Original article

Risk factors of early and late onset preeclampsia among Thai women

Rozanna Fang[a,*], Antoinette Dawson[a,*], Vitool Lohsoonthorn[a,b], Michelle A. Williams[a,c]
[a]Department of Epidemiology, Multidisciplinary International Research Training Program, University of Washington School of Public Health and Community Medicine, Seattle, WA 98195, USA; [b]Department of Preventive and Social Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok 10330, Thailand; [c]Center for Perinatal Studies, Swedish Medical Center, Seattle, WA 98104, USA.

Background: Little research has been conducted to specifically identify risk factors of early and late onset preeclampsia among Thai women.
Objective: To examine risk factors of the early and late onset of preeclampsia among Thai women.
Methods: A case-control study of 150 preeclampsia cases with an equal number of normotensive controls was conducted among women who delivered live-born singleton infants at King Chulalongkorn Memorial Hospital, Rajavithi Hospital, and Police General Hospital in Bangkok, Thailand between July 2006 and November 2007. Multivariable logistic regression procedures were used to calculate odds ratios (OR) and 95% confidence intervals (CI) of potential risk factors associated with preeclampsia.
Results: Pre-pregnancy body mass index ≥ 30 kg/m^2 (OR=5.25, 95%CI: 1.80, 15.32) and failure to use prenatal care services (OR=6.37, 95%CI: 1.26, 32.27) were associated with increased risk of preeclampsia. OR's of similar magnitude were observed when risk factors of early and late onset preeclampsia were assessed separately.
Conclusion: Advanced maternal age, obesity, and no utilization of prenatal care were covariates identified as risk factors for preeclampsia.

Keywords: Epidemiology, hypertension, preeclampsia, pregnancy, risk factors.

Preeclampsia is a pregnancy-specific hypertensive disorder that significantly affects maternal morbidity and mortality worldwide [1]. It occurs in 5-7% of all pregnancies, and is a leading cause of maternal deaths in developing countries [2, 3]. Preeclampsia is also an important determinant of perinatal morbidity and mortality, in part, because of its considerable association with preterm delivery and intrauterine growth restriction (IUGR) [1, 3].

Preeclampsia has been the subject of extensive research in recent years. A number of studies have identified advanced maternal age, nulliparity, high maternal body mass index (BMI), and pre-existing hypertension or diabetes as likely risk factors. Study results have been consistent across continents for these particular risk factors [4-6]. However, findings have been far less consistent for putative risk factors such as maternal race/ethnicity and socioeconomic status [5, 7, 8]. Furthermore, little research has been conducted to specifically identify risk factors of the early and late onset preeclampsia among Thai women.

In this study, we sought to identify risk factors of preeclampsia within this Southeast Asian population. This study was undertaken as an initial step towards better understanding the etiology of preeclampsia within a population that is undergoing rapid social, economic, and demographic changes.

*Contributed equally to this work.
Correspondence to: Dr. Vitool Lohsoonthorn, Department of Preventive and Social Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok 10330, Thailand. E-Mail: vitool@gmail.com

Materials and methods
Study population and data collection

A case-control study using one control for each case of preeclampsia was conducted among women who delivered live born singleton infants at King Chulalongkorn Memorial Hospital, Rajavithi Hospital, and Police General Hospital, Bangkok, Thailand between July 2006 and November 2007. Cases were women with a confirmed diagnosis of preeclampsia. Preeclampsia cases were identified by daily monitoring of all new admissions to antepartum, labor and delivery wards of participating hospitals. Of the 158 eligible cases approached, 154 (97.5%) agreed to participate in the study. Controls were women who delivered an infant at term ($\geq$37 weeks gestation) and who had no evidence of preeclampsia or any hypertensive disorder during pregnancy. Controls were selected from the same hospital of delivery as cases. An eligible control, delivering immediately after a case patient, was approached and recruited for the study. Of the 161 controls approached, 154 (95.7%) agreed to participate in the study.

