Predicitve relationship of eczema and wheeze

Table 4 shows sensitivity and specificity of eczema as screening test for wheeze in first and second six months of life. The sensitivity of eczema for detection of wheeze in first year of life was very low so was the positive predictive value of eczema in Table 5. We did not calculate sensitivity and specificity for each region because of low number of positive cases.

Table 1 Percentage of children with demographic and personal characteristics according to study sites

Factor	All	Central	South	North East	North	Bangkok
	N=4021	N=744	N=1045	N=846	N=748	N=638
Sex						
N	4021	744	1045	846	748	638
Male	49.7	41.2	51.9	48.7	49.6	50.6
Mother's education						
N	3982	739	1043	831	743	625
Illiterate	5.2	4.1	5.6	1.1	14.9	0.2
Primary school	47.9	61.4	60.5	61.1	28.7	16.0
Secondary school	28.7	25.6	26.3	30.5	29.6	32.9
College	8.4	5.5	4.1	4.7	11.3	20.4
Bachelor degree	9.8	3.4	3.5	2.6	15.5	30.5
Annual family income (Bath)*						
N	3918	732	1037	832	717	600
<50,000	24.8	14.8	36.5	29.5	32.9	0.7
50,000-100,000	25.1	29.9	35.2	27.4	20.4	4.5
100,001-200,000	25.7	32.5	21.6	26.5	22.3	27.3
>200,000	24.4	22.8	6.7	16.6	24.4	67.5
Mother smoked while pregnant						
N	3787	684	976	802	716	636
Yes	1.9	1.3	2.7	0.7	0.9	3.6
Smoker at home						
N	4019	744	1045	846	748	636
Yes	46.8	37.4	75.6	52.6	26.6	26.7
Mother has allergy						
N	3786	687	974	801	716	608
Yes	6.4	10.9	2.7	1.9	4.3	15.8
Father has allergy						
N	3225	587	858	654	618	508
Yes	6.4	10.7	3.5	3.2	4.7	12.2

* 40 baht = 1 US dollar

Table 2 Peroid prevalence of eczema and wheeze in the first and second six months of life by area (n=4021)

Area	Number of Subjects	Prevalence of Eczema (%)		Prevalence of Wheeze (%)	
		0-6 months	6-12 months	0-6 months	6-12 months
Central	744	4.0	2.7	4.4	5.8
South	1045	7.2	6.2	10.1	12.4
North East	846	2.9	2.3	5.8	11.6
North	748	2.8	3.6	5.5	6.3
Bangkok	638	4.7	2.3	9.7	8.8
All	4021	4.8	3.6	7.2	9.3

Pearson' s correlation: eczema 0-6 mo. VS wheeze 0-6 mo. = 0.79

eczema 6-12 mo. VS wheeze 6-12 mo. = 0.43

Table 3 Odds ratio of eczema and wheeze in the first and second six months of life

Area	Wh6/Wh12	Ec6/Ec12	Ec6/Wh6	Ec12/Wh12	Ec6/Wh12	Wh6/Ec12
Central	1.7	1.3	4.9	0.8	1.8	-
South	3.7	6.2	1.1	1.1	1.2	1.1
North East	5.2	4.3	4.6	4.7	1.1	3.2
North	4.9	3.8	2.3	4.7	1.4	1.3
Bangkok	6.4	5.5	1.4	0.7	1.1	-
All	4.5 (3.4-6.0)	5.3 (3.4-8.2)	2.1 (1.4-3.2)	2.0 (1.2-3.1)	1.3 (0.8-2.1)	1.2 (0.6-1.2)

Wh 6 = wheeze 0-6 mo.

Wh 12 = wheeze 6-12 mo.

Ec 6 = eczema 0-6 mo.

Ec 12 = eczema 6-12 mo.

Table 4 sensitivity and specificity of eczema to predict wheeze in the first and second six months of life (n=4021)

Factors	Wheeze 0-6 mo.		Wheeze 6-12 mo.	
	Sensitivity	Specificity	Sensitivity	Specificity
Wheeze 0-6 mo.	-	-	21.6 (17.6-26.1)	94.2 (93.4-94.9)
Eczema 0-6 mo.	8.9 (5.9-12.8)	95.4 (94.7-96.1)	6.1 (3.9-9.1)	95.3 (94.6-95.9)
Eczema 6-12 mo.	-	-	6.4 (4.1-9.3)	96.6 (96.0-97.2)

Table 5 Predictive value of eczema to predict wheeze in the first and second six months of life (n=4021)

Factors	Wheeze 0-6 mo.		Wheeze 6-12 mo.	
	PPV	NPV	PPV	NPV
Wheeze 0-6 mo.	-	-	27.8 (22.8-33.4)	92.1 (91.2-93.0)
Eczema 0-6 mo.	13.4 (8.9-19.0)	93.1 (92.2-93.9)	11.8 (7.7-17.2)	90.8 (89.9-91.7)
Eczema 6-12 mo.	-	-	16.4 (10.8-23.5)	90.9 (90.0-91.8)

PPV = Positve predictive value

NPV = Negative predictive value

Discussion

The study showed that eczema prevalence declined where as that of wheeze increased risk with age. With five study cohorts of different geo-demographic settings, the peroid prevalence of eczema and wheeze varied to a maximum of 2-3 fold across areas with high correlation in the first six months and less so in the second six months. Association in individual level was not so strong and with less change overtime. Prediction across the two peroid was better for the same symptom than across symptoms. In using eczema to predict wheeze, the sensitivity and positive predictive value are both low and not useful enough for clinical use.

The variations in the prevalence of asthma and eczema across area are similar to previous reports from The International study of Asthma and Allergies in Childhood (ISAAC) which found variations of both symptoms with up to 15-fold differences between countries [4;5]. The concern of the prevalence variation is the potential source of bias such as cultural and linguistic differences. We minimized this bias by use the standard question for wheeze as in ISAAC questionnaire and pictures of rashes to detect eczema instead of asking question. The other explantion of the prevalence variations is the difference of IgE production in various population. Level of IgE has a positive correlation with risk of asthma and eczema[6;7]. Therefore, the prevalence of asthma amd eczema should depend on the IgE level in that population. The level of IgE is known to be influenced strongly by parasitic infections [8], genetics [9] and to a lesser extent exposure to tobacco [10]. In southern Thailand, the area is known to have high prevalence of soil-

transmitted helminths [11] which might influence on the prevalence of eczema and wheeze.

The current finding of decreasing eczema along the age is consistent with that from a large population-based study in the U.K. (ALSPAC) which showed incidence risks of atopic eczema being 21.0%, 11.2% and 3.8% at 0-6, 6-18 and 18-30 months of age.[12] The increase of peroid prevalence of wheeze in older children was reported in many articles. Tariq et al [13] found the increase of peroid prevalence of asthma from 8.7% in infancy to 14.9% at 4 years of age in a birth cohort of 1218 children. Luyt et al [14] reported the increase of prevalence of wheeze in 1650 children (7.5% in children under 1 year, 15.9% in children of 4 years and over; $p<0.001$). The changes of these two prevalences in an opposite direction resulted in less correlation between the prevalence of eczema and wheeze in the second six months than in the first six months of life. This is similar to the finding by Kjellman and Hattevig [15] who reported that children with early onset of eczema had a greater probability to develop respiratory symptoms as compared to children with later onset of eczema. The decline of correlation across the peroids also suggests that there was a reduction of sharing in etiology between eczema and wheeze. Infantile eczema usually associates with food allergy which always declines with age [16] while wheeze in infant is a group of heterogenous disorders [17].

We found significant association between eczema and wheeze in individual level only within the same six months peroid. Many investigators reported in hospital-based studies that children with atopic eczema have high prevalence of bronchial

hyperessponsiveness[18;19]. The biological pausibility is the correlation between IgE level, eczematous symptoms and bronchial hyperresposiveness. Children with visible eczema have high IgE level[6] and children with high IgE level have bronchial hyperresponsiveness[7]. Thus, the risk of wheeze increased when children had current eczema.

Despite the significant association, sensitivity of eczema for prediction of wheeze was rather low. In our study, 88% of wheeze occured in children without eczema. As expected, because of the low sensitivity and low prevalence of eczema and wheeze, the positive predictive value of eczema to predict wheeze was low as 11-13%.

The strength of the present study is the large sample size, minimal loss to follow up, and its prospective nature. However, objective measures of the disease activity such as pulmonary function and bronchial challenge test are not feasible in this age group. Different parental/ guardian might percieve and interprete the question and pictures provided different by leading to a misclassification of outcome measures. This misclassification bias is likely to be non-differential which dilute the level of association.

In conclusion, the predictive capacity of eczema and wheeze is not high enough to recommend them as screening instruments for respiratory prevention. The majority of wheeze occur in infants with no demonstrable eczema.

Reference List

(1) Sigurs N, Hattevig G, Kjellman B, Kjellman NI, Nilsson L, Bjorksten B. Appearance of atopic disease in relation to serum IgE antibodies in children followed up from birth for 4 to 15 years. J Allergy Clin Immunol 1994; 94(4):757-63.

(2) Kurukulaaratchy RJ, Fenn M, Matthews S, Arshad SH. Characterisation of atopic and non-atopic wheeze in 10 year old children. Thorax 2004; 59(7):563-568.

(3) Castro-Rodriguez JA, Holberg CJ, Wright AL, Martinez FD. A clinical index to define risk of asthma in young children with recurrent wheezing. Am J Respir Crit Care Med 2000; 162(4 Pt 1):1403-6.

(4) Worldwide variations in the prevalence of asthma symptoms: the International Study of Asthma and Allergies in Childhood (ISAAC). Eur Respir J 1998; 12(2):315-35.

(5) Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC. The International Study of Asthma and Allergies in Childhood (ISAAC) Steering Committee. Lancet 1998; 351(9111):1225-32.

(6) Perkin MR, Strachan DP, Williams HC, Kennedy CT, Golding J. Natural history of atopic dermatitis and its relationship to serum total immunoglobulin E in a population-based birth cohort study. Pediatr Allergy Immunol 2004; 15(3):221-229.

(7) Sears MR, Burrows B, Flannery EM, Herbison GP, Hewitt CJ, Holdaway MD. Relation between airway responsiveness and serum IgE in children with asthma and in apparently normal children. N Engl J Med 1991; 325(15):1067-71.

(8) Lynch NR, Hagel IA, Palenque ME, Di Prisco MC, Escudero JE, Corao LA et al. Relationship between helminthic infection and IgE response in atopic and nonatopic children in a tropical environment. J Allergy Clin Immunol 1998; 101(2 Pt 1):217-21.

(9) Blumenthal MN, Namboodiri K, Mendell N, Gleich G, Elston RC, Yunis E. Genetic transmission of serum IgE Levels. Am J Med Genet 1981; 10(3):219-228.

(10) Sherrill DL, Halonen M, Burrows B. Relationships between total serum IgE, atopy, and smoking: a twenty-year follow-up analysis. J Allergy Clin Immunol 1994; 94(6 Pt 1):954-962.

(11) Chongsuvivatwong V, Uga S, Nagnaen W. Soil contamination and infections by soil-transmitted helminths in an endemic village in southern Thailand. Southeast Asian J Trop Med Public Health 1999; 30(1):64-7.

(12) Wadonda-Kabondo N, Sterne JA, Golding J, Kennedy CT, Archer CB, Dunnill MG. A prospective study of the prevalence and incidence of atopic dermatitis in children aged 0-42 months. Br J Dermatol 2003; 149(5):1023-1028.

(13) Tariq SM, Matthews SM, Hakim EA, Stevens M, Arshad SH, Hide DW. The prevalence of and risk factors for atopy in early childhood: a whole population birth cohort study. J Allergy Clin Immunol 1998; 101(5):587-93.

(14) Luyt DK, Burton PR, Simpson H. Epidemiological study of wheeze, doctor diagnosed asthma, and cough in preschool children in Leicestershire. BMJ 1993; 306(6889):1386-1390.

(15) Kjellman B, Hattevig G. Allergy in early and late onset of atopic dermatitis. Acta Paediatr 1994; 83(2):229-231.

(16) Kjellman NI, Nilsson L. From food allergy and atopic dermatitis to respiratory allergy. Pediatr Allergy Immunol 1998; 9(11 Suppl):13-17.

(17) Martinez FD, Helms PJ. Types of asthma and wheezing. Eur Respir J Suppl 1998; 27:3s-8s.

(18) Corbo GM, Ferrante E, Macciocchi B, Foresi A, De A, V, Fabrizi G et al. Bronchial hyperresponsiveness in atopic dermatitis. Allergy 1989; 44(8):595-598.

(19) Salob SP, Laverty A, Atherton DJ. Bronchial hyperresponsiveness in children with atopic dermatitis. Pediatrics 1993; 91(1):13-16.

เอกสารหมายเลข 9.4

A population-based, prospective, cohort study on maternal risk factors for low birth weight in Thailand

By Dr.Sirikul Isaranurug

A population-based, prospective, cohort study on maternal risk factors for low birth weight in Thailand

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Summary

Background: Infants with a low birth weight (LBW) are at risk of premature death and those that survive have a greater risk for poor general health, neuromotor ability, cognitive and academic impairment, and behavioral problems. The study aimed to identify the maternal risk factors for LBW in Thailand.

Methodology: The Prospective Cohort Study in Thai Children (PCTC) is an ongoing population based birth cohort study. About 3 522 pregnancies were followed-up in 2000 at four districts across Thailand. The birth weights were retrieved from the Delivery Summary Sheet at each hospital. Babies born in hospital were weighed the first day, while those born at home were weighed within 3 days by a trained research assistant. Only singleton live births were included in the study.

