

Maternal Stress and Pregnancy Outcomes*

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Abstract

Aims of the study: To seek for magnitude of stress conditions among pregnant women, the clinical profile of stressed pregnant women and the potential of stress on adverse maternal and perinatal outcomes in Kinshasa. Methods: This is a multicentre 6-month case-control study dealing with consenting women having given birth at 13 biggest maternities of Kinshasa, the capital of DR Congo. Mother-infant pairs were considered cases (obviously stressed women) and controls (light or not stressed women) at the end of data processing according to their perceived stress scores (PSS). Stress events, potential stress factors and maternal general and physical characteristics were registered along with maternal and perinatal outcomes. Odds ratios calculation allowed finding influence of stress on occurrence of adverse outcomes. Results and conclusions: Our study concerned 1082 women whose 57.1% (n = 618) qualified as the stressed. General characteristics found significantly different between stressed and non-stressed women were marital status (the married and widowed more frequent among the stressed), instruction level (the educated more frequent among the stressed), socioeconomic status (the elevated more frequent among the stressed) and religion status (both the traditional and new charismatic more frequent among the stressed). Obstetric risk factors were similar in both groups that were different only according to stress factors, whether emotional (relationships) or pregnancy (preciousness) related. The 3 most prominent stress factors were parent's death (p 0.000), tension in family (p 0.000) and tension in couple (p 0.003). All expectedly compensating factors appeared significantly more frequent among stressed women. As of organic pregnancy outcomes infection, gastritis, hypertensive disorders and preterm labor were maternal ones significantly more frequent among stressed women. All non-organic outcomes (insomnia and depression) were significantly predominant among stressed women. In offspring, prematurity, low birth weight and perinatal death were significantly dominant among those born to stressed women. Odds ratios calculation showed significant potential of stress conditions on occurrence of all maternal complications but cesarean section. In

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offspring too, risk of outcomes' occurrence was enhanced by stress, except for macrosomia and neonatal distress.

Keywords

Maternal Stress, Maternal Outcomes, Perinatal Outcomes, Sub-Saharan Africa, DR Congo

1. Introduction

According to audits of maternal and perinatal mortality from developing countries, killing complications (hemorrhage, infection, hypertensive disorders, disordered fetal growth and prematurity) are strongly influenced by low maternal education, short interpregnancy interval, limited financial access to and late or no prenatal care, and suboptimal health care [1] [2]. Although emotional environment has been found generating perinatal morbidity [3]-[12], it is still far from ranging among major causative or determinant factors, and there has been no mention of stress in numerous global recommendations dealing with persistently high rates of perinatal mortality. This could be owed to the fact that considerable attention has been paid on mortality (an obvious matter) rather than morbidity (possibly discrete). Strong evidence, however, is being got that certain actions that are inefficient to reduce maternal and perinatal mortality do have significant influence on morbidity [1] [2], the burden of which is the highest in developing countries [13].

Emotional insult provokes an adaptive response involving the hypothalamic-pituitary-adrenal axis, with release of immune (interleukins 1, 6, and Tumor Necrosis Factor-alpha), hormonal (prostaglandins) and neurohormonal (corticotrophin-releasing hormone, hence cortisol and catecholamines) mediators [3] [5] [9] [14] [15]. This is the stress condition made of a systemic proinflammatory climate devoted to controlling the insult. Excessive response will result in distress, which is made of sensations meaning that a person perceives that he fails to mobilize personal and/or social resources needed for coping the situation [16]. This systemic proinflammatory state was found similar to that following exposure to various non-emotional situations involved in heavy production of free radicals or reactive oxygen species (ROS): trauma, infection, heat injury, ionizing radiation, toxins, obesity, certain foods, cigarette smoke, chemical agents, climate and environmental pollutants [17] [18]. If antioxidant activity fails to balance overproduction of ROS, oxidative stress will ensue, meaning oxidation of main macromolecules and DNA. Resulting alteration of vital cell mechanisms along with systemic inflammatory state is responsible for a lot of illnesses, including non-communicable chronic diseases [18].