After excluding women found to have pre-gestational hypertension or diabetes mellitus, 150 preeclampsia cases and 150 controls remained for analysis. All participants provided informed consent. The research protocol was reviewed and approved by Ethics Committees of the Faculty of Medicine, Chulalongkorn University, Rajavithi Hospital, Police General Hospital, and the Institutional Review Board, Division of Human Subjects Research, University of Washington.

After obtaining informed consent, enrolled participants were asked to participate in a 45-minute in-person interview in which trained research personnel used a structured questionnaire to elicit information regarding maternal socio-demographic, lifestyle, medical and reproductive characteristics. Participants', and delivery medical records, and prenatal medical records were also reviewed by trained obstetric research nurses who used a standardized abstraction form. Information abstracted from medical records included participants' pre-pregnancy weight, height, blood pressure, pregnancy complications, and condition of the newborn.

Analytical variable specification

Preeclampsia: The diagnosis of preeclampsia was made using the current American College of Obstetricians and Gynecologists (ACOG) guidelines [2]. These guidelines define preeclampsia as sustained pregnancy-induced hypertension with proteinuria. Hypertension was defined as sustained blood pressure readings of $\geq$140/90 mmHg (with reading taking place $\geq$6 hours apart). ACOG defines proteinuria as urine protein concentrations of $\geq$30 mg/dL (or 1+ on a urine dipstick) on two or more random specimens collected $\geq$ four hours apart. It has recently been suggested that early ($\leq$34 completed weeks gestation) and late (>34 completed weeks gestation) onset preeclampsia may have different etiologies. We therefore categorized preeclampsia cases according to gestational age at onset using the cut-point suggested by von Dadelszen et al. [9].

Other covariates. Covariates considered in this analysis included maternal socio-demographics and behavioral characteristics including maternal age, marital status, educational attainment, employment status, cigarette smoking, and alcohol consumption during pregnancy. Also considered were maternal reproductive and medical histories including parity, prior history of hypertensive disorders during pregnancy, history of abortion, maternal height, weight, and infant gender. Parity was reported as the number of previous pregnancies lasting more than 22 weeks gestation. Pre-pregnancy body mass index was calculated as weight (in kilogram) divided by the square of height (in meter).

Statistical analysis

Multivariable logistic regression procedures were employed to calculate odd ratios (OR) of potential risk factors associated with preeclampsia. Confidence intervals, at the 95% level were also reported for each unadjusted and adjusted OR. Confounding was assessed by entering potential cofounders into a logistic regression model one at a time, and by comparing the adjusted and unadjusted ORs. Final logistic regression models included covariates that altered unadjusted ORs by at least 10% [10]. We considered the following covariates as possible confounders in these analyses: maternal age, parity, marital status, and maternal educational attainment. Backward logistic regression modeling procedures combined with the change-in-estimate approach were used to select the final models reported in this manuscript. In order to determine the extent to which the risk factors of preeclampsia are most strongly associated with early onset disease (as cited by von Dadelszen et al. [9]), we repeated analyses for the early onset preeclampsia (n=29) and

the late onset preeclampsia (n=121) cases. All analyses were completed using SPSS version 16.0 statistical software.

Results

Socio-demographic and behavioral characteristics of preeclampsia cases (in aggregate and according to early/late onset) and normotensive controls are presented in **Table 1**. We observed evidence of a strong and statistically significant increased risk of preeclampsia among women who were 30-34 years of age (OR=2.40, 95%CI: 1.19, 4.83) and ≥35 years of age (OR=2.75, 95%CI: 1.35, 5.61). Women who were 30-34 years of age (OR=2.23, 95%CI: 1.06, 4.71) and ≥35 years of age (OR=2.69, 95%CI: 1.27, 5.70) were more likely to have late onset preeclampsia compared with women 25-29 years of age. Associations of similar magnitude were observed when analyses were limited to the early onset preeclampsia cases. However, these associations did not reach statistical significance. We observed no evidence of associations of preeclampsia with maternal educational attainment, marital status, or employment during pregnancy. Women who reported smoking or alcohol consumption during pregnancy had a reduced risk of preeclampsia; however, these associations did not reach statistical significance.