Results: Of the 3 322 live births with valid birth weight data, 8.6% had LBW. The incidence of LBW in Central, South, Northeast and North Thailand was 7, 8, 10 and 9 percent, respectively. The maternal factors associated with LBWs and having a high population attributable risk percent (PAR%) included: weight gain during pregnancy, maternal age, parity, consultation with health personnel, maternal occupation and home ownership at 16, 16, 16, 17, 12 and 11 percent, respectively.

Conclusions: Improving the nutritional status among pregnant women is crucial to lowering the incidence of LBW. Pregnant women over 35, on their first and more than second parity, consulting healthcare personnel less frequently, having a non-professional occupation, and not owning their own home, should be followed-up closely.

Keywords: Low Birth Weight; Maternal Risk Factors; Prospective Study; Population-Based Study

Introduction

In every pregnancy, parents hope for a healthy child; however, unexpected situations occur, such as low birth weight (LBW), premature birth, congenital anomaly or stillbirth. Infants with a LBW are at greater risk of death thereby bringing grief to the families, [1-3] and those that survive have a greater risk of poor general health, impaired neuromotor, cognitive and academic ability, and behavioral problems. [1,4-8]

A high national incidence of LBWs reflects poor maternal reproductive health, nutritional status, improper practices during pregnancy, and poor socioeconomic status. Relatedly, a negative maternal reproductive health history (*i.e.* a previous stillbirth, abortion or miscarriage, early neonatal mortality, complications during pregnancy, first or high parity) is frequently associated with a LBW. [8-14]

Maternal nutritional status judging by maternal height vis-à-vis pregnancy weight gain correlates with LBWs. [13,15-17] The incidence of LBWs is also influenced by maternal smoking, [8,13,15,17,18,20,22] alcohol consumption [5,19] and late and infrequent antenatal care [11,19,21,22]. Moreover, low social class [28], maternal age over 35 [8,11,12,24,25,27,28], and less maternal education [22,24,28] are correlated with an increasing incidence of LBWs.

Since 1992, Thailand has attempted to reduce the incidence of LBWs to < 7%, but without success. In 2001, the incidence of LBW babies was still 9%. [29] The Ministry of Public Health will again attempt to reduce LBWs to less than 7% by 2006. [30] Therefore, the associated risk factors should be defined in order to set up remediation strategies.

This population-based prospective cohort study is the first longitudinal study in Thailand. Its prospective focus should provide a direct estimate of the risk of developing disease in individuals having specific characteristics. Our aim was to explore the maternal risk factors associated with the occurrence of LBWs.

The population attributable risk percent (PAR%) will be used to identify the proportion of LBWs attributable to each maternal factor. If a proportional decrease in the incidence of LBWs occurs when such factors are controlled, such information would be very useful for resource allocation and prioritization of interventions.

Materials and Methods:

The main study

The PCTC is an ongoing population-based birth cohort study established to study the relation between prenatal factors, labor and delivery, and child development through to age 25 years. All pregnant women in the late stage of pregnancy at four districts representing the four geographic regions of Thailand, with an expected date of delivery within a 12 month period of recruitment, were included. Ultimately, 3 522 live births were followed-up in 2000.

Outcome and outcome measurements

Birth weight was retrieved from the *Delivery Summary Sheet* at each hospital. The babies born in hospital were weighed the first day; those born at home were weighed within 3 days by a trained research assistant.

A LBW was defined by a birth weight of < 2500 g. As in other developing countries, where women could not recall LMP, the separation between premature and small for gestational age babies was not possible.

The subjects born at home and not weighed (89) were excluded from the analysis as were the 23 stillbirths and 28 pairs of twins. So, of the 3 522 births 3 354 singletons were included in the analysis.

Study factors

Maternal exposure factors comprised: 1) socioeconomic conditions such as education, occupation, family income, home ownership, marital status and religion; 2) health services utilization such as attendance of prenatal care, consultation with health personnel and iron supplement compliance; and, 3) reproductive health and biological conditions such as past obstetric history, parity, planned pregnancy, maternal age and height. Maternal outcomes during pregnancy comprised complications, maternal diseases and weight gain. Smoking and alcohol consumption were excluded because so few pregnant Thai women smoke or drink that a risk analysis could not be done.

Past obstetric history included history of a previous stillbirth, abortion or miscarriage, premature birth and twin pregnancy. Complications during pregnancy included hypertension, bleeding per vagina, albuminuria and glycosuria. Maternal diseases included heart, thyroid, blood and liver, syphilis, HIV, tuberculosis and allergies.

Statistical methods

The unit of analysis was live births. The incidence of LBWs at each site was calculated per 100 live births. The risk was analyzed using the relative risk (RR), the population attributable risk (PAR) and the population attributable risk percent (PAR%).

1) The RR represents the incidence rate of LBWs among persons, *viz.*: the quotient of risk factors and the incidence rate of LBWs among non-risk-exposed persons.
2) The PAR% was defined by the incidence rate of LBWs among the total population minus the incidence rate of LBWs among non-risk-exposed persons. The PAR% is expressed as = $\left(\frac{b(RR-1)}{b(RR-1)+1}\right)\times 100$ where RR is the relative risk, and b is the proportion of the total population having the risk characteristics.

Results

Magnitude of the outcome

Of the 3 322 live births with valid birth weight data, 286 had LBWs. The overall LBW rate was 8.6%. The site in Northeast Thailand had the highest rate of LBWs (10.4%) (Table 1). Among the LBW babies, 82.5% had a birth weight between 2 000 and 2 500 g, 13.0% between 1 500 and 1 999 g and 4.5% < 1 500 g.

Most (83.8%) of the studied babies were delivered naturally, while 11.2% were delivered by cesarean section (Table 2).

Risk factors

Table 3 shows the incidence of LBW babies vis-à-vis maternal risk factors—the RR, PAR and PAR% were calculated accordingly. The top five factors with a high PAR% had fewer consultations with a health professional, maternal age under 20 or over 35, first parity or parity more than two, a non-professional occupation, and no home ownership.

Table 4 shows the incidence of LBW babies vis-à-vis maternal outcome factors and the most important factor attributed to LBW with a high PAR% was weight gain under 10 kg during pregnancy.

Discussion

The total fertility rate for Thai women between 15 and 49 years of age is 1.7. High parity is not common. The overall incidence of LBW babies in this study was 8.6%. The highest rate (10.4%) was in the Northeast, where most (82.5%) LBW babies had a birth weight between 2 000 and 2 500 g.

The maternal risk factors with a high PAR%, directly attributable to LBWs, was a weight gain less than 10 kg during pregnancy (PAR% = 16.0). The most important biological factors with high PAR% were a maternal age over 35 (PAR% = 16.3) and first or more than two parity (PAR% = 15.5).

Important socioeconomic factors included infrequent consultations with a health professional (PAR% = 16.9), having a non professional occupation (PAR% = 12), and not owning their own home (PAR% = 10.8).

Pregnancy weight gain parallels increasing fetal weight (one quarter of the total gain), enlargement of the uterus, increasing amniotic volume and development of the placenta, so maternal weight gain during pregnancy is a sensitive indicator of fetal growth and easy to monitor. Low weight gain during pregnancy is also potentially correctable through quality prenatal care and food supplementation.

Presently, 80% of pregnant Thai women attend prenatal care. All pregnant women should be trained to assess themselves for weight gain using the graph in the maternal and child health handbook given to all pregnant women at their first prenatal visit. Published research shows a significant link between LBW and pregnancy weight gain: the proportional decrease in the incidence of LBW babies by this factor is 16%--from 8.6 to 7.3 percent. [13,15-17,31,32]

The proportional incidence of LBW can be attributed to mothers over 35 (15.5%), while teenage mothers are more likely to be psychosocially disadvantaged and biologically immature. Older mothers suffer pregnancy complications including LBW more frequently than mothers between 20-30 years of age. It may be that age-related biological changes or a concert of factors bring about the increased risk. [33]

Thailand has had a Family Planning Program since 1970, which aims to prevent too early, too old, too many and too close pregnancy; notwithstanding, women under 20 and over 35 continue pregnancies. Birth spacing for adolescent women and birth control for older women could perhaps be emphasized since these two age-related factors could lower the incidence of LBW from 7.3 to 6.2 percent, as *per* the 9th Health Development Plan.[30]

However, a more sympathetic approach would be close monitoring of pregnancy weight gain (quality prenatal care), which might also help to reduce the incidence of LBWs for first parity. Another factor beyond the control of health services, which might be helpful, is improving the overall quality of life.

In conclusion, to achieve a reduction in the incidence of LBW babies, proactive activities should be considered, *viz.*: 1) ensure proper weight gain during pregnancy, and 2) provide good quality antenatal care to primiparous and middle-age pregnant women.

Acknowledgments

The authors would like to thank the Thailand Research Fund, the Health System Research Office and the Ministry of Public Health for supporting the Prospective Cohort Study in Thai Children and Mr. Bryan Roderick HAMMAN for assistance with the English-language presentation.

References

1. Hack M, Fanaroff AA. Outcomes of children of extremely low birth weight and gestational age in the 1990s. *Semin Neonatal* 2000; **5**: 89-106.
2. Yu VY, Wood C. Perinatal asphyxia and outcome of very low birth weight infants. *Med J Aust* 1978; **2**: 578-81.
3. Stanley FJ, Alberman EV. Infants of very low birth weight I: perinatal factors affecting survival. *Dev Med Child Neurol* 1978; **20**: 300-12.
4. Kelly YJ, Nazroo JY, McMunn A, Boreham R, Marmot M. Birth weight and behavioral problems in children: a modifiable effect? *Int J Epidemiol* 2001; **30**: 88-94.
5. Zubrich SR, Kurinczuk JJ, McDermott BM, McKelvey RS, Silburn SR, Davies LC. Fetal growth and subsequent mental health problems in children aged 4 to 13 years. *Dev Med Child Neurol* 2000; **42**: 14-20.
6. Torrioli MG, Frisone MF, Bonvini L et al. Perceptual-motor, visual and cognitive ability in very low birth weight preschool children without neonatal ultrasound abnormalities. *Brain Dev* 2000; **22**: 163-8.
7. Keller H, Ayub BV, Saigal S, Bar-Or O. Neuromotor ability in 5- to 7- years old children with very low or extremely low birth weight. *Dev Med Child Neurol* 1998; **40**: 661-6.
8. Forssas E, Gissler M, Shvonen M, Hemminki E. Maternal predictors of perinatal mortality: the role of birth weight. *Int J Epidemiol* 1999; **28**: 475-8.
9. Basso O, Olsen J, Christensen K. Risk of preterm delivery, low birth weight and growth retardation following spontaneous abortion: a registry-based study in Denmark. *Int J Epidemiol* 1998; **27**: 642-6.
10. Walraven GE, Mkanje RJ, van Asten HA, van oosmalen J, van Dongen PW, Dolmans WM. The etiology of low birth weight in a rural area of Tanzania. *Trop Med Int Health* 1997; **2**: 558-67.
11. Klufio CA, Amoa AB, Augerea L, Wurr F. A case-control study of singleton low birth weight at the Port Moresby General Hospital. *PNG Med J* 1997; **40**: 136-45.
12. Wessel S, Cnattingius S, Bergstrom S, Dupret A, Reitmaier P. Maternal risk factors for preterm birth and low birth weight in Cape Verde. *Acta Obstet Gynecol Scand* 1996; **75**: 360-6.
13. Orr ST, James SA, Miller CA, et al. Psychosocial stressors and low birth weight in an urban population. *Am J Prev Med* 1996; **12**: 459-66.
14. Sharma R, Synkewecz C, Raggio T, Mattison DR. Intermediate variables as determinants of adverse pregnancy outcome in high-risk inner-city populations. *J Natl Med Assoc* 1994; **86**: 857-60.
15. Rondo PH, Abbotte R, Rodrigues LC, Tomkins AM. The influence of maternal nutritional factor on intrauterine growth retardation in Brazil. *Pediatr Perinat Epidemiol* 1997; **11**: 152-66.
16. Ceesay SM, Prentice AM, Cole TJ, et al. Effects on birth weight and perinatal mortality of maternal dietary supplements in rural Gambia. *BMJ* 1997; **315**: 786-90.
17. Zimmer-Gumbeck MJ, Helfand M. Low birth weight in a public prenatal care program: behavioral and psychosocial risk factors and psychosocial intervention. *Soc Sci Med* 1996; **43**: 187-97.