Since placenta produces free radicals due to its content of transitional metals such as iron [11] [19], pregnancy represents, mostly by the second trimester, a basic stress condition. An additional insult (emotional or not) is expected to enhance stress mediators release that in turn is the basis for pluri-visceral damages, alteration of sub-decidual angiogenesis, materno-fetal transfer of stress substances and reduction in intrauterine blood flow [11] [12] [17]. This will lead to myometrial irritability and fetal inflammatory climate [20], the effects of which are accountable for in reportedly high frequencies of pregnancy loss [21], shortening of gestational age, prematurity, restriction of birth weight [3]-[6] [8]-[10] and neonatal intravascular hemorrhage [11] [12].

Besides, it is still to be addressed why reports on maternal stress have been exclusively focusing on offspring's consequences although stress related reduction of maternal immune functions along with increased susceptibility to infections [22] [23], diabetes [24], gastritis [14] and cardiovascular disturbances [25] might also be associated with more occurrences of strictly maternal morbidity.

Although the issue of maternal stress used to be dealt with in developed countries, in most if not all developing countries stress conditions can be presumed significantly due to poverty and/or conflicts situation's vulnerability. Our study aimed to seek for magnitude of stress conditions among pregnant women in Kinshasa (the capital of DR Congo) and potential influence of stress on both maternal and perinatal outcomes.

2. Methods

This multicentre study is a case-control one approved by the faculty scientific board, university of Kinshasa. Participants were consenting women having given birth during a 6-month period (from 15 February 2013 throughout 15 August 2013) at 13 biggest maternities of Kinshasa, the capital of DR Congo, namely: University

clinics, General Provincial Reference Hospital, Ngaliema Hospital, Kintambo Hospital, Binza Maternity of Sisters, Lisungi Maternity of Sisters, Kingabwa Maternity of Sisters, Saint Joseph Hospital, Bondeko Hospital, Kingasani Maternity of Sisters, Bumbu Maternity and Military Camp Kokolo Maternity.

The study sample consisted of mother-infant pairs registered in the sites of the study after excluding multiple pregnancies (42 twin pregnancies and 1 triplet), non-consenting women (110 cases of refusal) and those (25) failing to answer some items. Women were considered cases (having experienced important stress) and controls (light or not stressed) at the end of data processing according to their stress scores based on psychological approach [7] [26] [27]. Stress scores were established using 2 tools: 1) perceived stress scale (**PSS**) [28] and 2) Bradford somatic inventory (BSI) [29]. PSS was based on quantification of 14 items through a questionnaire to be fulfilled once (within one week after delivery) for every significant life event experienced not only during the last month (as originally recommended) but since the start of pregnancy. Due to memory failure of participants timing of stress exposure according to first, second and third trimester was abandoned although some authors [3] [8] suggested some trimesters to be more vulnerable in terms of fetal response to maternal stress. Each event was quoted from 1 to 5 according to recurrence of reminding that reflects the stress intensity. The final score ranges from 14 to 70, a score \geq 42 being considered obvious stress. **BSI** was built on responses to a 44-item questionnaire quoted according to existence/persistence of symptoms as 0 (no symptom), 1 (symptom existing for less than 15 days) and 2 (symptom persisting beyond 15 days), a final score \geq 44 representing obvious stress. Symptoms were physical (chest pain, cardiac palpitations, fatigue, dizziness, breathing problems, headaches, stomach cramps) or psychological (memory loss, nightmares, inability to concentrate, fear, loneliness, irritability and dispute-provoking). In each maternity, a midwife was approached by one of us, Dedetemo K.D. (DKD), in order to be provided with proper knowledge regarding the study, and thereafter trained in how to collect information needed. In each maternity, DKD conducted himself more than five interviews in pairs, making it sure that all study's items are easy to understand and answer. He clarified files of participants before validating them.

General characteristics studied were: age (years), marital status, instruction level, parity, gravidity, gestational age at confinement (in weeks derived from the last menstrual period, the earliest ultrasound and neonatal age according to pediatrician assessment). The kinds of stress factors were mostly related to emotional events such as unplanned or much desired pregnancy (after infertility or **first pregnancy after age 35**), preciousness due to previous loss (stillbirth), separation during pregnancy, tension in couple or in family, illness or death of relative and to past and present pregnancy risk factors (primi and multiparity, age < 18 years or \geq 35 years, mother-infant's problems of previous pregnancies). Being married or cohabiting, coming from high socioeconomic level and belonging to a new charismatic religion more likely than that traditional religions (Catholic, Evangelical, Kimbanguist, and Islamic) to provide with social support [30] [31] were considered potential buffers, expected to be compensative. Socioeconomic status was defined on the basis of usual criteria in our setting [32]-[35].

