intrauterine growth retardation (under the tenth percentile of Australian birthweights for gestational age) were examined in 503 live-born singletons recorded as born to an Aboriginal mother and routinely delivered at the Royal Darwin Hospital between January 1987 and March 1990. Infants born to mothers with body mass index less than 18.5 kg/m^2 had five times the risk of having low birthweight and 2.5 times the risk of intrauterine growth retardation. Population-attributable risk percentages suggest that 28 per cent of low birthweight and 15 per cent of growth retardation could be attributed to maternal malnutrition. Risk percentages for maternal smoking of more than half a packet of cigarettes a day were 18 per cent for low birthweight and 10 per cent for growth retardation. For growth retardation, 18 per cent could be attributed to a maternal age under 20 years. Risk factors for preterm birth were predominantly obstetric: the population-attributable risk percentage for pregnancy-induced hypertension was 26 per cent and for other obstetric conditions was 16 per cent. For Aboriginal births in the Darwin Health Region, maternal malnutrition and smoking are key elements in the prevention of low birthweight and intrauterine growth retardation. Teenage pregnancy is an important risk for intrauterine growth retardation, and pregnancy-induced hypertension is a risk for preterm birth. (*Aust N Z J Public Health* 1997; 21: 524–30)

ABORIGINAL birthweights are lower than those for non-Aboriginal Australians throughout the country.[1] Aboriginal neonatal and infant mortality rates remain two to three times those of the Australian population, and currently, Aboriginal young to middle-aged adults have death rates up to 10 times those of the rest of the Australian population.[2] Birthweight is probably the single most important factor affecting neonatal mortality,[3] and there is evidence of associations of low birthweight with risks of adult morbidity.[4] Therefore, a better understanding of the factors influencing Aboriginal birthweights may improve the health of Aboriginal infants during the neonatal period and beyond.

As birthweight is affected by both intrauterine growth and the duration of gestation, low birthweight may be caused by intrauterine growth retardation, preterm birth or both. The predictors of preterm birth and growth retardation are thought to be different and their distributions vary in different populations. However, owing to difficulties in determining the gestational ages of Aboriginal infants, many studies of Aboriginal births have been unable to distinguish preterm birth and growth retardation. In addition, the term *Aboriginal* hides a great deal of heterogeneity, and risk factors are likely to be different within the population. Therefore, the interpretation of findings may be confusing without some definition of the study population.

In this study of a defined Aboriginal population, we used the Dubowitz scoring system[5] to obtain accurate gestational ages.[6] Hence, we could examine whether potential risk factors were different for preterm births and growth-retarded infants.

Methods

Subjects

The Royal Darwin Hospital serves the population within the Darwin Health Region, which covers 120 000 km^2 and represents about two-thirds of the population and almost half of the Aboriginal population of the Northern Territory. Aboriginal mothers are routinely referred to the hospital for delivery, and the percentage of women having babies outside this hospital is low; in 1987, 89.2 per cent and in 1988, 90.7 per cent of Northern Territory Aboriginal mothers gave birth in a hospital.[7] Babies were eligible for the study if they were live-born singletons delivered at the Royal Darwin Hospital between January 1987 and March 1990 to a mother living in the Darwin Health Region and recorded in the delivery suite register as a self-identified Aborigine.

Details of the study population have been reported previously.[8] In brief, those studied were not randomly selected. This was because of the absence of the paediatrician and inability to find some of the mothers to obtain permission to enrol them in the study. However, there were no significant differences between the sex ratios or the mean birthweights of subsets and the total population fitting the selection criteria.

Correspondence to Dr S.M. Sayers, Menzies School of Health Research, PO Box 41096, Casuarina, NT 0811. Fax (08) 8927 5187.

Procedures

Within two hours of delivery, birthweights and crown–heel lengths were measured by midwives, and within four days of delivery the neonatal paediatrician (S.S.) interviewed the mothers, obtained their consent for enrolment in the study and examined their babies. The gestational age was estimated by using neurological and physical criteria described by the Dubowitz scoring system and previously evaluated for Aboriginal babies.[6] The paediatric research nurse weighed and measured the mothers before discharge when they were ambulatory.