18. Andres RL, Day MC. Perinatal complication associated with maternal tobacco use. *Semin Neonatol* 2000; **5**: 231-41.
19. Ruijter I, Miller JM. Evaluation of low birth weight in African Americans. *J Natl Med* 1999; **91**: 663-7.
20. Maruoka K, Yagi M, Akazawa K, Kinukawa N, Ueda K, Nose Y. Risk factors of low birth weight in Japanese infants. *Acta Pediatr* 1998; **87**: 304-9.
21. Malik S, Ghidiyal RG, Udani R, Waingankar P. Maternal biosocial factors affecting low birth weight. *Indian J Pediatr* 1997; **64**: 373-7.
22. Jacobson G, Schei B, Hoffman HJ. Psychosocial factors and small for gestational age infants in parous Scandinavian women. *Acta Obstet Gynecol Scand* Suppl 1997; **165**: 14-8.
23. Barros H, Tavares M, Rodrigue T. Role of prenatal care in preterm birth and low birth weight in Portugal. *J Public Health Med* 1996; **18**: 321-8.
24. Cheung YB, Yip SF. Social patterns of birth weight in Hong Kong 1984-1997. *Soc Sci Med* 2001; **52**: 1135-41.
25. Jolly M, Sebire N, Harris J, Robinson S, Regan L. The risk associated with pregnancy in women aged 35 years or older. *Hum Reprod* 2000, **15**: 2433-7.
26. Wergeland E, Strand K, Bordahl P. Strenous working conditions and birth weight, Norway 1989. *Acta Obstet Gynecol Scand* 1998; 77: 263-71.
27. Lao TT, Ho LF. The obstetric complications of teenage pregnancy. *Hum Reprod* 1997; **12**: 2303-5.
28. Karim E, Mascie-Taylor CG. The association between birth weight, sociodemographic variables and maternal anthropometry in an urban sample from Dhaka. *Ann Hum Biol* 1997; **24**: 387-401.
29. Ministry of Public Health, Thailand. The eighth health development plan (1997-2001). Nonthaburi: Ministry of Public Health, 1997.
30. Ministry of Public Health, Thailand. The ninth health development plan (2002-2006). Nonthaburi: Ministry of Public Health, 2001.
31. Hosseini M, Nastaran J. Relationship between pregnancy outcome and maternal BMI and weight gain. *International Congress Series* 2004; **1271** : 380-83.
32. Ehrenberg HM, Dierker L, Milluzzi C, Mereer BM. Low maternal weight, failure to thrive in pregnancy, and adverse pregnancy outcomes. *Am J Obstet Gynecol* 2003; **189** : 1726-30.
33. Yuan W, Steffensen FH, Nielsen GL, Moller M, Olsen J, Sorensen HT. A population-based cohort study of birth and neonatal outcome in older primipara. *Int J Gynecol Obstet* 2000; **68**: 113-8.

Table 1: Incidence rate of LBWs at the four study sites

Sites	Total Live births	Number of LBW	%LBW
Central	750	52	6.93
South	993	83	8.36
Northeast	826	86	10.41
North	753	65	8.63
Total	3 322	286	8.61

Note: Valid cases 3 322, missing 32 = 0.9%

Table 2 Number of LBW by birth weight and mode of delivery

Mode of delivery	Birth weight (G)				
	<1 000	1 000-1 499	1 500-1 999	2 000-2 499	Total (%)
Normal	2	8	33	196	239 (83.8)
Breech	-	1	1	4	6 (2.1)
Forceps	-	-	-	3	3 (1.1)
Vacuum	-	-	-	4	4 (1.4)
C/S	1	1	3	27	32 (11.2)
BBA	-	-	-	1	1 (0.4)
Total	3 (1.0)	10 (3.5)	37 (13.0)	235 (82.5)	285 (100)

Note: C/S = cesarean section
BBA = birth before arrival

Table 3: Incidence of LBWs by maternal exposure to risk factors

Exposure	LBW N	%	RR	PAR	PAR%
1) Socioeconomic status					
1.1. Maternal education (3 294 missing 60 = 1.8%)					
Grade six and lower (1 983)	171	8.62	1.00	0.0000	0.027
Grade seven and upper (1 311)	113	8.62	(0.80,1.26)		
1.2. Maternal occupation (3 310 missing 44 = 1.3%)					
Non-professional (3 178)	275	8.65	1.14	0.0103	12.015
Professional (132)	10	7.58	(0.62,2.09)		
1.3. Family income (3 269 missing 85 = 2.5%)					
Not enough (961)	92	9.57	1.17	0.0041	4.735
Enough (2 308)	189	8.19	(0.92,1.48)		
1.4. Home ownership (3 306 missing 48 = 1.4%)					
No (2 096)	192	9.16	1.19	0.0093	10.843
Yes (1 210)	93	7.69	(0.94,1.51)		
1.5. Marital status (3 306 missing 48 = 1.4%)					
Single parent (80)	9	11.28	1.31	0.0007	0.756
Live together (3 226)	276	8.56	(0.70,2.46)		
1.6. Religion (3 179 missing 175 = 5.2%)					
Islam (671)	58	8.64	1.00	0.0001	0.077
Buddhist (2 508)	216	8.61	(0.76,1.32)		
2) Health service utilization					
2.1. Prenatal care (2 957 missing 397 = 11.8%)					
Seldom (107)	9	8.41	1.06	0.0002	0.219
Often (2 850)	226	7.93	(0.56,2.01)		
2.2. Consultation with health personnel (2957 missing 397 = 11.8%)					
Seldom (2 275)	190	8.35	1.27	0.0135	16.974
Often (682)	45	6.6	(0.93,1.73)		
2.3. Iron tablet intake (2 956 missing 398 = 11.9%)					
Seldom (549)	45	8.2	1.04	0.0006	0.708
Often (2 407)	190	7.89	(0.76,1.42)		
3) Reproductive health and biological characteristics					
3.1. Maternal age (years) (3 306 missing 48 = 1.4%)					
Less than 20 and more than 35(756)	101	13.36	1.85	0.0141	16.298
20-35 (2 550)	184	7.22	(1.47,2.33)		
3.2. Parity (2 965 missing 389 = 11.6%)					
First and 2+ (1937)	169	8.72	1.28	0.0125	15.524
Second (1 028)	70	6.81	(0.98,1.68)		
3.3. Planned pregnancy (2 985 missing 369 = 11.0%)					
No (956)	85	8.89	1.16	0.0039	4.771
Yes (2 029)	156	7.69	(0.90,1.49)		
3.4. Past abnormal obstetric (3 292 missing 62 = 1.8%)					
No (2 622)	226	8.62	1.00	0.0001	0.088
Yes (670)	58	8.66	(0.76,1.32)		
3.5. Maternal height (3 118 missing 236 = 7.0%)					
<145 cm (141)	25	17.73	2.29	0.0045	5.532
≥ 145 (2 977)	230	7.73	(1.57,3.34)		

Table 4: Incidence of LBWs by maternal outcomes

Exposure	LBW N	%	RR	PAR	PAR%
1. Albumin in urine (2 892 missing 462 = 13.8%)					
Yes (99)	11	11.11	1.36	0.0010	1.201
No (2 793)	229	8.2	(0.77,2.40)		
2. Sugar in urine (2 897 missing 457 = 13.6%)					
Yes (11)	3	27.27	3.31	0.0007	0.868
No (2 886)	238	8.25	(1.25,8.75)		
3. Hypertensive disorder (3 123 missing 231 = 6.9%)					
Yes (14)	5	35.71	4.42	0.0012	1.512
No (3 109)	251	8.07	(2.17,9.02)		
4. Bleeding during pregnancy (3 123 missing 231 = 6.9%)					
Yes (41)	5	12.20	1.50	0.0005	0.649
No (3 082)	251	8.14	(0.65,3.43)		
5. Weight gain during pregnancy (2 221 missing 1 133 = 33.8%)					
≥10 kg (1 585)	97	6.12	1.67	0.0117	16.097
<10 kg (636)	65	10.22	(1.24,2.26)		

เอกสารหมายเลข 9.5

Parenting Styles During the First Year of Life and Its Consistency : The Prospective Cohort Study of Thai Children (PCTC) Project

By Dr.Rutja Phuphaibul

Parenting Styles During the First Year of Life and Its Consistency :The Prospective Cohort Study of Thai Children (PCTC) Project

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Introduction

Parenting styles have been emphasized as an important determinant for the future of children. Parenting attitude as well as their interaction with the child were found to be predictive of language and cognitive performance as well as physical predictors such as perinatal complication and health (Pryor and Woodward, 1998). It was also claimed to predict obesity, physical activity and hyperactivity problem in school aged children (Lederman et al.,2004; Morton, Campbell, Santich, and Worsley, 1999; Chen, 2002; McDonal, 1999; Alizadeh and Andries 2002). In adolescent, parenting styles were found to predict fruit and vegetable intake, physical activity, tobacco use, alcohol use, mental health status, risk taking behavior and sexual risk-taking behaviors (Lytle et al., 2003;Patock-Peckham, Cheong, Balhorn and Nagoshi, 2001; O'Byrne, Haddock and Poston, 2002 ; Schmitz, Lytle, Phillips, Murry, Birnbaum and Kubik, 2002; Jackson, 2002; Dwairy, 2004; Radziszewska, Richardson, Dent and Flay, 1996; Miller, Dilorio and Dudley, 2002; Huebner and Howell, 2003) While the most commonly found style was overprotect and reasoning (Gordon, 1999; Morton, Cambell, Santich and Worsley, 1999), the problem of depression was found among those experienced neglect and overprotect parenting styles in male than female (Lloyd and Miller, 1997).

Classification of the parenting styles, however were differed from studies to studies. The classification of parenting styles are varied between 3-8 categories (Moor 1987, ;Bee 1995, Khemmanee et al. 1995, Patock-Peckam, Cheong, Balhorn and Nagoshi, 2001). The styles were mainly focused on the parent's control and responsiveness behavior and attitude. The most commonly use classification were the 4 styles namely overprotect or permissive, control or authoritative, reasoning (democratic or authoritarian), and neglect (Darling 1999). Amidst the classifications, most of them were based on perceived experience during early years and the attitude of parent towards parenting. None came from current direct observation or interview on child rearing practice during the first year of life. It is expected that parenting styles during the first year of life can be identified, with uncertainty that whether it will be changed in the future. Notably, the procedure to verify the validity of the parenting styles have not been fully explored.

The Prospective Cohort Study of Thai Children (PCTC) is a prospective birth cohort study conducted in 5 different regions of Thailand with its cultural diversity started in 2000. One of the main purpose of this PCTC study is to link selected socioeconomic factors and child rearing to the future health outcomes of the children within this birth cohort. The second concern is the finding of the child rearing including parenting styles during the first year of life which emphasis on the validity of the instrument.

Objectives of the Study

The objectives of this study are:

1. To describe the parenting styles of parents and significant care takers in 5 child rearing situations at 6 months including 1). feeding time control and response ,2). foods introducing, 3). assisting when baby try to turn over, 4). sleeping time control and 5). responding to baby cry

2. To determine the consistency of parenting styles among the four categories: 1) authoritarian or control, 2) authoritative or reasoning, 3) permissive or overprotect, and 4) neglecting types in the five situations.

Material and Method

This PCTC study is an observational community-based designed to follow all fetus of 28^{th} to 38^{th} week pregnancy from 4 selected districts in different regions and Bangkok Metropolitan, the capital city, of Thailand. The birth cohort, born over one year period during 2000-2002 in each site, is observed and planed to follow up until the children reach the age of 24. Time of recruitment had varied among sites.

Expected number of children born from each site was 800-900 thus providing a total of about 4,000 children and families. The parents or the primary care takers were interviewed for important data regarding socioeconomic background and their practices to identify parenting styles. In addition, community profile and changes over time would be included in the longitudinal study protocol.

This reported data on child rearing behaviors that reflected parenting styles was collected when the child was 6 months old. The measurement of child rearing practices will be repeated at 5, 10, 5 and 20 years of age. Additionally, this study has been approved from the National Ethical Committee, Ministry of Public Health. All family were clarified all study procedures and possible risk before signing the consent form.

Instrument development

After the systematic review , we adopted concepts used by Darling (1999), because it has been steadily used and could reflect the actual parental respond and demand during caring for the infant. The 4 categories for parenting styles in the study were control, reasoning, overprotect, and neglect. The original questionnaire was initially developed by the researcher team , it was later reviewed and commented by the panel of PCTC committee. Prior to the try put of the questionnaire, it was send to 4 experts in the area of infant care for comment and suggestion. Then it was further developed according to the experts' feedback. The questionnaire was tried out in one district using parents who are not in the study for 10 families. The result from the try out brought suggestion for the further development of the questionnaire.

Finally, the instrument consisted of 5 child rearing situations with 4 items to choose which reflected their actual performances or responses. Those situations were 1). feeding time control and response ,2). foods introducing, 3). assisting when baby try to turn over, 4). sleeping time control and 5). responding to baby cry. In each item, the respond would have a mutually exclusive choice among the 4 parenting styles answers as seen in the appendix. Those mutually exclusive choices for child rearing patterns were : control, reasoning, overprotect and neglect.

The research assistants who collected the data were full time employed recruited from the baccalaureate degree graduate. Using manual guideline, the research assistants were trained and tested until the researchers reliability was insured. During the data collection, supervisory visits were made by the researcher team on the spot. Since the subjects and the research assistant were met both at home and at the hospitals for at least 5 times, acquaintance between the subjects and the research assistant or the interviewers had been well developed prior to the data collection. On the day of data collection, the research assistant made a home visit with prior appointment. After explanation of the objectives of the study, the research assistant asked the questions with neutral tone, and requesting the parents or significant care giver to make the choice for each item. If the answer was indecisive, then the data was treated as the "other" category. All questionnaires were transported to the station where they were checked by the field manager before forwarding them to the data management center.

Statistical analysis

In order to examine the parenting styles in 5 child rearing situations, descriptive statistics with frequency and percentage were employed. To analyze the consistency of categorization of parenting styles in five situation, weighted kappa statistics was used. In brief, for each pair among the five situations, tabulation was done on one behavior against the other for all subjects. The example of cross tabulation between the feeding and sleeping time management situations and weighted table for kappa statistics was shown in Table 1.

Table 1: Example of weighted kappa among 2 situations

Milk time	Sleeping time			
	Control	Reasoning	Overprotect	Neglect
Control	1	0.7	0	0
Reasoning	0.7	1	0.3	0.3
Overprotect	0	0.3	1	0
Neglect	0	0.3	0	1

From the illustrated Table 1 using STATA 8.0, the weighted kappa was 0.089 which is a level of low consistency. Repeating this for all pairs, there are $C_2^5 = 10$ possible pairs composing a kappa matrix.