Adverse outcomes of just terminated pregnancy dealt with any obstetric complication, mostly threatened abortion/preterm labor, premature delivery (\geq 28 and <37 week gestation), hypertensive disorders (first-time recognization during pregnancy), diabetes, infection(including urinary infection, chorioamniotitis, and post-partum endometritis), gastritis (**first-time diagnosis and treatment during pregnancy**), insomnia, depression (feeling of worthlessness/lifelessness, emptiness/hopelessness, and helplessness), cesarean section, fetal/neonatal distress (Apgar score at the 5th minute considered) or mortality (within the first 48 hours following birth), macrosomia (birth weight \geq 4000 g) and low birth weight (LBW < 2500 g).

3. Statistical Analysis

Data collection was made through EPIDATA 3.1, thereafter analyzed by use of SPSS 17.0. Means and proportions of stressed and non-stressed women were compared by use of Student's t and Chi-square respectively. Odds ratios calculation allowed finding influence of stress itself and that of obstetric risk factors on occurrence of maternal/infant's adverse outcomes. Calculations were intended to be furthered after controlling for potential buffering factors. Maternal obstetric pathologies linked to just terminated pregnancy were taken as co-morbidities of stress and analyzed as covariates likely to address the direction of perinatal outcomes causation.

4. Results

Our study concerned 1082 women whose 57.1% (n = 618) qualified as stressed according to PSS assessment,

while this rate dropped to 1.8% (n = 20) with Bradford scale. So, due to smallness of the sample using Bradford scale (to be discussed later) further calculations were restricted to assessment according to PSS. Overall study sample was aged 28 ± 6.4 , with mean parity, gravidity, abortus and gestation age of 3 ± 1.7 , 3 ± 1.9 , 0.38 ± 0.7 , and 38 ± 0.7 weeks respectively. Neonatal variables included birth weight (3074 ± 580 g) and Apgar score at the 5th minute (8.1% under 7/10). Socio-demographic characteristics are presented in Table 1.

The majority of study sample was married (61.4%), educated (81.1%) and of low socioeconomic level (58%). General characteristics found significantly different between stressed and non-stressed women were marital status (the married and widowed more frequent among the stressed), instruction level (the less educated more frequent among the stressed), socioeconomic status (the elevated more frequent among the stressed) and religion status (both the traditional and new charismatic more frequent among the stressed).

Table 2 represents both stressors and obstetric risk factors in order to address relationships between stress and perinatal outcomes. We included expected compensating factors within it.

Stress factors that built differences between stressed and non-stressed women were pregnancy (preciousness) and emotional (relative's illness/death and tension in couple/family)-related. As of obstetric risk factors known to influence perinatal outcomes both groups were similar, except for illiteracy and previous stillbirth, a stressor as well, that was significantly more frequent among stressed. All expectedly compensating factors were found significantly more frequent among stressed women.

Of maternal outcomes (Table 3), infection, gastritis, hypertensive disorders and preterm labor were organic ones significantly more frequent among stressed women. Non-organic outcomes (insomnia and depression) were significantly predominant among stressed women. In offspring, prematurity, low birth weight and perinatal death were significantly dominant among those born to stressed women. Odds ratios calculation showed the potential of stress conditions in at least doubling the risk for all maternal complications but cesarean section. In offspring, risk for prematurity, LBW and perinatal death were enhanced by stress from 1.5 times to twice.

Variables	0	verall	Stressed						
	N	Mean	n	%	Mean	n	%	Mean	р
Maternal age	1082	28 ± 6.41	618	57.1	$28.6\pm6,\!5$	464	42.9	27.8 ± 6.3	0.7
Parity	1082	3 ± 1.7	618	57.1	2.6 ± 1.7	464	42.9	2.6 ± 1.7	0.9
Gravidity	1082	3 ± 1.9	618	57.1	2.9 ± 1.9	464	42.9	2.9 ± 1.9	0.7
Abortion	1082	0.4 ± 0.7	618	57.1	0.4 ± 0.8	464	42.9	0.35 ± 0.7	0.3
Gestational age	1082	38 ± 2.1	618	57.1	38.5 ± 2.1	464	42.9	38.5 ± 2.1	0.6
Marital status									
Married	664		408	61.4	-	256	38.6		0.000
Single	207	-	109	52.6	-	98	47.4	-	0.08
Separated	21	-	13	61.9	-	8	38.1		0.42
Cohabitating	184	-	101	54.9	-	83	45.1	-	0.2
Widowed	6	-	6	100	-	0	0	-	0.03
Instruction level									
Illiterate and primary school	204	-	139	68.1	-	65	31.9	-	0.000
Secondary	586	-	323	55.1	-	263	44.9	-	0.08
Post-secondary	292	-	170	58.2	-	122	41.8	-	0.3
Socioeconomic Status									
Low	665	-	386	58.0	-	279	42.0	-	0.2
Middle	283	-	156	55.1	-	127	44.9	-	0.2
Elevated	134	-	89	66.4	-	45	34.6	-	0.01
Religion									
Traditional	304	-	199	65.4	-	105	34.6	-	0.000
New charismatic	723	-	429	59.3	-	294	40.7	-	0.02
Other	55	-	36	65.4	-	19	34.6	-	0.2