The paediatrician obtained information from the mother about home location, maternal ancestry (maternal knowledge of a non-Aboriginal ancestor), smoking and alcohol ingestion. Maternal and obstetric history was extracted from case notes (Table 1). The variables were confined to those that could reliably be determined, and therefore related primarily to the current maternal circumstances.

Definitions used for perinatal outcomes

Gestational age was defined according to the World Health Organization's convention, where 36 weeks' gestation means exactly 36 weeks or any fraction up to 36 weeks and six days. Preterm was defined as less than 37 weeks' gestation. Low birthweight was defined as under 2500 g, and intrauterine growth retardation as less than the tenth percentile of birthweights for gestational age and sex.[9]

Data quality

Perinatal outcomes were complete for all subjects. Data for maternal explanatory variables were virtually complete (less than 1 per cent of values missing) for all variables of interest except for maternal body mass index (BMI, 29 per cent of values missing) and antenatal attendance adequacy (14 per cent missing) (Table 1).

Analysis

The effect of each explanatory variable on each perinatal outcome was investigated by univariate analysis; chi-squared tests were used for contingency tables and two-sample t tests for continuous variables.

Multiple linear regression was used to predict birthweight. The outcomes: low birthweight, intrauterine growth retardation and preterm birth were modelled with logistic regression. Gestational age and infant sex were controlled for in all models. Explanatory variables were included in the initial model if they were in the best subset of explanatory variables to fit the regression (SPIDA) and/or P values were <0.05 in the univariate analysis.[10] The least significant variable was removed until all explanatory variables were significant ($P < 0.05$). At this stage, maternal ancestry was added to the model to determine whether this would explain any additional variance. Lastly, based on clinical judgment, a limited number of interaction terms were evaluated.

Analyses for the predictors of birthweight were done on four data sets: a complete data set that excluded records missing maternal BMI and antenatal attendance data ($n = 321$), a data set excluding records with missing BMI ($n = 357$), a data set excluding records with missing antenatal attendance ($n = 431$) and a substituted data set ($n = 503$), which used substitutions for all missing values. For the outcomes of low birthweight, intrauterine growth retardation and preterm birth, logistic regression analyses were on the four data sets. Missing values for predictor variables were replaced

Table 1: Possible explanatory variables for perinatal outcomes of 503 single live Aboriginal births at Royal Darwin Hospital, 1987–1990

Description of variable[a]	%[b]	Total
Maternal characteristics		
Age		
<20 years	32.0	503
>35 years	4.6	503
Body mass index (postpartum)		
<18.5 kg/m²	15.4	357
≥25 kg/m²	20.4	357
Cigarettes per day		
up to half a packet	19.8	500
over half a packet	33.8	500
Drinks alcohol	12.7	498
Primipara	34.0	503
Inadequate antenatal care[c]	57.3	431
Rural resident	76.1	503
Only Aboriginal ancestor	76.7	503
Maternal history for current pregnancy		
Anaemia (haemoglobin <100 g/L)	26.3	502
Urinary tract infection (positive culture)	19.9	502
Chronic respiratory conditions	3.4	502
Diabetes (gestational or other)	3.6	502
Gestational diabetes	2.8	502
Prior diabetes[d]	0.8	502
Genital diseases (treated during pregnancy)	21.9	503
Chlamydia	5.4	502
Syphilis	3.8	502
Gonorrhoea	2.2	503
Candida	11.1	503
Trichomonas	4.8	503
Other genital infections	2.2	503
Other medical disease	20.9	503
Acute respiratory infection	7.6	502
Heart disease	1.0	502
Hypertension	0.8	502
Hepatitis B	6.0	502
Skin problems	3.4	503
Other medical conditions	5.2	502
Obstetric history for current pregnancy		
Prolonged rupture of membranes (>24 hours)	3.4	502
Pregnancy-induced hypertension[e]	8.8	502
Other obstetric conditions	4.0	502
Antepartum haemorrhage	2.2	502
Other obstetric problems	2.0	492

Notes:
(a) Variables were coded 1 = true and 0 = false.
(b) Percentages do not include missing values.
(c) Started after week 16, or <50 per cent of recommended visits.
(d) Abnormal glucose tolerance test prior to pregnancy.
(e) Diastolic blood pressure ≥90 mm Hg, with oedema and proteinuria.

by a column mean in the linear regression model, whereas in the logistic analysis, pairwise-variance covariance was used.