For frequency analysis, observed combination of choices among all possible combination of 5^5 were tabulated. Those who chose the same style 3 or more times out of the 5 situations were categorized according to the styles they chose.

Results

The characteristics of the families were describing the 5 regions are in Table 2.

Table2: Characteristics of the samples by regions (N=4,088).

Family characteristics	Regions					Total
	Central	South	Northeast	North	Bangkok	
Income per year (U.S.$)	5,153	2,196	2,925	4,366	9,352	4,474
Age of mother	26.9	26.9	26.4	26.7	26.5	26.7
Educational levels					-	
- Illiterate	4.1	5.9	1.2	14.7	0.1	5.3
- Primary	16.7	60.0	61.3	29.4	18.1	47.5
- Middle and high	25.4	26.0	30.1	29.1	32.7	28.4
- Vocational	5.5	4.4	4.6	11.5	20.1	8.6
- Bacc. and higher	3.2	3.5	2.6	14.9	30.6	10.2
Occupations						
- Farmers and farm labor	23.6	56.4	56.4	29.4	0.3	35.9
- Profession/government officers	2.4	2.4	2.5	9.5	14.5	5.8
- Clark and service	23.8	21.9	14.3	23.2	59.0	24.9
- Factory workers and labors	31.8	6.5	15.1	20.1	6.5	15.5
- Unemployed/house wife	18.3	21.8	11.8	17.7	19.6	18.0
Religions						
- Buddhist	99.6	34.1	99.5	87.4	95.6	80.1
- Islam	0.4	65.7	0.4	0.0	2.3	17.3
- Christ	0.0	0.2	0.0	2.1	1.1	0.6
- Animism	0.0	0.0	0.0	10.5	0.0	1.9
- Others/ not spec.	1.3	1.5	1.2	2.2	11.0	3.0

Southern and northern subjects had the lowest income where illiteracy was the most common in the north where 15% of the subjects were hill tribes. Farming was the most common occupation in the northeastern , the north and the south, but not among those in central and Bangkok areas. Although Buddhism was the most predominating among the sample, Islam was the second. Animism among hill tribe people was evident in the northern region.

To examine the pattern, finally the frequency and the Kappa value of each parenting were tabulated as in Table 3 . The findings reveal that 66.80% of parenting styles could be identified using 3 out of 5 criteria, while the most common style were overprotect (42.76%) and reasoning (22.73%). Other 33.19% could not be clearly identify their styles . Table 4 demonstrate the details of repeated patterns of the parenting styles.

As in Table 3 , kappa matrix depicted that kappa values were low. It was indicating low consistency among all situations.

Table 3: Kappa Matrix of parenting styles according to the 5 child rearing situations

Parenting styles	Kappa				
	Milk	Food	Assist	Sleep	Cry
Feed milk	1	-0.0419	-0.0176	0.0891	0.0688
Food introduction	-0.0419	1	-0.0092	0.0219	0.0118
Assist when turn	-0.0176	-0.0092	1	0.0188	-0.0129
Sleep time	0.0891	0.0219	0.0188	1	-0.0111
Response to cry	0.0688	0.0118	-0.0129	-0.0111	1

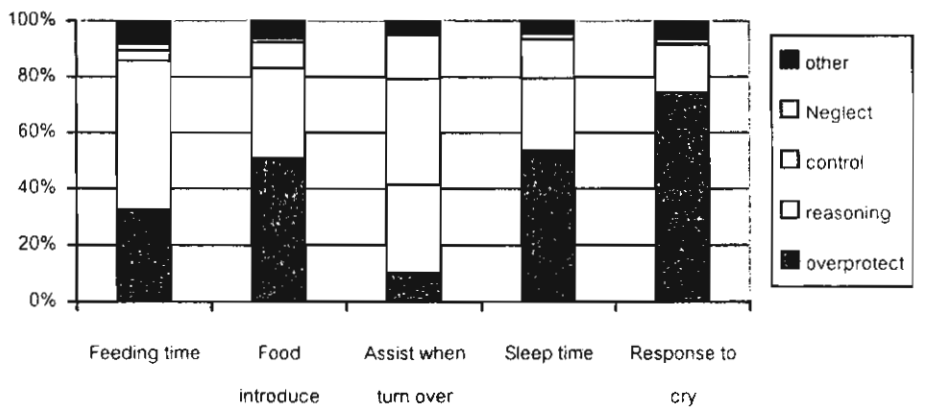
Table 3 shows that overprotection was the most common followed by reasoning. Control and neglecting styles were very few. Over a quarter were the combination of 2 pairs and a random combination. Figure1 shows distribution of style break down by the five child rearing situations. All are consistent with the results in Table 4 that overprotection was the most common style, except "assisting when turn over" where control was the common choice.

Table 4: Repetition of parenting styles patterns (N = 4,088)

Parenting styles	Combination of styles*	n	%	Total n(%)
Overprotect	222xx	1,208	29.55	1,748 (42.7)
	2222x	493	12.06	
	22222	47	1.15	
Reasoning	333xx	721	17.64	929 (22.7)
	3333x	192	4.70	
	33333	16	0.39	
Control	444xx	48	1.17	51 (1.2)
	4444x	3	0.07	
Neglect	111xx	3	0.07	3 (0.07)
Combination of 2 pairs	2233x	714	17.47	1,031 (24.1)
	2244x	171	4.18	
	3344x	101	2.47	
	Others pairs	45	1.09	
Random combination	Non specific	335	25.41	326 (7.9)

*Note : 1 = neglect, 2 = overprotect, 3 = reasoning, 4 = control, 5 = others , x = random

Figure 1 Percentage of parenting styles in each of the 5 situations of child care.



Discussion

We have here by documented that among diversified demographic background of the subjects in this study, the parenting styles according to situations (feeding, food introduction, assisting, sleeping arrangement and responding to baby cry) have a very low consistency. Overprotect parenting style was the most common choice where as reasoning was the second. Assisting when turn over was distinctively different from other condition as "control" was the most common choice. Overprotection was therefore the obvious response, while the child control or parent's demand was not as obvious in this age group.

This study is the first to use kappa analysis for analysis the consistency of parenting styles. The finding shows a low consistency of parenting styles in this study. When examine the consistency of the styles of the same care takers , the kappa values showed that the agreement between each of the styles appeared to be very low. It means that parenting styles during the first year of life seem to be in random patterns. This suggested that the subjects chose different responses in different situations. Although overprotection is the prominent parenting style, but not consistence among the same subject. It does not prevail the consistency of parenting behaviors or respond among 5 situations. As this study is the first to explore parenting styles when the infants were 6 months old , it might suggest that the pattern of parenting styles are not stable as parent are experimenting their infant care practice. They responded differently in different situation. Therefore, further study to predict parenting styles for the infants from there actual performances similar to this study should explore in specific situations. We expect that the parenting styles will be less at random pattern.

The instrument in this study was dissimilar to others studies using uniformed instrument such as Parental Authority Questionnaire (Buri), Maternal Attitude scale, or Parenting Dimensions Inventory (PDI) which are focused on parenting attitude and behaviors in general that effect older child's social adjustment and health behaviors (Rintala Herson and Hudler-Hull, 2000; Mile and Holditech, 1995). In this study, the instrument used in this study focus on parenting styles in 5 situational child care practice. It offers a direct assessment and unlink to any child outcomes , thus it is less subjected to bias at this stage of the study.

As the PCTC project will continue to examine the parenting styles and their effects on the child outcomes physically and psychologically at older age. Its limitation however due to the instrument which expected to be different as the child grows older. Due to the changing needs of the child at later age, the behaviors of caretakers are expected to be changed accordingly. The comparison of parenting styles posts the challenge for future analysis of this prospective cohort study.

References

Alizadeh H. and Andries C. (2002). Interaction of parenting styles and attention deficit hyperactivity disorder in Iranian parents. **Child & Family Behavior Therapy.** 24, 3, 37-52.

Bee H. (1995). **The Growing Child.** New York : Harper Collins Publishers.

Chen J. (2002). **Factors associated with children's health in Taiwan and the United States.** Doctoral Dissertation, University of California, San Francisco.

Darling N. (1999) Parenting style and its correlates. **ERIC Clearinghouse on Elementary and Early Childhood Education,** Champaign, IL. no:ED427896.1999,1-3.

Dwairy M. (2004). Prenting styles and mental health of Arab adolescents in Israel. **Transcultural Psychiatry**. 42, 2, 233-253.

Gordon LC. (1999) **Linking gender differences in parenting to a typology of family parenting styles and adolescent developmental outcomes.** Dissertation (Ph.D.) Iowa state University.

Huebner A.J. and Howell L.W. (2003). Examining the relationship between adolescent sexual risk-taking and perceptions of monitoring, communication, and parenting styles. **Journal of Adolescent Health**. 33, 2, 71-78.

Holden GW. (1997) . **Parents and the dynamics of child-rearing.** Westview Press: Scranton PA.

Hsieh ,1998 Hsieh C. (1998). **Relating parenting styles and children's temperament to behavioral adjustment and academic achievement of Taiwanese Children (China).** Dissertation (Ed.D.).Texas Tech University.

Jackson C. (2002). Perceived legitimacy of parental authority and tobacco and alcohol use during early adolescence. **Journal of Adolescent Health**. 31, 5, 425-432.

Khemmani T. et al. (1995) Principles and model of early childhood development in Thai cultural ways : Selected research findings relating to social context and child transition from home to school . **ERIC Clearinghouse on Elementary and Early Childhood Education.** Champaign, IL. No : ED390574. Paper presented at the Annual Meeting of the American Educational Research Association. (San Francisco, CA, April 18-22,1995).

LeCuyer-Maus and Houch ,2002 Le Cuyer-Maus EA. and Houck GM. (2002). Maternal characteristics and maternal limit-setting styles. **Public Health Nursing** , 5 , 336-344.

Lederman S.A. Akabas S.R., Moore B.J., Bently M.E., Devaney B., Gillman M.W., Kramer M.S., Mennella, J.A., Ness, A. and Wardle A. (2004). Summary of the presentations at the conference on preventing childhood obesity. **Pediatrics**. 114, 1146-1174.

Lloyd C. and Miller PM. (1997). The relationship of parental style to depression and self-esteem in adulthood . **Journal of Nervous & Mental Disease** , 185 ,655-663.

Lytle L.A., Vanell S., Murray D.M., Story M. Perry C. Birbaum A.S. and Kubik M.Y. (2003). Predicting adolescent's intake of fruit and vegetables. **Journal of Nutrition Education and Behaviors.**35, 4, 170-178.

McDonald C. (1999). Parenting style was associated with hyperactivity in school aged boys. **Evidence-Based Nursing**. 2, 1, 25.

Miles M.S. and Holditch-Davis D. (1995). Compensatory parenting : how mother describe parenting their 3-year-old, prematurely born children. **Journal of Pediatric Nursing: Nursing Care of Children and Families.** 10, 4, 243-253.

Miller J.M., Dilorio C. and Dudley W. (2002). Parenting style and adolescent's reaction to conflict: is there a relationship? **Journal of Adolescent Health.** 31, 6, 463-468

Moore SG. (1987) . The role of parents in the development of peer group competence(1987). **ERIC Clearinghouse on Elementary and Early Childhood Education** ,Champaign ,IL no: EG346992. Paper presented at the Annual Meeting of the Biennial Meeting of the Society for Research in Child Development . Baltimore, MD , April 23-26,1987.

Morton H., Campbell K., Santich B. and Worsley A. (1999). Parental strategies and young children's snacking behavior : a pilot study. **Australian Journal of Nutrition and Dietetics.** 56, 4, 215-220.

Naksusuk S. 2001. **Factors Effecting Mental Health of Middle School Student Under Bangkok Metropolitan: Case Studies.** Master Thesis (Social Work). Huo Cheaw University.

O'Byrne K.K., Haddock C.K. and Poston W.S.C. (2002). Parenting style and adolescent smoking. **Journal of Adolescent Health.** 30, 6, 418-425.

Patock-Peckham J.A., Cheong J., Balhorn M.E. and Nagoshi C.T. (2001). A social learning perspective: a model of parenting styles, self-regulation, perceived drinking control, and alcohol use and problems. **Alcoholism: Clinical and Eperimental Research.** 25, 9, 1284-1292.

Pryer J. and Woodward L. (1998). **Families and parenting. From Child to Adult.** Oxford University Press : London, 246-258.

Radziszewska B. Richardson JL. Dent CW. and Flay BR. (1996). Parenting style and adolescent depressive symptoms, smoking, and academic achievement : ethnic, gender, and SES differences. **Journal of Behavioral Medicine.**19 , 289-305.

Rintala D.H., Herson L. and Hudler-Hull T. (2002). Comparison of parenting styles of person without spinal cord injury and their children's social competence and behavior problems. **Journal of Spinal Corf Medicine.** 23, 4, 244-256.

Lloyd and Miller . 1997

Russek LG.and Schwartz GE. (1997) . Perception of parental rearing predict health status in midlife : a 35-year follow up of the Harvard Mastery of Stress study. **Psychosomatic Medicine**, 59 , 144-14.