Variables		Stressed women			Non-stressed women		
	Total	n	%	n	%	Р	
Stress factors							
Unplanned pregnancy	380	221	58.15	159	41.9	0.33	
Much-desired pregnancy	119	76	63.9	43	36.1	0.07	
Precioussness	49	38	77.6	11	22.4	0.02	
Previous prematuriy	9	5	55.5	4	44.5	0.6	
Relative's illness	102	68	66.7	34	33.3	0.006	
Partner's death	10	9	90	1	10	0.02	
Parent's death	156	108	69.2	48	30.8	0.000	
Tension in couple	157	102	65	55	35	0.003	
Tension in family	13	13	100	0	0	0.000	
Obstetric risk factors							
Maternal age < 18	39	21	53.8	18	46.2	0.4	
Maternal age \geq 35	190	110	57.9	80	42.1	0.2	
Primiparity	398	225	65.5	173	34.5	0.4	
Parity ≥ 6	73	39	53.4	34	46.6	0.5	
Spontaneous abortion ≥ 3	196	108	55.2	88	44.8	0.5	
Previous stillbirth	49	38	77.6	11	22.4	0.02	
Previous prematurity	9	5	55.5	4	45.5	0.6	
Low socioeconomic status	665	386	58	279	42	0.2	
Illiterate and primary school	204	139	68.1	65	31.9	0.000	
Expectedly compensating factors							
Married	664	408	61.4	256	38.6	0.000	
High socioeconomic level	134	84	63.6	48	36.8	0.02	
New charismatic religions	429	429	59.3	294	36.1	0.02	

Table 2. Stress factors and obstetric risk factors among stressed and non-stressed women.

Furthering calculations on stress factors in order to see which ones were individually most linked to specific adverse outcomes (**Table 4**) showed that unplanned pregnancy, much-desired pregnancy and preciousness were not involved in these individual relationships. Tension in couple was significantly linked to gastritis, hypertensive disorders, prematurity and LBW, while tension in family was linked to gastritis, insomnia, depression, prematurity and LBW. Death of relative was linked to gastritis and LBW. No specific stressor was found significantly linked to perinatal death, an outcome twice as frequent among children born by stressed women.

In order to see whether maternal pathologies linked to just terminated pregnancy (gastritis, hypertensive disorders, infection, insomnia and depression) might act as covariates (co-morbidities) of stress likely to amplify perinatal outcomes causation, they were analyzed (adjusted risk ratio) in dichotomous model (stress with or without the pathology). We thus found that prematurity and perinatal death were concerned (significant rise in occurrence of outcome considered). For prematurity, the most deleterious combinations were stress + hypertensive disorders (p 0.004; OR 2.9; CI 1.3 - 6.1), stress + depression (p 0.04; OR 4.3; CI 1.1 - 17.2) and stress + infection (p 0.004; OR 2.3; CI 1.3 - 4.0). For perinatal death, the only deleterious combination was stress + infection (p 0.03; OR 2.4, CI 1.1 - 5.3).