The proportions of low birthweight, growth retardation and preterm birth that might be eliminated by removing specific risk factors were estimated by population-attributable risk percentages, calculated using the formula $(I_t-I_o)/I_t\times100$, where I_t is the incidence of a perinatal outcome in the total population and I_o is the incidence of a disease in the unexposed population.[11]

Results

Infants

The study sample (n = 503) consisted of 57.7 per cent of the live-born singletons born at the Royal Darwin Hospital between January 1987 and March 1990 to an Aboriginal mother living in the Darwin Health Region. The mean birthweight of the studied infants was 3080 g (standard deviation (SD) 606 g), 14 per cent were of low birthweight, 7.4 per cent were preterm and 25 per cent had intrauterine growth retardation. Of the 70 babies with low birthweight, 47 per cent were preterm and 70 per cent had growth retardation (Table 2). Thirteen babies were both preterm and growth retarded.

Mothers

The mean maternal age was 23.0 years (SD 5.8 years). Of the mothers whose height and weight had been measured (n = 357) the mean postpartum maternal BMI and height were 22.3 kg/m^2 (SD 4.1 kg/m^2) and 159.7 cm (SD 5.2 cm), respectively, and 15.4 per cent of mothers had BMI less than 18.5 kg/m^2. The prevalence of maternal disease was high: 63 per cent of mothers were in one or more of the disease groups, although only 16 per cent were reported to have had obstetric complications for the current pregnancy. Over a quarter of mothers had had anaemia during pregnancy and a fifth had had urinary tract infection or had evidence of genital infection. A third of mothers smoked more than half a packet of cigarettes a day (Table 1).

Table 2: Relationships between low birthweight (LBW[a]), intrauterine growth retardation (IUGR[b]), and preterm birth for 503 Aboriginal infants born at Royal Darwin Hospital, 1987–1990

Classification	Preterm birth	Term birth	Total
LBW			
IUGR	13	36	49
No IUGR	20	1	21
No LBW			
IUGR	0	77	77
No IUGR	4	352	356
Total	37	466	503

Notes:
(a) LBW = birthweight <2500 g.
(b) IUGR = less than the tenth percentile of birthweights for gestational age and sex.[9]

Data quality

As maternal BMI and adequate antenatal attendance were thought to be important variables, the potential bias caused by differences between the subjects with complete data (321) and absent data (182) was evaluated. There were no significant differences between the two groups in perinatal outcomes. The prevalence of anaemia, genital disease and other medical disease was significantly higher among mothers with BMI and antenatal attendance data than those without (Table 3). Mothers with complete data were more likely to be rural dwellers and have only Aboriginal ancestors. Across the four data sets, the statistically significant determinants of birthweight were gestational age and sex of the infant, maternal height, BMI, smoking and chronic respiratory conditions. The variables of the baby being the first-born, having diabetes, and having only Aboriginal ancestors were statistically significant in all but the complete data set, whereas prolonged rupture of membranes was statistically significant only in the larger two data sets (Table 4).

Risk factors for low birthweight, intrauterine growth retardation and preterm birth

Risk factors for low birthweight, intrauterine growth retardation and preterm birth were generally consistent in the two larger data sets, although only some of these variables were significant in the smaller data sets. For preterm birth, smoking more than half a packet of cigarettes per day was significant ($P<0.05$) in the two smaller data sets (data not shown).

When we used the substituted data set (n = 503) and controlled for sex and gestation, a low maternal BMI and smoking more than half a packet of ciga-

Table 3: Comparison of maternal characteristics for data sets with and without maternal body mass index (BMI) and antenatal attendance data, for 503 Aboriginal infants born at Royal Darwin Hospital, 1987–1990