Schmitz K.H., Lytle L.A., Phillips G.A., Murry D.M., Birnbaum A.S. and Kubik M.Y. (2002). Psychosocial correlates to physical activity and sedentary leisure habits in young adolescents: the teen eating for energy and nutrition at school study. **Preventive Medicine.** 43, 2, 266-78

Appendix

Parenting styles when the infants were at 6 months questionnaires

1. How do you usually manage the milk feeding time for the baby?	Styles
a. Feed almost all the time, do not Want the baby to get hungry. b. Approximate the feeding time every 3-4 hours, but also observe the baby's hungriness. c. Set the feeding time table every 3-4 hours and always feed accordingly. d. No specific timetable, feed according to my convenience.	a. Overprotect or permissive b. Reasoning or authoritative or democracy c. Control or authoritarian d. Neglect
2. What did you do when the baby refused new kinds of food ?	Styles
a. Up to the baby whether he would eat Or not . b. Played with the baby and try to give new kind of food as much as possible. c. Forced the baby to take the food, or to make his month open. d. No need to bother. It is no big deal.	a. Overprotect or permissive b. Reasoning or authoritative or democracy c. Control or authoritarian d. Neglect
3. When the baby is trying to turn over, what did you do?	Styles
a. Always used hand to support and assist when the baby was turning. b. Let the baby do by himself. Can use hand to prevent bumping. c. Watch the baby doing the turn without help, so the baby will be strong d. No need to bother. The baby can do it naturally.	a. Overprotect or permissive b. Reasoning or authoritative or democracy c. Control or authoritarian e. Neglect
3. How do you usually manage the bed Time at night for the baby?	Styles
a. No specific bed time. Allow sleeping any anytime. It up to the baby. b. Approximate the bedtime to put the baby to bed . c. Always put the baby to sleep according to the timetable. d. No specific bed time. It's up to my convenience.	a. Overprotect or permissive b. Reasoning or authoritative or democracy c. Control or authoritarian d. Neglect
5. When the baby is fussy or crying, what do you always do?	Styles
a. Pick the baby up immediately, do not let him cry. b. Comfort the baby and try to see what cause his crying to help. c. Scold or hit the baby to stop the baby cry. d. Let him cry, the baby will stop crying finally.	a. Overprotect or permissive b. Reasoning or authoritative or democracy c. Control or authoritarian d. Neglect

Exposure to environmental tobacco smoke among infants in southern Thailand : A study of urinary cotinine

By Dr. Wanaporn Anuntaseree

Exposure to environmental tobacco smoke among infants in southern Thailand : A study of urinary cotinine

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Abstract

Objective: To determine the extent and determinants of environmental tobacco smoke exposure among infants in The-pa district in southern Thailand.

Design: Cross-sectional survey including interviewing information from the parents and measurement of urinary cotinine, a metabolite of nicotine, from all eligible infants.

Setting/ Participants: The infants aged 1 year, in The-pa district, Songkla, who were conducted under the Prospective Cohort Study Of Thai Children (PCTC).

Results: Of 774 infants enrolled in this study, 73.9 % were reported to have environmental tobacco smoke exposure in household. Number of smokers in household ranges from 1- 7 persons. Prevalence of father and mother smoking were 86.5 and 0.5% respectively. Urinary cotinine were detectable in 40.3 % of infants which mean $\pm$ SD of 251.3$\pm$ 562.5 ng/mg creatinine, median of 61.8, and range of 2.2 - 6431.0 ng/mg creatinine. Factors significantly associated with detectable utinary cotinine in infants were maternal smoking (p=0.032), father's age of less than 25 and greater than 35 years old (p 0.016), and father's education of less than grade 6 (P=0.032).

Conclusion: Theses results demonstrate the high prevalence of infants exposure to household smoking and high prevalence of detectable urinary cotinine. These measurements provide an objective and quantitative baseline against which cotinine measurements in future can be compared.

Key words: smoking, cigarette, tobacco, cotinine, nicotine

Introduction

Background

Environmental tobacco smoke exposure or passive smoking is defined as inhaling other people's cigarette smoke. Such exposure was previously considered as only a nuisance rather than a health hazard, however, many recent studies demonstrated the relationship with acute and chronic health effects among non-smokers. These effects include respiratory and cardiovascular systems. Several studies have shown that exposure to cigarette smoke during the first year of life significantly increase an infant's risk or developing pneumonia or bronchitis. Additionally, some studies have suggested that passive smoke exposure in children can lead to the development of recurrent respiratory symptoms such as cough, wheezing and recurrent respiratory infection as well as decrease in pulmonary function[1-11]

In the pass decade many studies have investigated the health effects of passive exposure to tobacco smoke in non smoking children. Most studies have relied on questionnaire measures of parental smoking as the indicator of exposure. Such measures ignore exposure by people from outside the household, the extent in which parents smoke in the presence of the child, and other modifying factors such as the ventilation of the room. These studies may therefore have underestimated the real effect of passive smoking in children. Biochemical measures allow recent exposure to be estimated directly.[12-13]

Cotinine, a metabolite of nicotine, is the best biochemical marker for quantifying passive exposure to smoke. It is specific to tobacco, has a half life of about 20 hours, and can be detected at low concentrations by gas-liquid chromatography. Salivary or urinary concentrations are comparable with those in the blood and provide a non-invasive way of measuring passive smoke exposure.[12-15]

Objectives

This study set out to assess the extent and determinants of exposure to environmental tobacco smoke in the Thai infants in southern Thailand.

Subjects and methods

Subjects :

Study population is the infants in the-pa district, Songkhla who were conducted under the Prospective Cohort Study of Thai Children (PCTC).

Methods

Data for this study are collected as part of a larger study of the PCTC. Well-trained data collectors make visits and interview the mothers of all eligible infants at their homes when the infants are approximately 1 year old. Information obtained include parent's education, occupation, house size, household crowding and household smoking in the same room as the infant's presence in the preceeding week. Urine samples for cotinine and creatinine are obtained from all infants on the consecutive week.

Statistical analysis

The association between several potential predictors of environmental tobacco smoke exposure and urinary cotinine is investigated by comparing geometric means and medians among groups by analysis of variance; a test for trend examined the presence of an increasing or decreasing urinary cotinine level across various categories

Results

From the 1,076 infants eligible to participate in the study, 774 provided urine samples. These 774 infants were enrolled in this study. Of these, 73.9 % were reported to have environmental tobacco smoke exposure in household during the week before the data collection. Number of smokers in household ranged from 1- 7 persons. Prevalence of father, mother and grand-father smoking during the infant's presence were 86.5, 0.5 and 21.4% respectively. Urinary cotinine were detectable in 312 (40.3%) infants with mean $\pm$ SD of 251.3$\pm$ 562.5 ng/mg creatinine, median of 61.8, and range of 2.2 - 6431.0 ng/mg creatinine. The association between several potential predictors of environmental tobacco smoke exposure and urinary cotinine was investigated. As the urinary cotinine levels did not distribute normally, comparing geometric means and medians could not be done. Therefore, the percentage of infants, who had detectable urinary cotinine, was compared with those who had not. The potential predictors of environmental tobacco smoke exposure were as follow: environmental smoke exposure in the home, number of smokers, parental smoking, parental age, education, religion and occupation (Table 1). Factors significantly associated with detectable utinary cotinine in infants were maternal smoking ($p=0.032$), father's age of less than 25 and greater than 35 years old ($p=0.016$), and father's education of less than grade 6 ($P=0.016$).

Table 1. Detectable urinary cotinine in infants according to variables

Variables	Detectable urinary cotinine		P
	No	Yes	
Sex			
Male	56.3	43.7	0.041*
Female	63.6	36.4	
Smoking in home			
No	57.5	42.5	0.48
Yes	60.4	39.6	
Number of smoker in home			
0	57.9	42.1	0.372
1	61.9	38.1	
>1	56.0	44.0	
Father smoke			
No	65.8	34.2	0.298
Yes	59.5	40.5	
Mother smoke			
No	60.7	39.3	0.032*
Yes	0	100	
Grand father smoke			
No	60.4	39.6	0.957
Yes	60	40	
Father's age			
≤25	54.4	45.6	0.016*
26-30	64.7	35.3	
31-35	65.9	34.1	
>35	53.2	46.8	
Father's religion			
Buddhist	62.8	37.2	0.217
Muslim	58.1	41.9	
Father's education (years)			
≤ 6	56.5	43.5	0.032*
> 6	64.6	35.4	
Mother's education (years)			
≤ 6	59.6	40.4	0.909
> 6	60.0	40.0	
Father's occupation			
Manual	59.8	40.2	0.809
Non-manual	58.6	41.4	
Mother's occupation			
Manual	59.2	40.8	0.692
Non-manual	60.7	39.3	

Discussion

This study is the first study reported about urinary cotinine level and its determinants in the sample of infants in Thailand. We found that 40 % of infants had detectable urinary cotinine. Only few studies reported about cotinine level in children and its determinants. A study from the United States population by the Third National Health and Nutrition Examination Survey, 1988 to 1991 revealed that of children aged 2 months to 11 years, 43% lived in a home with at least 1 smokers. Serum cotinine levels indicated more widespread exposure to nicotine. Of non-tabacco users, 87.9% had detectable levels of serum cotinine.[14] Our study has shown lower prevalence of detectable urinary cotinine when compared to that study. The difference in findings may be due to the difference age of study population. The older children might expose to environmental smoke from both in household and non-household sources. The prevalence of detectable urinary cotinine in our study was also lower than that from the study in infants in north calorina.[16] We have demonstrated that factors significantly associated with detectable urinary cotinine in infants were maternal smoking, father's age of less than 25 and greater than 35 years old, and father's education of less than grade 6. These findings were similar to other study in a sample of 1,072 children and adolescents in Italy, during 1990-1991.[13] The urinary cotinine level increased with a decreasing level of parternal education and with an increasing index of household crowding. They demonstrated a strong relation between passive exposure to smoking and the amount of maternal and parternal self-reported smoking. We did not find the association of urinary cotinine with father smoking, this may be due to the date of urine collection were not the same week of interviewing. As urine samples were collected at 1-4 weeks after interviewing, the correlation with exposure to father smoking could not be found.

Previous study in Thailand, the prevalence of smoking is consistently high. The National Statistics Office, office of the Prime Minister, conducted household surveys in the whole country in 1976, 1981, 1986, 1988, 1993 and found that the numbers of smokers were 8.6, 9.7, 10.3, 10.1 and 13.9 million persons, respectively.[17-18]

In Southern Thailand, Jaravechsarn W. had performed a cross-sectional study to determine the relationship between passive smoking and acute respiratory infection in 729 infants aged 2-6 months in an urban area of Hat-Yai and found that the prevalence of cigarette smoking in the home was 52%.[19] This suggested that prevalence of smoking in the home had increase from 1996 to 2004, as we found that 73.9% of our study population were reported to have environmental tobacco smoke exposure.

Conclusion

We demonstrated the high prevalence of infant exposure to household smoking and high prevalence of detectable urinary cotinine. Factors significantly associated with detectable utinary cotinine in infants were maternal smoking, father's age of less than 25 and greater than 35 years old, and father's education of less than grade 6.

Suggestion

Data on the extent and determinants of exposure to environmental tobacco smoke in the Thai infant will provide prevalence information for risk assessment and public health prevention strategies, identify population subgroups at increased risk for exposure and provide baseline exposure levels that can be compared with future Thai infant population levels to evaluate the effectiveness of interventions aimed at reducing environmental tobacco smoke exposure.

Urinary cotinine measurements provide an objective and quantitative baseline against which cotinine measurements in future surveys can be compared, to monitor trends in population exposure to tobacco smoke and to assess the effectiveness of public health actions to reduce smoking and exposure to environmental tobacco smoke.

References

1. Pedreira FA, Guandolo VL, Feroli EJ, Mella GW, Weiss IP. Involuntary smoking and incidence of respiratory illness during the first year of life. Pediatrics 1985;75:594-597.
2. Greenberg RA, Bauman KE, Strecher VJ, Keyes LL, Glover LH, Haley NJ, Stedman HC, Loda FA. Passive smoking during the first year of life. Am J Public Health 1991;81:850-853.
3. Wright AL, Holberg C, Martinez FD, Taussig LM, Group Health Medical Associates. Relationship of parental smoking to wheezing and non wheezing lower respiratory tract illnesses in infancy. J Pediatr 1991;118:207-214.
4. Fergusson DM, Horwood LJ, Shannon FT, Taylor B. Parental smoking and lower respiratory illness in the first three years of life. J Epidemiol Community Health 1981; 35:180-184.

5. Neuspiel DR, Rush D, Butler NR, Golding J, Buur PE, Kurzon M. Parental smoking and post-infancy wheezing in children : A prospective cohort study. Am J Public Health 1989;79:168-171.

6. Mascola MA, Vunakis HV, Tager IB, Speizer FE, Hanrahan JP. Exposure of young infants to environmental tobacco smoke : Breast-feeding among smoking mothers. Am J Public Health 1998;88:893-896.

7. Oddoze C, Dubus JC, Badier M, Thirion X, Pauli AM, Pastor J, Bruguerolle B. Urinary cotinine and exposure to parental smoking in a population of children with asthma. Clin Chemistry 1999;45:505-509.

8. Margolis PA, Keyes LL, Greenberg RA, Bauman KE, LaVange LM. Urinary cotinine and parental history (questionnaire) as indicators of passive smoking and predictors of lower respiratory illness in infants. Ped Pulm 1997;23:417-423.

9. Wang X, Wypij D, Gold DR, Speizer FE, Ware JH, Ferris BG, Dockery DW. A longitudinal study of the effects of parental smoking on pulmonary function in children 6-18 years. Am J Respir Crit Care Med 1994;149:1420-1425.

10. Chilmonczyk BA, Salmun LM, Megathlin KN, Neveux LM, Palomaki GE, Knight GJ, Pulkkinen AJ, Haddow JE. Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. N Engl J Med 1993;328:1665-1669.

11. Bek K, Tomac N, Delibas A, Tuna F, Tezic HT, Sungur M. The effect of passive smoking on pulmonary function during childhood. Postgrad Med J 1999;75:339-341.