Table 2 summarizes reproductive and medical history characteristics of preeclampsia cases and normotensive controls. Apparently, women with a prior history of pregnancy-associated hypertensive disorders had a statistically significant increased risk of preeclampsia (OR=10.17, 95%CI: 1.26, 82.02). This association was particularly strong for early onset preeclampsia (OR=21.45, 95%CI: 2.19, 210.57). A family history of hypertension was also associated with increased preeclampsia risk (OR=2.12, 95%CI: 1.06, 4.22). Women with a family history of hypertension had a 2.14-fold increased risk of late onset of preeclampsia (95%CI: 1.04, 4.40). An association of similar magnitude was observed for early onset preeclampsia (OR=2.01, 95%CI: 0.66, 6.09), though because of the small sample size the association did not reach statistical significance.

We observed a positive linear trend of preeclampsia risk in relation to maternal pre-pregnancy BMI (**Table 3**). Women who were underweight (<18.5 kg/m^2) had a 62% reduced risk of preeclampsia (OR=0.38, 95%CI: 0.18, 0.81) as compared with their counterparts who had a normal

pre-pregnancy BMI (18.5-24.9 kg/m^2). Conversely, obese women (≥30 kg/m^2) had a 4.76-fold increased risk of preeclampsia (OR=4.76, 95%CI: 1.73, 13.12) compared with women who had a normal pre-pregnancy BMI. Associations of similar directions and magnitudes were observed when analyses were repeated for early and late onset preeclampsia. We observed a modest, though non-significant increased risk of preeclampsia for women who received no prenatal care during pregnancy (OR=2.66, 95%CI: 0.79, 8.91).

Results from three multivariable logistic regression models are summarized in **Table 4**. Patterns of associations were similar regardless of whether we assessed preeclampsia in aggregate or stratified into early and late onset subgroups. However, adjusted ORs were less precise (as reflected by relatively wider 95%CI's) for subgroup analyses of early onset preeclampsia. For all models, young (<20 years) and advanced (30-34 years and ≥35 years) maternal age were risk factors of preeclampsia. Also, a significant linear trend between pre-pregnancy BMI and preeclampsia risk was observed for all subcategories of preeclampsia. Women who did not receive prenatal care during the index pregnancy had 6.37-fold increased risk of preeclampsia as compared with those women who initiated prenatal care during the first trimester (95%CI: 1.26, 32.27). An association of similar magnitude was observed for late onset preeclampsia cases (OR=7.19, 95%CI: 1.36, 38.11). Prior history of hypertensive disorders during pregnancy (OR=7.83, 95%CI: 0.88, 69.44) and a positive family history of hypertension (OR=1.92, 95%CI: 0.87, 4.23) were positively associated with preeclampsia risk, though these covariates did not reach statistical significance.

Discussion

This case-control study indicates that advanced maternal age, pre-pregnancy obesity status, and no utilization of prenatal care are associated with a significantly increased risk of preeclampsia in Thai women. These results are concordant with findings from earlier studies that evaluate the risk factors of preeclampsia in other populations [4, 5, 11, 12]. Moreover, results from our analyses of early and late onset preeclampsia case groups support the position held by Huppertz [3] who recently noted no clear difference between the early and late onset preeclampsia.

Table 1. Odds ratios (OR) and 95% confidence intervals (CI) for socio-demographic and behavioral characteristics of preeclampsia cases and normotensive controls, Bangkok, Thailand, 2006-2007.