	BMI and antenatal attendance data		
	not missing n = 321	missing n = 182	
Maternal characteristic	%	%	P
Age <20 years	34.0	28.6	0.21
Age ≥34 years	4.7	4.4	0.87
Up to ½ packet cigarettes a day	19.9	19.2	0.92
Over ½ packet cigarettes a day	31.8	36.8	0.20
Drinks alcohol	8.8	19.7	<0.001
Primipara	33.6	34.6	0.83
Rural resident	90.3	51.0	<0.001
Only Aboriginal ancestors	90.3	52.7	<0.001
Anaemia	30.2	19.2	0.008
Urinary tract infection	22.1	15.9	0.10
Chronic respiratory conditions	3.7	2.7	0.56
Diabetes (gestational or other)	3.4	3.8	0.80
Genital disease	24.9	16.5	0.03
Other medical disease	23.6	15.9	0.04
Prolonged ruptured membranes	3.4	3.3	0.95
Pregnancy-induced hypertension	9.7	7.1	0.35
Other obstetric conditions	3.7	4.4	0.71

Table 4: Birthweight determinants for four data sets for 503 Aboriginal infants born at Royal Darwin Hospital, 1987–1990

	Data sets excluding records with missing values									Data set with substitutions for missing values[d] n = 503		
	Attendance and BMI[a] n = 321			BMI[b] n = 357			Attendance[c] n = 431					
Determinants	ß[e]	SE[f]	P	ß[e]	SE[f]	P	ß[e]	SE[f]	P	ß[e]	SE[f]	P
Constant	−6314	997	<0.001	−6145	908	<0.001	−6539	818	<0.001	−6359	730	0.001
Male baby	145	51	0.004	167	47	<0.001	164	43	<0.001	180	39	<0.001
Week of gestation	158	17	<0.001	154	16	<0.001	169	12	<0.001	165	12	<0.001
Centimetre of height	16	5	<0.001	16	5	<0.001	15	4	<0.001	16	4	<0.001
Unit of BMI	42	7	<0.001	40	6	<0.001	39	6	<0.001	36	5	<0.001
Cigarettes per day												
over ½ packet	−221	58	<0.001	−215	54	<0.001	−202	50	<0.001	−230	45	<0.001
up to ½ packet	−63	67	0.345	−73	62	0.241	−33	58	0.567	−76	52	0.146
Chronic respiratory condition	−352	136	0.010	−342	132	0.010	−3006	116	0.009	−314	109	0.004
First-born	−99	55	0.072	−107	50	0.034	−139	46	0.003	−148	42	<0.001
Diabetes	215	139	0.123	257	125	0.041	381	118	0.001	438	105	<0.001
Prolonged rupture of membranes	−136	141	0.335	−149	131	0.256	−262	120	0.029	−265	109	0.016
Only Aboriginal ancestors	−110	88	0.213	−152	76	0.048	−194	57	0.001	−235	47	<0.001

Notes:
(a) BMI = body mass index. Excluded records with missing data on BMI and antenatal attendance. r^2 goodness-of-fit = 0.41.
(b) Excluded records with missing data on BMI only. r^2 goodness-of-fit = 0.42.
(c) Excluded records with missing data on antenatal attendance only. r^2 goodness-of-fit = 0.48.
(d) Substitutions were used for all missing values. r^2 goodness-of-fit = 0.49.
(e) ß = coefficient.
(f) SE = standard error.

rettes a day increased the risks of both low birthweight and growth retardation. In addition, maternal age under 20 years was a risk factor for growth retardation, but not for low birthweight or preterm birth. Mothers with only Aboriginal ancestors had a higher risk of having infants with both low birthweight and growth retardation (Tables 5 and 6). When sex was controlled for, the risk factors for preterm birth were related to the current obstetric history (Table 7). As smoking was significant in the complete data set, it is included in Table 7. Removing smoking from the model reduced the explained variance to 20 per cent, whereas removing prolonged rupture of membranes reduced the explained variance to 14 per cent.

The population-attributable risks suggest that 28 per cent of low birthweight could be attributed to mothers having a BMI less than 18.5 kg/m^2 (I_t = 49/357, I_o = 30/302) and 18 per cent attributed to mothers smoking more than half a packet of cigarettes a day (I_t = 70/500, I_o = 38/331). Of the identified risk factors for intrauterine growth retardation, 18 per cent could be attributed to mothers under 20 years of age (I_t = 126/503, I_o = 70/342), 15 per cent attributed to mothers having a BMI under 18.5 kg/m^2 (I_t = 96/357, I_o = 69/302) and 10 per cent to mothers smoking more than half a packet of cigarettes a day (I_t = 126/500, I_o = 75/331).