12. Cook DG, Whincup PH, Jarvis MJ, Strachan DP, Papacosta O, Bryant A. Passive exposure to tobacco smoke in children aged 5-7 years : individual, family and community factors. BMJ 1994;308:384-389.

13. Deli'Orco V, Forastiere F, Agabiti N, Corbo GM, Pistelli R, Pacifici R, Zuccaro P, Pizzabiocca A, Rosa M, Altieri I, Perucci CA. Household and community determinants of exposure to involuntary smoking : A study of urinary cotinine in children and adolescents. Am J Epidemiol 1995;142:419-427.

14. Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR. Exposure of the US population to environmental tobacco smoke. JAMA 1996;275:1233-1240.

15. Pattishall EN, Strope GL, Etzel RA, Helms RW, Haley NJ, Denny FW. Serum cotinine as a measure of tobacco smoke exposure in children. AJDC 1985;139:1101-1104.

16. Greenberg RA, Bauman KE, Glover LH, Strecher VJ, Kleinbaum DG, Haley NJ, Stedman HC, Fowler MG, Loda FA. J Pediatr 1989;114:774-780.

17. Anonymous. Survey of health and smoking in 1991. Bangkok : National Statistics Office, Office of the Prime Minister, 1993.

18. Nitayarumphong S, Saelim S, Thernsirikunchai L, Ativantichayapong S, Sangvanis N, Petcharoen N. Bangkok : Health Planning Division, Office of the Permanent Secretary for Public Health, MOPH, 1992:22-23.

19. Jaravechsarn W. Cigarette smoking in the home and acute respiratory infection in infants aged 2-6 months. Thesis in Epidemiology. Prince of Songkla University 1996.

เอกสารหมายเลข 9.7

Late introduction of complimentary food causes stunting at ly-old : the Prospective Cohort study of Thai Children

By Dr.Ladda Mo-suwan

Draft - 19 Dec 2004

Title Page

Title Late introduction of complimentary food causes stunting at 1 y-old: the Prospective Cohort study of Thai Children

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Introduction

Linear growth is one of the two most important indices of child well-being widely used throughout the world. In developing countries, about 200 million or 40% of children under 5 years of age are affected by retarded linear growth or stunted (height- for-age z scores <2.0) (1). Lengths of these children falter early after birth, and most stunting occurs before the age of 3 years and persists into adulthood (2) Recent study revealed that stunting could be carried over from mother to her offspring (3) The consequences of becoming and remaining stunted include increased risk of infection and mortality, delays in cognitive development, decreased work capabilities, and possible relation to cardiovascular morbidities in adults (4-7). Given that potential for catch-up growth among stunted children is limited after age 2 years, intervention to eliminate stunting should therefore implement prenatally and very early in life (8,9).

The etiology of stunting is multi-factorial including micronutrient deficiencies, inadequate protein intake, prenatal nutrition, repeated infections, maternal height, poor maternal-child interaction, and maternal literacy (10-13). As most stunting begins early during infancy, food given to infants probably plays an important role.

The Prospective Cohort Study of Thai Children is the first longitudinal study of children in Thailand started in 2001. It plans to follow children from about the 28th week of gestation to 24 years of age. Weights and lengths of subjects as well as infant feeding pattern were collected. This report investigates the effect of timing of feeding complimentary foods on linear growth status at the age of 1 year.

Subjects and Methods

Study sites

The Prospective Cohort Study of Thai Children (PCTC) is an observational community-based study designed to follow different regional birth cohorts from about the 28th week of gestation to 24 years of age. A sample of the Thai population was achieved in four cohorts from a chosen district in each of the four regions and a hospital-based sample in

Bangkok, the capital. Eligible sites were accessible year-round, averaged between 800 and 900 births annually, and, had a hospital director (and associates) committed to long-term management. The four sites were: Panomtuan district of Kanchanaburi province in the west, Thepa district of Songkhla province in the south, Kranuan district of Khon Kaen province in the northeast, and Muang district of Nan province in the north. Details of this study were reported elsewhere (14)

Subjects

All pregnant women who resided or planned to raise their children in the study area for at least 5 years were recruited. They were identified in the beginning of the third trimester by community survey or by retrieval from registries of the Antenatal Care Clinics (ANC) of hospitals, health centers and private clinics. Each birth cohort included births over a one-year period. Starting dates for each cohort were staggered to spread enrolments over a two year period so the babies were born between October 15, 2000 and September 14, 2002.

Collection of data

The study families were initially visited at 28^{th}-32^{nd} week gestation, then at birth, 1 month, 3 months, 6 months and 12 months. Interviews were arranged at home and in hospital as *per* the study's protocol. Linguistic and religious variations in the presentation of information occur between the regions of Thailand, so research assistants (RA) were selected and trained to be sensitive yet incisive. Monitoring and quality control were set up from the beginning to ensure the study's reliability and validity. Re-interviews were conducted on a random sub-sample to check for consistency and any interviewer bias.

Anthropometric measurements

Birth weights and lengths were retrieved from hospital records. In the case of delivery at home, weight and length of the newborn was collected by the RA within 3 days after birth using a digital Tanita® Scale and a portable length board. The RA measured subjects again at 6 months ($\pm$ 1 week) and 12 months ($\pm$ 1 week) of age by home visit. In this report, only length measurements were included in the analysis.

Length status was classified using the WHO Reference data for the height of children (15) as stunting if having length-for-age (LFA) and gender less than median -2SD).

Infant feeding

Type of milk feeding was collected by interviews at 21 (± 3) days, 6 months and 12 months of age. Milk feeding status was categorized at each 6 months period as predominant breast feeding, bottle feeding, or mixed feeding (breast feeding + bottle feeding).

In order to avoid a recall bias, a specific calendar with easy-to-apply stickers was given to mothers (or the primary caregivers).for keeping a record of ages at which complimentary foods were introduced (Figure 1). Timing of introduction of complimentary foods was categorized as <4 months, 4-6 months or >6 months for rice, cereal, banana, papaya, egg yolk, meat/pork, poultry, fish and liver. For the whole egg, it was grouped as <7 months, 7-9 months or >9 months.

Maternal and family data

Maternal age, education level, occupation, and height and baseline family data were collected by interviews around 28^{th}-32^{nd} week gestation. Education levels were categorized as illiterate, primary level, secondary level, or university level. Classification of maternal occupation was modified from that of the National Statistical Office into 5 groups namely farmer, laborer, service or business, professional, or housewife. Family income was grouped into 3 levels: group 1 – the poorest (income < P25), group 2 – the middle group (income between P25 and 75), group 3 – the well-to-do group (income > P75).

Statistical analysis

Distributions of explanatory variables among children of each length status category were examined. Associations between variables and length status category were explored using ANOVA for continuous variables and chi-square test for categorical variables.

Determinants of stunting were explored using logistic regression analysis. All main-effect variables were initially included, and the model reduced using a strategy of stepwise elimination of variables not significantly contributing (i.e. $p > 0.05$) to the fit of the model, as indicated by change in the log-likelihood of successive hierarchical models. The variables,

study site and sex, were retained in the model regardless of their statistical significance. Analyses were performed using STATA 7.0. (15)

Ethics

The National Ethics Committee, the Ministry of Public Health, approved the study. The study was explained to the registered families then they were invited to participate. Verbal permission was obtained followed by written informed consent.

Results

Of the 4,245 live-born infants recruited into the initial cohort, 60 infants were twins. The present report included only the 4,185 singletons. Length was recorded at birth for 3,889 infants (92.9 %), at 6 months for 3,496 infants (83.5 %), and at 12 months for 4,017 infants (95.9 %). The Table 1 shows that prevalence of stunting declined from birth to 6 months but rose to a higher level at 12 months. For children who had length measured at both 6 and 12 months, 6.7% of children not stunted at 6 months had become stunted at one year old, while only 1.07% of those stunted at 6 months had grown out of it at one year.

The distribution of maternal and family variables between the stunted and the non-stunted groups are shown in the Table 2. Significant differences among length status categories were evident for several variables. Compared to the non-stunted group, stunted children had mothers who were shorter, had a significantly lower level of education, and were more in the agricultural and labor sectors but less in the professional and service sectors. Stunted subjects more commonly belonged to families in the lowest income quartile, to Muslim parents or Spirit worshippers.

The Table 3 shows that stunted subjects were fed only breast milk in a significantly greater proportion than the non-stunted group both during the first six months and the second six months. Comparing to the non-stunted children, rice, fruit (banana or papaya), egg yolk, liver, fish, meat or pork, vegetable were fed to stunted children at a significantly older age.

A logistic regression model was developed by a stepwise backward elimination of variables from a saturated main-effects model incorporating all variables listed in the Tables 2 and 3. After eliminating of non-significant variables, variables remaining in the model were type of milk given from 7 to 12 months, timing of feeding liver and meat/pork, maternal education level, and family income. These factors could explain variance of being stunted at 1 y-old by 14%. The relative risk ratios for each level of these variables are shown in the Table 4.

Compared to breast milk, feeding bottle milk during the second half of the first year reduced risks for stunting by 36%. Early introduction of animal protein, liver, reduced the risk by 4 folds. Likewise, late introduction of meat/pork increased risks by 150%. The risk of being in the families of lowest income quartile (RR = 2.09, 95% confidence interval = 1.64 - 2.68) was no longer significant after adjusted for other factors. On the contrary, being in the families in the uppermost income quartile has become a significant risk.

The risk factors for stunting including type of milk feeding, age at introduction of meat/pork, and maternal education level were also found to be the risk factors for becoming stunted as shown in the Table 5. Direction of association was also similar.

Discussion

Points to be discussed:

- Comparing prevalence and timing of stunting in this cohort with studies from other developing countries e.g. WHO Collaborative study 2002 → early faltering of weight and length as early as 3 months, high incidence of stunting in the Asian countries....
- Breast-fed is shorter → a well-known phenomenon due to using mixed fed growth standard (no appropriate reference for the breast-fed at the moment), however findings from developed countries e.g. European group showed comparable growth → health and nutrition of lactating mothers are the key issue

- Importance of complimentary foods especially animal protein (a good source of protein and multiple micronutrients – Zn, Fe) to linear growth of infants → citing studies from the longitudinal studies in Kenya, supplemental feeding studies.
- Consequences of stunting to health and development → importance of early eradication stunting by good quality complimentary foods.

Table 1 Mean length and length status of the PCTC cohort of singleton infants over the first year of life

Anthropometric Variables	Overall (4,185)	Study site					*P*-value[1]
		PT (779)	TP (1,056)	KN (860)	Nan (771)	BKK (719)	
Mean length ± sd							
At birth	*n = 3,889*	*n = 745*	*n* = 970	*n* = 786	*n* = 755	*n* =633	0.000
	49.4 ± 2.4	49.8 ± 2.4	49.7 ± 2.5	48.8 ± 2.2	48.6 ± 2.3	49.9 ± 2.0	
6 months	*n* =3,496	*n* =690	*n* =912	*n* =774	*n* =675	*n* =445	0.000
	65.9 ± 2.6	66.5 ± 2.5	65.7 ± 2.5	65.8 ± 2.4	64.8 ± 2.5	67.0 ± 2.4	
12 months	*n* =4,107	*n* =747	*n*=1,038	*n* =833	*n* =743	*n* =656	0.000
	73.7 ± 3.2	74.2 ± 3.1	73.1 ± 3.2	73.7 ± 3.1	72.9 ± 3.1	74.9 ± 3.06	
Stunting, %							
At birth	4.6	3.6	4.6	5.3	6.9	1.9	0.000
6 months	3.8	1.2	3.7	3.5	8.6	1.4	0.000
12 months	8.8	4.8	11.9	7.3	15.1	3.1	0.000
Change of length status from 6 to 12 months %[2]							0.000
Becoming stunted	6.70	4.69	9.78	5.09	9.45	1.9	
Still stunted	2.70	0.44	2.75	2.22	6.60	0.95	

Still not stunted	89.53	94.28	86.59	91.38	82.01	96.68
Becoming not stunted	1.07	0.59	0.88	1.31	1.95	0.47

Note: PT = Panomtuan, TP = Thepa, KN = Kranuan, BKK = Bangkok; [1] ANOVA for the continuous variable and chi-square test for the categorical variables.