Covariates	Controls (n = 150) n	Controls %	Preeclampsia (n = 150) n	Preeclampsia %	Unadjusted OR	(95%CI)	Early Onset Preeclampsia (n = 29) n	Early OR	(95% CI)	Late Onset Preeclampsia (n = 121) n	Late OR	(95% CI)
Maternal age (years)												
<20	11	7.3	14	9.3	1.91	(0.78, 4.67)	3	2.10	(0.47, 9.43)	11	1.86	(0.72, 4.81)
20-24	47	31.3	35	23.3	1.12	(0.61, 2.05)	4	0.66	(0.18, 2.38)	31	1.23	(0.65, 2.33)
25-29	54	36.0	36	24.0	1.00	(Reference)	7	1.00	(Reference)	29	1.00	(Reference)
30-34	20	13.3	32	21.3	2.40	(1.19, 4.83)	8	3.09	(0.99, 9.62)	24	2.23	(1.06, 4.71)
≥35	18	12.0	33	22.0	2.75	(1.35, 5.61)	7	3.00	(0.93, 9.72)	26	2.69	(1.27, 5.70)
Maternal education (years)												
≤6	45	30.0	55	36.7	0.79	(0.34, 1.86)	7	0.34	(0.09, 1.29)	48	0.98	(0.39, 2.44)
7-12	94	62.7	78	52.0	0.54	(0.24, 1.21)	17	0.40	(0.12, 1.29)	61	0.59	(0.25, 1.43)
>12	11	7.3	17	11.3	1.00	(Reference)	5	1.00	(Reference)	12	1.00	(Reference)
Marital status												
Married	76	50.7	84	56.0	1.00	(Reference)	14	1.00	(Reference)	70	1.00	(Reference)
Unmarried	69	46.0	61	40.7	0.80	(0.50, 1.27)	15	1.18	(0.53, 2.62)	46	0.72	(0.44, 1.19)
Separated	5	3.3	5	3.3	0.90	(0.25, 3.25)	0	-		5	1.09	(0.30, 3.91)
Smoked in pregnancy												
No	145	96.7	149	99.3	1.00	(Reference)	28	1.00	(Reference)	121	1.00	(Reference)
Yes	5	3.3	1	0.7	0.19	(0.02, 1.69)	1	1.04	(0.12, 9.21)	0	-	
Alcohol use in pregnancy												
No	140	93.3	149	99.3	1.00	(Reference)	29	1.00	(Reference)	120	1.00	(Reference)
Yes	10	6.7	1	0.7	0.09	(0.01, 0.74)	0	-		1	0.12	(0.01, 0.92)
Employed during pregnancy												
No	46	30.7	55	36.7	1.31	(0.81, 2.12)	6	0.59	(0.23, 1.55)	49	1.54	(0.93, 2.54)
Yes	104	69.3	95	63.3	1.00	(Reference)	23	1.00	(Reference)	72	1.00	(Reference)
Exercise during pregnancy												
No	120	82.2	126	85.7	1.00	(Reference)	24	1.00	(Reference)	102	1.00	(Reference)
Yes	26	17.8	21	14.3	0.77	(0.41, 1.44)	3	0.58	(0.16, 2.06)	18	0.81	(0.42, 1.57)

Table 2. Odds ratios (OR) and 95% confidence intervals (CI) for reproductive and medical history characteristics of preeclampsia cases and normotensive controls, Bangkok, Thailand, 2006–2007.