For preterm births, 26 per cent could be attributed to pregnancy-induced hypertension (I_t = 37/502, I_o = 25/458) and 16 per cent to other obstetric conditions (I_t = 37/502, I_o = 30/482).

Discussion

In this study we defined a population of Aboriginal mothers and used reliable gestational age estimations to determine the specific associations of some established risk factors with preterm birth and intrauterine growth retardation. Previous studies of Aboriginal pregnancies have documented birth antecedents, but there is a paucity of data on specific links between maternal factors and perinatal outcomes with reliable gestational age estimates.

Despite intensive follow-up, 29 per cent of mothers were missing BMI measurements and 14 per cent missing antenatal attendance details. The neonatal paediatrician was able to interview mothers soon after the birth, but many mothers, when they were ambulatory and able to be weighed and measured, could not be found within the hospital or its surroundings. Missing antenatal details were mainly caused by mothers changing locations of antenatal care during their pregnancy, and although they had received antenatal care, specific details of attendance could not be determined.

Values for BMI and antenatal attendance values were not missing randomly (Table 3); mothers with missing values had fewer abnormalities and were more likely to be urban dwellers with a non-Aboriginal ancestor. To assess the effect of these missing values, birthweight was modelled with the four described data sets (Table 4). The estimates for most of the significant variables remained consistent; however, the baby being the first-born, diabetes, prolonged rupture of membranes and

Table 5: Risk factors for low birthweight for 503 Aboriginal infants born at Royal Darwin Hospital, 1987–1990

Risk factor	Odds ratio	CI[a]	P
Male	0.6	0.3 to 1.2	0.130
Week of gestation	0.3	0.2 to 0.4	<0.001
Body mass index[b]			
<18.5 kg/m²	5.1	2.1 to 12.0	<0.001
>25.5 kg/m²	0.3	0.1 to 1.0	0.050
Cigarettes per day			
more than ½ packet	2.8	1.3 to 6.1	0.009
up to ½ packet	0.7	0.3 to 2.0	0.557
Only Aboriginal ancestors	4.1	1.5 to 11.3	0.006

Notes:
(a) CI = 95% confidence interval.
(b) In this model, with body mass index as a categorical variable, 42.8% of the variance was explained, compared with 44.2% if body mass index was fitted as a continuous variable.

Table 6: Risk factors for intrauterine growth retardation for 503 Aboriginal infants born at Royal Darwin Hospital, 1987–1990

Risk factor	Odds ratio	CI[a]	P
Body mass index[b]			
<18.5 kg/m²	2.5	1.4 to 4.6	0.004
>25.5 kg/m²	0.4	0.2 to 0.8	0.016
Maternal age <20 years	1.9	1.2 to 2.9	0.006
Cigarettes per day			
more than ½ packet	1.8	1.1 to 3.0	0.015
up to ½ packet	1.0	0.6 to 1.8	0.990
Only Aboriginal ancestors	2.7	1.5 to 5.0	0.002

Notes:
(a) CI = 95% confidence interval.
(b) In this model, with body mass index as a categorical variable, 8.6% of the variance was explained, compared with 10.5% if body mass index was fitted as a continuous variable.

Table 7: Risk factors for preterm birth for 503 Aboriginal infants born at Royal Darwin Hospital, 1987–1990

Risk factor[b]	Odds ratio	CI[a]	P
Male	1.0	0.5 to 1.0	0.939
Pregnancy-induced hypertension	12.7	5.2 to 30.9	<0.001
Prolonged ruptured membranes	18.7	5.9 to 59.7	<0.001
Other obstetric problem	15.0	5.0 to 44.9	<0.001
Cigarettes per day			
more than ½ packet	2.2	0.9 to 5.0	0.069
up to ½ packet	1.4	0.5 to 4.0	0.494

Notes:
(a) CI = 95% confidence interval.
(b) 21% of the variance explained by these factors.