Table 2 Maternal and family characteristics of the singleton infants by length status

Maternal and family characteristics	Length status		χ^2 *P*-value
	Stunted	Not-stunted	
Maternal age (year), %	*n =352*	*n = 3,665*	0.447
<20	14.20	11.51	
20-<30	51.99	53.86	
30-<40	30.68	30.75	
>=40	3.87	3.13	
Maternal height, mean ± sd	*n = 347* 151.4 ± 6.3	*n = 3,613* 155.6 ± 5.7	0.000
Maternal education level, %	*n =352*	*n = 3,665*	0.000
Illiterate	15.91	4.27	
Primary level	53.98	46.96	
Secondary level	26.70	37.87	
University level	3.41	10.62	
Maternal occupation, %	*n =352*	*n = 3,640*	0.000
Agricultural	33.24	22.66	
Professional	1.70	6.24	
Laborer	34.09	27.55	
Service/business	15.06	25.60	
Housewife	15.91	17.94	
Order of pregnancy, %	*n =352*	*n = 3,665*	0.239
1	26.70	30.59	
2	29.55	29.63	
>= 3	43.75	39.78	

Maternal and family characteristics	Length status		χ^2
	Stunted	Not-stunted	*P*-value
Family income, %	*n* =352	*n* = 3,665	0.000
Q1	40.06	23.08	
Q2 + Q3	40.91	49.41	
Q4	19.03	27.50	
Religion, %	*n* = 351	*n* = 3,666	0.000
Buddhism	60.68	80.47	
Islam	27.92	17.29	
Christians	1.14	0.58	
Spirit	10.26	1.63	

Table 3 Feeding of the PCTC singleton infants during the first year of life

Type of feeding	Length status		χ^2 *P*-value
	Stunted	Not-stunted	
Milk feeding from birth to 6 months	*n =346*	*n = 3,572*	0.000
Breast milk only	55.78	41.10	
Bottle feeding only	3.18	6.61	
Mixed breast and bottle	41.04	52.30	
Milk feeding from 7 to 12 months	*n =346*	*n = 3,572*	0.000
Breast milk only	42.98	29.22	
Bottle feeding only	18.42	35.86	
Mixed breast and bottle	38.60	34.92	
Age at introduction of rice	*n =344*	*n = 3,561*	0.009
Before 4 months	30.52	37.63	
During 4-6 months	60.76	56.50	
After 6 months	8.72	5.87	
Age at introduction of banana or papaya	*n =347*	*n = 3,589*	0.000
Before 4 months	41.79	57.20	
During 4-6 months	46.11	38.12	
After 6 months	12.10	4.68	
Age at introduction of egg yolk	*n =345*	*n = 3,539*	0.000
Before 4 months	8.99	14.52	
During 4-6 months	69.57	71.21	

Type of feeding	Length status		χ^2 *P*-value
	Stunted	Not-stunted	
After 6 months	21.45	14.27	
Age at introduction of whole egg	*n =346*	*n = 3,465*	0.724
Before 7 months	30.06	30.71	
During 7-9 months	47.69	49.61	
After 9 months	22.25	19.68	
Age at introduction of liver	*n =346*	*n = 3,501*	0.000
Before 4 months	0.87	3.91	
During 4-6 months	39.60	49.76	
After 6 months	59.54	46.33	
Age at introduction of fish	*n =345*	*n = 3,483*	0.028
Before 4 months	1.45	1.46	
During 4-6 months	80.58	85.85	
After 6 months	17.97	12.69	
Age at introduction of poultry	*n =345*	*n = 3,470*	0.153
Before 4 months	1.74	0.98	
During 4-6 months	74.49	78.62	
After 6 months	23.77	20.40	
Age at introduction of meat/pork	*n =345*	*n = 3,485*	0.000
Before 4 months	1.74	2.09	
During 4-6 months	72.17	82.98	
After 6 months	26.09	14.92	

Type of feeding	Length status		χ^2 *P*-value
	Stunted	Not-stunted	
Age at introduction of vegetable	*n =346*	*n = 3,518*	0.000
Before 4 months	2.60	6.91	
During 4-6 months	51.16	63.53	
After 6 months	46.24	29.56	

Table 4 Adjusted relative risks for factors associated with stunting*

Factor	Relative Risk	95% confidence interval	*P*-value
Type of milk fed from 7 to 12 months			
Breast milk only	1		
Bottle only	0.64	0.44 - 0.94	0.024
Mixed breast and bottle	0.99	0.74 - 1.32	0.930
Age at introduction of liver			
Before 4 months	0.20	0.05 - 0.86	0.031
During 4-6 months	1		
After 6 months	1.28	0.98 - 1.67	0.067
Age at introduction of meat/pork			
Before 4 months	1.91	0.77 - 4.71	0.162
During 4-6 months	1		
After 6 months	1.49	1.09 - 2.05	0.014
Family income			
Group 1 (Q1)	1.27	0.95 - 1.70	0.108
Group 2 (Q2 + Q3)	1		
Group 3 (Q4)	1.64	1.13 - 2.39	0.009
Maternal education level			
Illiterate	1.31	0.85 - 2.03	0.215
Primary level	1		
Secondary level	0.77	0.57 - 1.06	0.112
University level	0.40	0.19 - 0.81	0.011

* All relative risk ratios are adjusted for sex, length status at birth, study site, and maternal height. Pseudo R^2 of the model is 0.15. Number of cases included in the regression analysis is 3,433.

Table 5 Adjusted relative risks for becoming stunted during the second half of the first year*

Factor	Relative Risk	95% confidence interval	*P*-value
Type of milk fed from 7 to 12 months			
Breast milk only	1		
Bottle only	0.64	0.41 - 0.99	0.049
Mixed breast and bottle	1.00	0.73 - 1.39	0.985
Age at introduction of meat/pork			
Before 4 months	1.08	0.32 - 3.61	0.905
During 4-6 months	1		
After 6 months	1.73	1.20 - 2.47	0.003
Maternal education level			
Illiterate	1.16	0.68 - 1.96	0.588
Primary level	1		
Secondary level	0.73	0.51 - 1.05	0.086
University level	0.35	0.14 - 0.89	0.029

* All relative risk ratios are adjusted for sex, length status at birth, study site, and maternal height. Pseudo R^2 of the model is 0.11. Number of cases included in the regression analysis is 2,982.

References

1. de Onis, M., Frongillo, E.A., BlÖssner. M. (2000) Is malnutrition declining? An analysis of changes in levels of child malnutrition since 1980. Bull. World Health Organ. 78:1222-1233.

2. Shrimpton R, Victora CG, de Onis M, Lima RC, BlÖssner M, Clugston G. (2001) Worldwide timing of growth faltering: Implications for national interventions. Pediatrics 107:E75.

3. Stein AD, Barnhart HX, Wang M, Hoshen MB, Ologoudou K, Ramakrishnan U, Grajeda R, Ramirez-Zea M, Martorell R. (2004) Comparison of linear growth patterns in the first three years of life across two generations in Guatemala. Pediatrics 113:e270-e275. URL: http://www.pediatrics.org/cgi/content/full/113/3/e270.

4. Ibrahim MM, Wall S, Persson LA. (1998) The impact of short stature on child morbidity in a rural African community. Ann. J. Trop. Pediatr. 18:145-154.

5. Dowdney, L., Skuse, D., Morris, K., Pickles A. (1998) Short normal children and environmental disadvantage: a longitudinal study of growth and cognitive development from 4 to 11 years. J. Child Psychol. Psychiatry 39:1017-1029.

6. Sub-Committee on Nutrition. (1997) Stunting and young child development. In: Third Report on the World Nutrition Situation. United Nations Administrative Committee on Coordination/ Sub-Committee on Nutrition, Geneva, Switzerland.

7. Forsen T, Eriksson J, Qiao Q, Tervahauta M, Nissinen A, Tuomilehto J. (2000) Short stature and coronary heart disease: a 35-year follow-up of the Finnish cohorts of The Seven Countries Study. J. Intern. Med. 248:326-332.

8. Golden MH. (1994) Is complete catch-up possible for stunted malnourished children? Eur. J. Clin. Nutr. 48 (Suppl 1):S58-71.

9. Martorell HR, Khan LK, Schroeder DG. (1994) Reversibility of stunting epidemiological findings in children from developing countries. Eur. J. Clin. Nutr. 48 (Suppl 1):S45-57.

10. Espo M, Kulmala T, Maleta K, Cullinan T, Salin ML, Ashorn P. (2002) Determinants of linear growth and predictors of severe stunting during infancy in rural Malawi. Acta. Pediatr. 91:1364-1370.

11. Frongillo EA, Jr, de Onis M, Hanson KMP. (1997) Socioeconomic and demographic factors are associated with worldwide patterns of stunting and wasting of children. J. Nutr. 127:2302-2309.

12. Hernandez-Diaz S, Peterson KE, Dixit S, Hernandez B, Parra S, Barquera S, Sepulveda, J. & Rivera, J.A. (1999) Association of maternal short stature with stunting in Mexican children: common genes vs common environment. Am. J. Clin. Nutr. 53:938-945.

13. Waterlow, J.C., SchÜrch, B. (1994) Causes and Mechanisms of linear growth Retardation. Eur. J. Clin. Nutr. 48:S1-S216.

14. Choprapawan, C., Mo-suwan, L., Phupaibool R, Sa-Ngha P, Thinkamrob B. Nimnuan C, et al. Prospective Cohort Study of Thai Children (PCTC): design and significant first year findings.

15. World Health Organization. (1978) Reference data for the weight and height of children.

Pattern and causes of perinatal death in the Prospective Cohort Study of Thai Children (PCTC) : the importance of stillbirth

By Dr.Ladda Mo-suwan

TITLE PAGE

Title **Pattern and causes of perinatal deaths in the Prospective Cohort Study of Thai Children (PCTC): the importance of stillbirth**

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Keywords perinatal death, Prospective Cohort of Thai Children, stillbirth, early neonatal death

SUMMARY

Up-to-date data on the cause of perinatal death are essential for planning effective maternal and child health policies. An active surveillance of pregnant women from 28 weeks gestation was carried out to determine the magnitude and investigate some causes of perinatal death in four districts of Thailand as part of the Prospective Cohort Study of Thai Children. A semi-structured verbal autopsy questionnaire and/or review of medical records for the hospitalized cases by two pediatricians and one neonatologist were used to independently extract the cause of death. A total of 3,525 pregnant women were registered from July 2000 to March 2002. The total number of babies born alive was 3,529 including 28 pairs of twins. The perinatal mortality rate (PNMR) was 10.69 per 1,000 total births, the stillbirth rate was 6.75 per 1,000 births, and the early neonatal mortality rate was 3.97 per 1,000 live-births. Birthweight specific PNMR were 307.6, 22.5, 4.6, 4.3 per 1,000 births for the birthweight below 1,500 g, 1,500 to 2,499 g, 2,500 to 3,499 g, and 3,500 g and over, respectively. It was found that congenital anomalies were important causes of early neonatal death with anencephaly accounting for two-thirds. About 61.5 % of the perinatal deaths were agreed to by the pediatricians and neonatologist as preventable. About 90% of the preventable stillbirths occurred in the antepartum period. In conclusion, the surveillance system of the present study revealed stillbirth as a major contribution to perinatal mortality. Though the PNMR in this prospective population-based study was not high, about two-thirds of them could have been prevented by appropriate antepartum and intrapartum care.

Introduction

Infant mortality rate is one of the important indices of a nation's health condition. In recent decades, with the advancement of medical care and public health service, post-neonatal deaths in most countries have declined dramatically. As in the United States, deaths in the first month of life consequently account for about two-thirds of all infant deaths.[1] In order to reduce the perinatal mortality rate, more reliable statistics are needed to identify potentially preventable causes. They are also essential to indicate those areas of health care service that are most in need of improvement.

In Thailand, the Ministry of Public Health has routinely calculated mortality rates by using data retrieved from birth certificates and death certificates. Like most other developing countries,[2-3] registration of births and deaths is incomplete and the data is of poor quality, especially in rural and remote areas. Not surprisingly, the infant death rate (26.1 per 1,000 live-births) from the 4th national survey of population change[4] in 1994-5 was five times that of the vital statistics report of the same period (5.2 per 100 live-births).[5] Discrepancies in these figures were due to under-registration of neonatal deaths during the first two weeks of life. Most deaths at this age take place at home. Causes of deaths given by non-medical persons can be inaccurate. Furthermore, the present system could not reveal the exact magnitude and causes of pregnancy termination due to under-reporting of abortion and stillbirths. This unreliable information may reduce the effectiveness of intervention programs to prevent perinatal deaths.

The Prospective Cohort Study of Thai Children is the first longitudinal study of children in Thailand. It plans to follow children from about the 28th week of gestation to 24 years of age. This report describes the pattern and causes of perinatal deaths of the subjects, the data being collected by an active surveillance system.

Materials and Methods

Study sites

The Prospective Cohort Study of Thai Children (PCTC) is an observational community-based study designed to follow all fetuses of 28th to 38th week gestation from 4 selected districts in 4 different geographical regions and Bangkok Metropolitan, the capital city of Thailand. Details of this study were reported elsewhere.[6] This report is based on the data of the four districts only, including Panomtuan district of Kanchanaburi province in the west, Thepa district of Songkhla province in the south, Kranuan district of Khon Kaen province in the northeast, and Muang district of Nan province in the north. Data from the Bangkok sub-sample are not reported here.

Subjects

All pregnant women who resided or planned to raise their children in the 4 selected districts and who expected to give birth during the defined period of one calendar year were registered. They were identified in the beginning of the third trimester by community survey or by retrieval from registries of the Antenatal Care Clinics (ANC) of hospitals, health centers and private clinics. Registration of subjects in the project was introduced sequentially. The first pregnant woman was enrolled in the Panomtuan district in July 2000 and the first child was born on 15 October 2000. The last child of these four districts was born in Nan on 19 March 2002.

Case identification

Baseline data of pregnant women and their families were collected by interview at 28-36 weeks gestation. Home visits to these women were carried out weekly from 36 weeks onwards. Delivery outcome was retrieved from the records of health centers or district hospitals. In the case of delivery at home or termination of pregnancy before term or infant death, their families or village leaders would report directly to the PCTC branch office at each site.

Fetal death of 28 – 40 weeks gestation was defined as a stillbirth, while death during the first seven days of life was defined as an early neonatal death. Perinatal death included stillbirths and early neonatal deaths.

Investigation into the cause of death

Verbal autopsy is commonly used to investigate the cause of death in population based studies where most deaths occur at home or in the peripheral hospital and necropsy or physical autopsy cannot be performed. Symptoms and signs prior to death were collected by an interview of the parents or caretaker.[7] In the present study, a semi-structured questionnaire was designed to cover common causes of early neonatal death described in the recent national report.[8]

The attending pediatrician of each site interviewed parents and caretakers at about two weeks after the event to allow time for the families to recover from their grief, but not later than one month. For the hospitalized cases, information was also retrieved from the hospital records. Causes of stillbirth and early neonatal death were then extracted from the verbal autopsy questionnaire by two pediatricians (L.M. and S.I.) and one neonatologist (P.C.). In the case of a non-agreement, a consensus was reached at a meeting of the group. Causes of death were defined based on Wigglesworth's classification[9] into "stillborn", "congenital malformation", "neonatal asphyxia", "immaturity" and "specific causes". The classification was modified by further categorization of stillbirth into "obstetric problems", "medical problems of mother", "macerated fetus", and "congenital anomalies"; and replacing the specific causes category in the early neonatal death with individual specific diseases.