Covariates	Controls (n = 150)		Preeclampsia (n = 150)		Unadjusted OR		Early Onset Preeclampsia (n = 29)			Late Onset Preeclampsia (n = 121)		
	n	%	n	%	OR	(95% CI)	n	OR	(95% CI)	n	OR	(95% CI)
Parity												
Nulliparous	90	60.0	82	54.7	0.80	(0.51, 1.27)	14	0.62	(0.28, 1.38)	68	0.86	(0.53, 1.39)
Multiparous	60	40.0	68	45.3	1.00	(Reference)	15	1.00	(Reference)	53	1.00	(Reference)
History of hypertensive disorders during pregnancy												
Nulliparous	90	60.0	82	54.7	0.93	(0.58, 1.48)	14	0.83	(0.35, 1.96)	68	0.95	(0.58, 1.56)
Parous-no prior history	59	39.3	58	38.7	1.00	(Reference)	11	1.00	(Reference)	47	1.00	(Reference)
Parous-prior history	1	0.7	10	6.7	10.17	(1.26, 82.02)	4	21.45	(2.19, 210.57)	6	7.53	(0.88, 64.75)
Number of previous abortion												
0	113	75.3	115	76.7	1.00	(Reference)	21	1.00	(Reference)	94	1.00	(Reference)
1	32	21.3	31	20.7	0.95	(0.54, 1.66)	7	1.18	(0.46, 3.02)	24	0.90	(0.50, 1.64)
≥2	5	3.3	4	2.7	0.79	(0.21, 3.00)	1	1.08	(0.12, 9.68)	3	0.72	(0.17, 3.10)
Family history of hypertension												
No	135	90.0	123	82.0	1.00	(Reference)	24	1.00	(Reference)	99	1.00	(Reference)
Yes	14	9.3	27	18.0	2.12	(1.06, 4.22)	5	2.01	(0.66, 6.09)	22	2.14	(1.04, 4.40)
Missing	1	0.7	0	0.0			0			0		

Table 3. Odds ratios (OR) and 95% confidence intervals (CI) for current pregnancy characteristics of preeclampsia cases and normotensive controls, Bangkok, Thailand, 2006-2007.

Covariates	Controls (n = 150)		Preeclampsia (n = 150)		Unadjusted OR		Early Onset Preeclampsia (n = 29)			Late Onset Preeclampsia (n = 121)		
	n	%	n	%	OR	(95% CI)	n	OR	(95% CI)	n	OR	(95% CI)
Pre-pregnancy BMI (kg/m²)												
<18.5	31	20.7	11	7.3	0.38	(0.18, 0.81)	2	0.38	(0.08, 1.72)	9	0.39	(0.17, 0.86)
18.5-24.9	93	62.0	86	57.3	1.00	(Reference)	16	1.00	(Reference)	70	1.00	(Reference)
25-29.9	16	10.7	25	16.7	1.69	(0.85, 3.38)	7	2.54	(0.90, 7.16)	18	1.49	(0.71, 3.14)
≥30.0	5	3.3	22	14.7	4.76	(1.73, 13.12)	3	3.49	(0.76, 16.05)	19	5.05	(1.80, 14.18)
Missing	5	3.3	6	4.0			1			5		
Maternal height (cm)												
Low stature (≤145)	3	2.0	2	1.3	0.65	(0.11, 3.97)	0	-		2	0.81	(0.13, 4.94)
Normal (>145)	145	96.7	148	98.7	1.00	(Reference)	29	1.00	(Reference)	119	1.00	(Reference)
Missing	2	1.3	0	0.0			0			0		
Prenatal care onset												
Care initiated in 1st trimester	67	44.7	63	42.0	1.00	(Reference)	15	1.00	(Reference)	48	1.00	(Reference)
Care initiated after 1st trimester	79	52.7	77	51.3	1.04	(0.65, 1.65)	13	0.74	(0.33, 1.65)	64	1.13	(0.69, 1.86)
No prenatal care	4	2.7	10	6.7	2.66	(0.79, 8.91)	1	1.12	(0.12, 10.72)	9	3.14	(0.91, 10.80)
Infant gender												
Female	68	45.3	76	50.7	1.00	(Reference)	17	1.00	(Reference)	59	1.00	(Reference)
Male	82	54.7	74	49.3	0.81	(0.51, 1.27)	12	0.59	(0.26, 1.31)	62	0.87	(0.54, 1.41)

Table 4. Adjusted odds ratios (OR) and 95% confidence intervals (CI) according to selected factors, Bangkok, Thailand, 2006-2007.