Aboriginal ancestry failed to remain significant in the smallest data set, which excluded records with missing BMI and antenatal attendance values. The numbers of mothers with diabetes and prolonged rupture of membranes (which have a large effect on birthweight) were reduced in the complete data set and therefore were no longer statistically significant. On the other hand, the effect of the baby being the first-born on birthweight is relatively small and required the larger data sets to achieve statistical significance. Less than 10 per cent of mothers in the complete data set had a non-Aboriginal ancestor, compared with 23 per cent in the substituted data set (Table 3), and the birthweights in this smaller comparative group were not statistically different from birthweights of those with only Aboriginal ancestors. Despite the exposed biases in the data sets, the consistency of the results across the four data sets suggest that the estimates obtained from the analysis of the substituted data set are robust. As the purpose of the modelling was to identify potentially modifiable risk factors, maternal ancestry was always added to the models last after other explanatory variables were determined, thus minimising its effect.

Our results are in general agreement with the meta-analysis of 895 non-Aboriginal studies on low birthweight reported by Kramer, in which the risk factors for intrauterine growth retardation were mainly social and biological, in contrast to those for preterm birth, which related primarily to reproductive history.[12]

The mean maternal height (160 cm) is consistent with previous reports of maternal Aboriginal stature,[13] and compares favourably with reported means for other populations.[14] Birthweight increased by 16 g for every centimetre in height and by 36 g for every unit increase in BMI (kg/m²) (Table 4). Height did not remain a significant independent factor for the other perinatal outcomes studied.

Chronic maternal respiratory conditions had a detrimental effect on birthweight but were not significant for the other perinatal outcomes. Severe respiratory illness associated with early maternal death has been reported to shorten gestation[15] and reduce birthweight.[16] However, none of the 17 women with bronchiectasis, bronchial asthma or chronic bronchitis had severe illness. The small number of mothers with chronic respiratory conditions may have some other unmeasured risk factor for birthweight. The consistency of this effect across the four data sets warrants further investigation.

The Food and Agriculture Organization of the United Nations uses BMI less than 18.5 kg/m² to estimate the prevalence of malnutrition in women.[17] When this BMI cut-off was used, there was at least a medium prevalence of malnutrition for the mothers in this study, and considering that the measurements were postpartum, a higher prevalence of malnutrition may exist. These mothers were probably malnourished long before pregnancy, as recently 20 per cent of Aboriginal children under two years of age have been estimated to be malnourished.[18]

Infants born to mothers with BMI under 18.5 kg/m² had five times the risk of having low birthweight and over two and a half times the risk of having intrauterine growth retardation. The combination of these risks with the prevalence of

maternal malnutrition suggests that 28 per cent of low birthweight and 15 per cent of intrauterine growth retardation could be attributed to continuing poor nutrition of Aboriginal women in the Darwin Health Region. These findings are consistent with a previous study of two Top End communities which used less reliable gestational-age data.[19]

Cigarette smoking has been shown to be associated with a mean reduction of 150 to 270 g in infant birthweight.[20] A lower birthweight with maternal smoking also occurred in our study, and infants born to smokers of more than half a packet of cigarettes a day had almost three times the risk of having low birthweight and more than one and a half times the risk of intrauterine growth retardation. The proportions of low birthweight and growth retardation attributable to smoking indicate that it is an important influence on fetal growth.

High rates of Aboriginal teenage pregnancies have been reported,[21] although specific effects of lower Aboriginal maternal age on birthweight are inconclusive. Aboriginal teenage pregnancies have been reported with preterm births[21] and intrauterine growth retardation.[22] In our study, mothers under 20 years of age had almost twice the risk of intrauterine growth retardation.

Other factors likely to explain the variance of fetal growth were not identified in our study. Only the variable of maternal ancestry contributed additional variance. Maternal ancestry has no remedial elements; nevertheless, it may identify areas where social policy and health services need to be directed. Morrison et al. and Jonas et al. have linked social background and pregnancy outcomes for non-Aboriginal Australians, and part of the unexplained variance in this study may be due to unmeasured maternal social and psychological circumstances.[23,24]

The risk factors for preterm births, both spontaneous and induced, were related to obstetric disorders of the current pregnancy. The association of Aboriginal preterm births with pregnancy-induced hypertension is consistent with the findings of Blair, who, using fetal-ultrasound estimates of Aboriginal gestational age, identified pregnancy-induced hypertension, genital infections and diabetes as factors associated with Aboriginal preterm births.[25] Smoking has been reported as shortening gestation; however, in this study, it was significant only in the smaller data sets. Prolonged rupture of membranes could be considered an outcome, and removing this variable from the model reduced the explained variance from 21 per cent to 14 per cent. The lack of explanatory factors for preterm birth may be caused by grouping spontaneous and induced births, a failure to study important retrospective reproductive-history factors,[26] and possible underdiagnosis of diabetes.