At the end of the questionnaire, the raters were asked to conclude whether death of that case was preventable.

Ethical clearance

This study was approved by the National Ethical Committee, Ministry of Public Health. Families were fully informed of all study procedures and any possible risks before signing an informed consent form.

Data analysis

The stillbirth rate (SBR) and perinatal mortality rate (PNMR) were calculated per 1,000 total births while early neonatal mortality rates (ENMR) were calculated per 1,000 live-births. Statistical analysis was performed using STATA version 7.0.

Results

A total of 3,525 pregnant women were registered in the study. Twenty-four pregnancies ended with stillbirths. The SBR was therefore 6.75 per 1,000 births. The total number of babies born alive was 3,529 including 28 pairs of twins. Fourteen infants did not survive the first week of life. The ENMR was 3.97 per 1,000 live-births and the PNMR was thus 10.69 per 1,000 total births. Details of perinatal mortality by study sites are shown in the Table 1.

Insert Table One about here

From the total of 38 deaths, 35 cases (92%) were completely investigated; the other 3 cases included only the diagnosis. About one third (39%) of deaths occurred from 12 pm to 6 am. Seventy-two per cent died in the hospital and the male to female ratio was 1.4:1 and 3.3:1 for stillbirth and early neonatal death, respectively. Weight was recorded in 25 cases of deaths (65.8%). A breakdown of the perinatal mortality by birthweight shows an extremely high rate of death in the under 1,500 g group. The detail is shown in the Table 2.

Insert Table Two about here

Causes of stillbirth

The most common cause of stillbirth, as shown in the Table 3, was obstetric problems of which intrauterine asphyxia contributed to about one-half. Macerated fetuses and congenital anomalies accounted for 20.8% and 12.5% of stillbirths, respectively. A case of diaphragmatic hernia was diagnosed intrauterine by ultrasound but the parents refused referral for delivery and corrective operation at the University hospital. Details of maternal characteristics were not available in three cases of stillbirth.

Insert Table Three about here

Causes of early neonatal death

The most common cause of death in the first seven days of life was congenital anomalies of which two-thirds were anencephaly. As shown in the Table 4, perinatal asphyxia, meconium aspiration, and neonatal sepsis were next in importance. Hypothermia and neonatal sepsis were both considered the consequence of child neglect.

Insert Table Four about here

Maternal characteristics

The pregnancies of 1.1% of women aged 20 years and older ended with perinatal deaths while for younger mothers the figure was 0.4%. The PNMR of the mothers who did laboring work or who were farmers were around 2 times that of those who did non-laboring work. All babies of mothers in professions or who had university education were born alive and survived the first week. The PNMR of the primiparous mothers was two-thirds that of the multiparous mothers. With regard to antenatal care, 3.8% of women did not register for the

service or did, but visited only once. Of those who paid frequent or regular visits, the PNMR was 9.5 per 1,000 births whereas the non-registered group had a PNMR of 17.1. The cesarean section rate of the perinatal death group was 20% while that of the non-death group was 16.5%. Due to the small number of deaths, it was not appropriate to perform any statistical analysis to investigate whether any of these maternal factors were statistically significant risk factors in perinatal death. The perinatal mortality rate by maternal characteristics is shown in the Table 5.

Insert Table Five about here.

Preventable death

In the opinion of the three pediatricians, almost two-third (61.5%) of perinatal deaths reported in this study could have been prevented by appropriate antenatal or intrapartum care. As shown in the Table 6, the stillbirths rated as preventable included macerated fetuses (2 cases), antepartum hemorrhage (2 cases), maternal sepsis (1 case), diabetes mellitus (1 case), intrauterine asphyxia (1 case), and difficult labor (1 case). Early neonatal deaths rated as preventable were neonatal sepsis (2 cases), perinatal asphyxia (1 case), hypothermia (1 case), and meconium aspiration syndrome (1 case).

Insert Table Six about here.

Discussion

This report described the results of a study of an active surveillance of perinatal death in a community-based setting. Stillbirths accounted for 70% of death in the perinatal period. Causes of death could be concluded in 87.5% of stillbirths studied and in all of the early neonatal deaths. Almost two-thirds of the deaths were judged to have been preventable.

Though the PNMR of the PCTC sample was much lower than that of Turkey (34.9 per 1,000 births) from a prospective study[10] and that of rural China (69 per 1,000 births) from a retrospective cohort study in 1999,[11] this PCTC figure showed no improvement over the country's figure of five years ago (Table 7). Besides it was still higher than the rate of developed countries such as that of the UK in 2001[12] (8.0 per 1,000 births), even though the UK figure represented fetal deaths at or over 24 weeks gestation whereas that of the PCTC included deaths at an older age (at or over 28 weeks gestation). Given that the national figure was derived from provincial hospitals which have higher than usual complicated referral cases, the present figure from this community-based study should have been lower.

The surveillance system of the present study revealed stillbirths as a major contributor to perinatal mortality. This is in contrast to findings of other developing countries.[10-11] A prospective study from Turkey[10] revealed that stillbirths contributed to about a half of perinatal deaths, while in a retrospective cohort study from rural China[11] it accounted for about one-third. In addition, the ENMR of the present study was one-fourth of Turkey of 17.2 per 1,000 live-births[10] and only one-tenth of rural China of 46.0,[11] yet slightly over the UK 2001 figure of 2.7.[12] Though the early neonatal deaths of the PCTC sample has declined by about two-thirds compared to the previous country survey,[8] it should be noted that it was 2.5 times that of the reported figure of 1.6 per 1,000 live-births from the death registration system in 2001.[13] To further reduce the perinatal mortality rate in Thailand, attention should therefore be focused on reducing the stillbirths.

About 40% of stillbirths in the PCTC sample were rated as preventable by the three pediatricians. Almost all of preventable fetal deaths in this study could have been prevented by higher-quality antenatal obstetric and medical care and the appropriate mode of delivery. Timely cesarean section could have been rescued some of these cases. Although the cesarean section rate (20% in the perinatal death group and 16.5% in the non death group) in this study was above the optimum rate of 15% as recommended by the WHO for the high-risk area,[14] the needed cases might not have had the chance to receive an appropriate care.

Communication and the information provided for expectant mothers during the antenatal care visits are thus very important. It is noted that the educational levels of mothers was often very limited; half received only primary education and 6.3% were illiterate. If these deaths could have been prevented, the SBR would approach the UK 2001 figure of 5.3.[12]

The findings of this study showed that deaths of the preterm and low birthweight babies were 4 to 60 times more prevalent than that of the normal weight group. Prevention of preterm delivery as well as upgrading the quality of care of the very low birthweight (VLBW) infants would substantially reduce the perinatal death rate. Since the advanced technology of VLBW care is not yet available country-wide for Thailand, emphasis should be on prevention. As was shown in a randomized controlled study in rural Gambia, maternal dietary supplementation and eradication of anemia can reduce stillbirth and early neonatal death rates by about half as well as decrease the low birthweight prevalence by 40%.[15] In the PCTC sample, anemia was not a significant risk factor but weight gain during pregnancy of less than 10 kg and maternal height below 145 cm increased the risk almost three times (Isranurug S, personal communication). Thus, intervention to prevent low birthweight in a setting like that of the PCTC study should include nutrition education and/or supplementation for pregnant women plus improvement of nutrition of pre-pubertal girls.

Like the mortality pattern of western countries two decades ago,[16] congenital anomalies were important causes of early neonatal deaths in this study. The most common anomaly in this study was anencephaly, one form of neural tube defect. To prevent this condition, the United Kingdom MRC Vitamin study Research Group has recommended folic acid supplementation for pregnant women as well as women in the reproductive age.[17] Despite this, the importance of folic acid supplementation has not yet been recognized as a public health issue in Thailand.

Although the PNMR in this sample was not high, about two-thirds of them could have been prevented by appropriate antepartum and intrapartum care. Intervention should be

strategically planned to serve the at risk groups especially the low education mothers. Improvement of the antenatal care should highlight the importance of nutrition and vitamin supplement as well as self care.

Acknowledgements

The authors express their heartfelt gratitude to the families who participated in the study. The research was supported by the Thailand Research Fund, the Health System Research Institute, the Ministry of Public Health and the World Health Organization.

References

1. Luke B, Williams C, Minogue J, Keith L. The changing pattern of infant mortality in the US: The role of prenatal factors and their obstetrical implications. *International Journal of Gynecology and Obstetrics* 1993;40:199-212.
2. Kitange H, Swaui AB, Masuki G, Kilima PM, Alberti KG, McLarty DG. Perinatal mortality in rural Tanzania. *World Health Forum* 1994;15:82-84.
3. Walraven GE, Mkanje RJ, Roosmalen J, van Dongen PW, Dolmans WM. Perinatal mortality in home births in rural Tanzania. *European Journal of Obstetrics and Gynecologic Reproductive Biology* 1995;58:131-134.
4. National Statistical Office, Office of the Prime Minister. *Report of the 1994-1995 Survey of Population Change*. Bangkok: Aksorn Thai Press Ltd, 1997.
5. Ministry of Public *Health. Public Health Statistics A.D. 1994-1995*. Bangkok: Veteran Welfare Press. 1995.
6. Choprapawon C, Mo-suwan L, Phupaibool R, Sa-Ngha P, Thinkamrob B, Nimnuan C, *et al*. Prospective Cohort Study of Thai Children (PCTC): design and significant first year findings. *Under consideration for publication*, 2004
7. Baqui AH, Black RE, Arifeen SE, Hill K, Mitra SN, Al Sabir A. Causes of children deaths in Bangladesh: results of a nationwide verbal autopsy study. *Bulletin of the World Health Organization* 1998; 76: 161-171.
8. Amornwichet P, Kanchana S, Kullert N. *Infant mortality in Thailand 1995-1996*. A report of Bureau of Health Promotion, Department of Health, Ministry of Public Health. 1999.
9. Wigglesworth JS. Monitoring perinatal mortality. A pathophysiological approach. *Lancet* 1980;2:684-686.
10. Erdem G. Perinatal mortality in Turkey. *Paediatric and Perinatal Epidemiology* 2003;7:17-21.

11. Wu Z, Viisainen K, Wang Y, Hemminki E. Perinatal mortality in rural China: retrospective cohort study. *British Medical Journal* 2003;327:1319-1322.

12. Office for National Statistics. Deaths 1921-2002.Childhood, infant and perinatal mortality: Stillbirth and infant death rates by age at death. Retrieved May 29, 2004 from http://www.statistics.gov.uk/StatBase/xsdataset.asp?vlnk=7981&Pos=1&ColRank=1&Rank=176

13. Ministry of Public Health. Number and Rates of Deaths under 7 days per 1,000 Livebirths by Sex and Regions: 1994-2001. Retrieved Aug 19, 2004 from http://203.157.19.191/2.2.14.xls

14. World Health Organization. Appropriate technology for birth. *Lancet* 1985;2:436-437.

15. Ceesay SM, Prentice AM, Cole TJ, Foord F, Poskitt EME, Weaver LT, *et al*. Effects on birthweight and perinatal mortality of maternal dietary supplements in rural Gambia: 5 year randomized controlled trial. *British Medical Journal* 1997;315:786-790.

16. Edouard L, Alberman E. National trends in the certified causes of perinatal mortality, 1968 to 1978. *British Journal of Obstetrics and Gynaecology* 1980; 87: 833-838.

17. MRC Vitamin Study Research Group. Prevention of neural tube defects: results of the Medical Research Council Vitamin Study. *Lancet* 1991;338:131-137.

Table 1 Births, stillbirths, early neonatal deaths, and perinatal death rates by study sites

Study Sites	Liveborns	Stillbirths (per 1,000 births)	Early neonatal deaths (per 1,000 live-births)	Perinatal deaths (per 1,000 births)
Overall	3,529	24 (6.75)	14 (3.97)	38 (10.69)
Panomtuan	796	8 (9.95)	6 (7.53)	14 (17.41)
Thepa	1,071	8 (7.41)	3 (2.80)	11 (10.19)
Kranuan	872	4 (4.56)	3 (3.44)	7 (7.99)
Nan	790	4 (5.03)	2 (2.53)	6 (7.56)

Table 2 Perinatal mortality by birthweight[a]

Birthweight	Perinatal death, n/N	Perinatal mortality rate (per 1,000 births)
Under 1,500 g	4/17	307.6
1,500 – 2,499 g	7/311	22.5
2,500 – 3,499 g	12/2623	4.6
3,500 g and over	2/472	4.3

[a]3,423 out of 3,525 cases had birthweight data.

Table 3 Causes of stillbirths

Causes	Number of cases (%)	Details
Obstetric problems	9 (37.5)	Intrauterine asphyxia (5)[a], antepartum hemorrhage (3), velamentous cord (1)
Macerated fetus	5 (20.8)	
Congenital anomalies	3 (12.5)	Anencephaly (1), diaphragmatic hernia (1), Thanatophoric dysplasia (1)
Medical problems of mothers	2 (8.3)	Diabetes mellitus (1), maternal sepsis (1)
Difficult labor	1 (4.2)	
Others	1 (4.2)	Hydrops fetalis
Inconclusive	3 (12.5)	

[a]Figure in parenthesis is the number of cases.