Covariates	All Preeclampsia (n = 150)		Early Onset Preeclampsia (n = 29)		Late Onset Preeclampsia (n = 121)	
	Adjusted OR†	(95% CI)	Adjusted OR†	(95% CI)	Adjusted OR†	(95% CI)
Maternal age (years)						
<20	1.99	(0.70, 5.68)	1.80	(0.27, 12.01)	1.94	(0.64, 5.86)
20-24	1.11	(0.57, 2.18)	0.67	(0.17, 2.70)	1.13	(0.56, 2.27)
25-29	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)
30-34	2.30	(1.03, 5.17)	1.55	(0.38, 6.33)	2.23	(0.97, 5.13)
≥35	2.04	(0.89, 4.67)	1.87	(0.45, 7.74)	1.99	(0.85, 4.68)
Trend test p-value	0.229		0.314		0.249	
Alcohol use in pregnancy	0.10	(0.01, 1.06)	-		-	
Smoked in pregnancy	0.14	(0.01, 2.21)	-		-	
Exercise during pregnancy		-	0.55	(0.11, 2.76)	-	
Family history of hypertension	1.92	(0.87, 4.23)	-		1.83	(0.83, 4.03)
History of hypertensive disorders during pregnancy						
Nulliparous	1.31	(0.71, 2.39)	0.98	(0.34, 2.80)	1.45	(0.78, 2.70)
Parous-no prior history	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)
Parous-prior history	7.83	(0.88, 69.44)	24.30	(2.05, 287.87)	4.77	(0.50, 45.34)
Pre-pregnancy BMI (kg/m²)						
<18.5	0.31	(0.13, 0.73)	0.38	(0.07, 2.24)	0.29	(0.12, 0.73)
18.5-24.9	1.00	(Reference)	1.00	(Reference)	1.00	(Reference)
25-29.9	1.71	(0.81, 3.58)	2.70	(0.82, 8.91)	1.64	(0.75, 3.57)
≥30.0	5.25	(1.80, 15.32)	4.07	(0.81, 20.42)	5.33	(1.83, 15.51)
Trend test p-value	<0.001		0.009		<0.001	
Prenatal care onset/utilization						
Care initiated in 1st trimester	1.00	(Reference)	-		1.00	(Reference)
Care initiated after 1st trimester	0.99	(0.57, 1.70)	-		1.04	(0.59, 1.84)
No prenatal care	6.37	(1.26, 32.27)	-		7.19	(1.36, 38.11)

Each column represents a different logistic regression model. Covariates not included in final models are indicated as (-). Each OR and 95%CI are adjusted for all other covariates listed in this table.

Maternal age at extremes (<20 and >40 years) was identified as a risk factor of preeclampsia in a Saudi Arabian population [13]. Advanced maternal age (>35 years) had also been shown in other populations to be associated with increased risk of preeclampsia [4]. Similar patterns of risk were observed in our study of Thai women, where young (<20 years) and older (≥30 years) women were at an increased risk of preeclampsia regardless of whether the condition was classified as early or late onset.

Obesity is perhaps the most consistently reported modifiable risk factor of preeclampsia [4, 11, 14-17]. Mahomed et al. reported that Zimbabwean women with preeclampsia had a higher BMI (27.6±4.4 kg/m^2) compared with normotensive women (25.2±3.8 kg/m^2) [16]. Mittendorf et al. reported a 2.7-fold increased risk of preeclampsia in obese women compared to women with a pre-pregnancy BMI between 18-30 kg/m^2 [11]. We observed a statistically significant linear trend of increased preeclampsia risk with increasing BMI. Similar linear trends have been reported by other investigators [16-18]. Although precise biologic mechanisms for associations of preeclampsia risk with maternal adiposity are unknown, several possible mechanisms have been proposed. Investigators have speculated that hyperlipidemia, oxidative stress and diffuse endothelial dysfunction are more common among obese versus lean pregnant women [17-19]. Others have postulated that the association is secondary to alterations in cardiac output [20]. Although compelling, these hypothesized mechanisms have yet to be evaluated in carefully-designed cohort studies of pregnant women.