The population-attributable risk percentages suggest that 26 per cent of preterm births could be attributable to pregnancy-induced hypertension. Compared with non-Aboriginal women, Aboriginal women are thought to have higher rates of pregnancy-induced hypertension, which may be because of late antenatal presentation.

Maternal age[21] maternal birthweight,[27] antenatal care,[28] anaemia,[24] urinary tract infection[29,30] and cardiac conditions[29] have all been implicated as affecting Aboriginal birthweight or the duration of gestation. In our study, anaemia and urinary tract infections were common, but effective therapeutic intervention after diagnosis probably prevented them having an effect. The number of mothers with individual medical conditions was small (Table 1) and therefore unlikely to be found significant. Grouping the medical conditions made no difference to the result.

Antenatal attendance was defined as inadequate if the first antenatal visit was after the sixteenth week of pregnancy or the mother attended less than 50 per cent of recommended visits. The lack of association of this measure with the perinatal outcomes is unlikely to be due to the 14 per cent missing values and may mean current antenatal practices are inappropriate for this population.

From this study, programs to reduce smoking during pregnancy and delay child-bearing of young adults would be expected to decrease Aboriginal low birthweight, particularly that caused by intrauterine growth retardation. Measures to improve maternal nutrition are continuing in innovative programs. However, nutritional effects on the fetus may persist over more than one generation,[31] and nutritional rehabilitation of Aboriginal mothers may be needed over generations before the effect of undernutrition on fetal growth is fully corrected.

A general failure to recognise the heterogeneity of preterm births may be hindering the identification of preventable factors for preterm birth. Hence, grouping of different types of preterm birth in a collaborative study on Aboriginal births with reliable gestational estimates would be valuable. Research directed towards identifying specific life-style patterns and social stresses associated with poor outcomes and the cultural influences and effectiveness of antenatal attendance and care for Aboriginal mothers may be helpful.

Conclusions

For Aboriginal births in the Darwin Health Region, maternal malnutrition and smoking are key elements in the prevention of low birthweight and intrauterine growth retardation. In addition, teenage pregnancy is an important risk for intrauterine growth retardation, and pregnancy-induced hypertension is a risk for preterm birth.

Acknowledgments

This work was supported by the National Health and Medical Research Council of Australia. We thank Dorothy Mackerras for advice on form and content of the manuscript and especially thank the Aboriginal mothers who agreed to themselves and their babies being part of this study.