Positive personal histories of hypertensive disorders of pregnancy [12, 14, 15] and family history of hypertensive disorders [21, 22] are well known risk factors of preeclampsia. Preeclampsia is reported to recur in 13-18% of subsequent pregnancies [23, 24]. In accordance with previous studies [12, 14, 15], we observed increased preeclampsia risk among women with a prior history of pregnancies complicated by hypertensive disorders. Odegard et al. [12], in their study of Norwegian women, reported that those with a prior history of preeclampsia, compared with women without such a history, had a 21.5-fold increased risk of the disorder in another pregnancy (95%CI: 9.8, 47.2). The high recurrence risk of preeclampsia, and evidence of familial aggregation of hypertensive disorders [22] supports the concept that a subgroup

of women may be predisposed to developing this very dangerous complication of pregnancy.

Nulliparity has been identified as a risk factor for preeclampsia in several populations [4, 5, 11, 12]. However, we observed only a weak positive association of preeclampsia risk with this covariate. Additionally, although numerous authors have observed that smoking confers a decreased risk of preeclampsia [4, 5, 11, 12, 25], we did not identify maternal cigarette smoking during pregnancy as a statistically significant preeclampsia risk factor in the present study. The low frequency of mothers who reported smoking during pregnancy prohibited careful evaluation of this covariate among Thai women enrolled in our study.

von Dadelszen et al. [9] and Ilekis et al. [26] have recently supported the concept that preeclampsia is a heterogeneous disorder to be subdivided into the early and late onset case-groups. Using the criteria proposed by von Dadelszen et al. [9], we classified preeclampsia cases into the early and late onset subgroups and sought to empirically explore the possibility of heterogeneity in risk factors for the two groups of preeclampsia cases. Consistent with prior reports [3], we noted that the vast majority of preeclampsia cases (81%) were classified as the late onset cases while 19% were classified as early onset cases. Consistent with the literature summarized by Huppertz [3], we noted little evidence to support the thesis that there may be substantial differences in the epidemiology of the early and late onset preeclampsia. Although it would be highly desirable to define homogeneous subgroups of preeclampsia, great care must be taken to avoid introducing non-biological factors (e.g., health services factors such as variations in maternal utilization patterns of prenatal care or variations in clinical management protocols of the disorder).

In the present study, we were able to empirically assess risk factors of clinical subtypes of preeclampsia, using well-trained personnel to collect information from all participants. All interviewers were blinded to participants' case-control status and a well structured questionnaire was used to systematically collect risk factor information. The high participation rates for cases and controls (97.5% and 95.7%) also served to attenuate concerns about the selection bias. However, a number of limitations should be considered when interpreting results from our study. First, our study was limited in evaluation of several risk factors previously reported to influence the risk of preeclampsia. In particular, we did not have information

pertaining to maternal mood and anxiety disorders [27], migraine disorders [28] or maternal dietary habits before and during pregnancy [29]. Second, the low prevalence of certain maternal risk factors (i.e., cigarette smoking and alcohol consumption during pregnancy) among women limited our estimation of precise odds ratios for these covariates. Third, since we studied women residing in metropolitan Bangkok, our observations may not be generalizable to Thai women residing in rural settings. Nevertheless, our findings are largely consistent with those reported from other more diverse populations. Lastly, the case-control study design does not allow us to dismiss the possibility of recall bias.

Our findings and those by other investigators point to public health and clinical measures that may be taken to potentially attenuate the incidence of preeclampsia and mitigate associated maternal-fetal complications resulting from the disorder.

In conclusion, advanced maternal age, obesity, and no utilization of prenatal care were covariates identified as risk factors for preeclampsia.

Acknowledgment

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