References

1. Lancaster PAL. The health of Australia's mothers and babies. *Med J Aust* 1996; 164: 198–9.

2. Gracey M, Veroni M. Comparative hospitalisation and mortality rates of Aboriginal and non-Aboriginal Western Australians in their sixth and seventh decades. *Aust N Z J Med* 1995; 25: 27–31.
3. McCormick MC. The contribution of low birth weight to infant mortality and childhood morbidity. *N Engl J Med* 1985; 312: 82–90.
4. Barker DJP, Martyn CN. The maternal and fetal origins of cardiovascular disease. *J Epidemiol Community Health* 1992; 46: 8–11.
5. Dubowitz LMS, Dubowitz VA. A clinical manual: gestational age of the newborn. Manila: Addison–Wesley, 1977.
6. Sayers SM, Powers JR. An evaluation of three methods used to assess the gestational age of Aboriginal neonates. *J Paediatr Child Health* 1992; 28: 312–17.
7. *Annual report 1988–89.* Darwin: Northern Territory Department of Health and Community Services, 1989.
8. Sayers SM, Powers JR. Birth size of Australian Aboriginal babies. *Med J Aust* 1993; 159: 586–91.
9. Guaran RL, Wein P, Sheedy M, Walstab J, et al. Update of growth percentiles for infants born in an Australian population. *Aust N Z J Obstet Gynaecol* 1994; 34: 1: 39–50.
10. Gebski V, Leung O, McNeil D, Lunn A. SPIDA, Version 6 [computer program]. Sydney: Statistical Laboratory, Macquarie University, 1992.
11. Hennekens CH, Buring JE. *Epidemiology in medicine.* 1st edn. Boston: Little, Brown, 1987: 90–3.
12. Kramer MS. Intrauterine growth and gestational duration determinants. *Pediatrics* 1987; 80: 502–11.
13. Seward JF, Stanley FJ. Comparison of births to Aboriginal and Caucasian mothers in Western Australian. *Med J Aust* 1981; 2: 80–4.
14. World Health Organization. *Maternal anthropometry and pregnancy outcomes.* A WHO collaborative study. *Bull WHO* 1995; 73: 15–17.
15. Cohen LF, di Sant'Agnese PA, Friedlander J. Cystic fibrosis and pregnancy. A national survey. *Lancet* 1980; 842–4.
16. Gordon M, Niswander KR, Berendes H, Kantor AG, Fetal morbidity following potentially anoxigenic obstetric condition. VII. Bronchial asthma. *Am J Obstet Gynec* 1970; 106: 421–9.
17. *Physical status: the use and interpretation of anthropometry.* Report of WHO expert committee. WHO technical report series no. 854. Geneva: World Health Organization, 1995.
18. Ruben AR, Walker AC. Malnutrition among rural Aboriginal children in the Top End of the Northern Territory. *Med J Aust* 1995; 162: 400–3.
19. Rae CJ. *Maternal nutritional status among Aborigines in the Northern Territory: impact on birthweight* [thesis]. Darwin: Menzies School of Health Research, University of Sydney, 1989; 106.
20. Butler NR, Goldstein H, Ross EM. Cigarette smoking in pregnancy: its influence on birth weight and perinatal mortality. *BMJ* 1972; 2: 127–30.
21. Stanley FK, Mauger S. Birth-weight patterns in Aboriginal and non-Aboriginal singleton adolescent births in Western Australia, 1979–1983. *Aust N Z Obstet Gynaecol* 1986; 26: 49–54.
22. Julienne A. A comparative study of perinatal outcome among Aboriginal and non-Aboriginal hospital confinements in rural NSW, 1981. Working paper no.3. Sydney: NSW Department of Health, 1983.
23. Morrison J, Najman JM, Williams GM, Keeping JD, et al. Socio-economic status and pregnancy outcome. An Australian study. *Br J Obstet Gynaecol* 1989; 96: 298–307.
24. Jonas O, Roder D, Chan A. The association of maternal and socioeconomic characteristics in metropolitan Adelaide with medical, obstetric and labour complications and pregnancy outcomes. *Aust N Z Obstet Gynaecol* 1992; 32: 1–5.
25. Blair E, Morich P, Stanley F. Why do Aboriginal newborns weigh less? Gestational age at delivery: estimation, distribution and determinants. *Aust N Z J Obstet Gynaecol* 1994; 34: 158–63.
26. Creasy RK. Preventing preterm birth. *N Engl J Med* 1991; 325: 727–9.
27. Dugdale AE, Hendrikz J, Musgrave IA, Streatfield K. The effect of mother's birthweight on the birthweights of her children. In: Gray A. *A matter of life and death.* Proceedings of a workshop of the National Centre of Epidemiology and Population Health; 1989; Kiola, NSW. Canberra: Aboriginal Studies Press, 1990: 25–31.
28. Keeping JD, Chang AMZ, Morrison J, Esler EJ. Poor antenatal attendance and obstetric performance. *Aust N Z J Obstet Gynaecol* 1980; 20: 139–43.
29. Hart G, MacHarper T, Moore D, Roder D. Aboriginal pregnancies and births in South Australia, 1981–1982. *Med J Aust* 1985; 143: S54–6.
30. Schultz R, Read AW, Straton JAY, Stanley FJ, et al. Genitourinary tract infections in pregnancy and low birth weight; a case–control study in Australian Aboriginal women. *BMJ* 1991; 303: 1369–73.
31. Harding JE, Johnston BM. Nutrition and fetal growth. *Reprod Fertil Dev* 1995; 7: 539–47.