5. Discussion

Retrospective measure of stress using perceived stress scale (PSS) after delivery appeared to detect numerous women (57.1%) having experienced significant stress condition during just terminated pregnancy. Such a prevalence is much higher than the reportedly average 25% expected during pregnancy [36]. We have no other es-

		Stressed		Non-stressed						
	Overall sample N (%)	n	%	n	%	р	OR		CI	
Maternal outcomes										
Threatened abortion/ Preterm labor	25 (2.3)	21	84	4	16	0.002	4.5	1.5	13.	
Hypertensive disorders	93(8.6)	66	71	27	29	0.001	2.2	1.4	3.5	
Gastritis	213 (19.7)	142	66.7	71	33.3	0.000	1.9	1.4	2.6	
Depression*	14 (1.3)	12	85.7	2	14.3	0.02	5.1	1.1	22.	
Insomnia	244 (22.6)	165	67.6	79	32.4	0.000	2.0	1.5	2.7	
Cesarean section	46 (4.3)	26	56.5	20	43.5	0.5	1	0.5	1.8	
Infection	378(34.9)	240	63.5	138	36.5	0.000	1.8	1.4	2.3	
Perinatal outcomes										
Prematurity	64 (5.9)	43	67.2	21	32.8	0.03	1.8	1.0	3	
Perinatal death	31 (2.8)	24	77.4	7	22.6	0.01	2.6	1.1	6.2	
LBW	141 (13)	93	65.9	48	34.1	0.01	1.5	1.1	2.2	
Macrosomia	64 (5.9)	31	48.4	33	51.6	0.2	0.8	0.5	1.3	
Neonatal distress	88 (8.1)	50	56.8	38	43.2	0.5	1.0	0.5	1.6	

Table 3. Adverse maternal and perinatal outcomes according to maternal stress.

*Including 2 cases of puerperal psychosis.

Table 4. Maternal and perinatal outcomes according to individual stress factors (p-value, OR, Confidence interval).

Factors	Threatened abortion/ Preterm labor	Hypertensive disorders	Gastritis	Depression	Insomnia	Infection	Prematurity	Perinatal death	LBW
Unplanned pregancy	-	-	-	-	-	-	-	-	-
Much-desired pregnancy	-	-	-	-	-	-	-	-	-
Precioussness	-	-	-	-	-	-	-	-	-
Relative's illness	-	-	-	-	-	p 0.000 OR 2.5 (1.6 - 3.8)	p 0.05 OR 2 (1 - 4)	-	-
Partner's death	p 0.04 OR 2.5 (1 - 6)	-	p 0.002 OR 7.4 (2 - 27)	-	p 0.001 OR 2 (1.3-3)	p 0.01 OR 6 (1.5 - 24.7)	-	-	p 0.000 OR 11 (3 - 41.4)
Parent's death	-	-	p 0.01 OR 2.3 (1.1 - 2.5)	p 0.03 OR 3.6 (1.1 - 11.8)	-	p 0.001 OR 1.9 (1.3 - 2.7)	-	-	-
Tension in couple	p 0.007 OR 3.2 (1.4 - 7.4)	p 0.009 OR2 (1.2 - 3.3)	p 0.000 OR 2.3 (1.6 - 3.5)	p 0.000 OR 11.4 (3.7 - 35.4)	p 0.000 OR 2.8 (1.9 - 4.1)	p 0.000 OR2.3 (1.6 - 3.3)	-	-	p 0.002 OR 2 (1.3 - 3)
Tension in family	-	p 0.01 OR17.7 (1.8 - 175)	p 0.02 OR 3.9 (1.3 - 12)	p 0.04 OR 11.7 (1.1 121)	p 0.02 OR 3.8 (1.2 - 12)	-	p 0.007 OR 6.4 (1.6 - 24.5)	-	p 0.04 OR 3.6 (1 - 12)

timations for comparison in Sub-Saharan Africa where the issue seems not to matter. Based on the rate of distress which was also identified after delivery and varying from 22.1% to 52.9% of the whole obstetric population in Sao Paulo, Brazil [37], our prevalence indicates that the issue of maternal stress should be considered common in obstetric practice. Further comparison with previous global literature remains difficult to be carried out, due to differences in tools to assess emotional stress during pregnancy and depending on whether it is done with or without additional biochemical markers and during usual life circumstances or following exposure to catastrophic/traumatic events or particular situations of chronic stress [38]-[40]. Global broad variety in how to measure stress during pregnancy has been, however, offset by persistence of similar trends regarding high risk of prematurity and low birth weight. Due to lack of similar study of the phenomenon in African environment, we had no choice but to use a formal tool that fortunately distinguishes two groups of mother-infant pairs as attested by our results. Low detection of stressed women (1.8%) using Bradford somatic inventory (BSI) can be explained by the fact that the majority of our study sample (81.1%) was educated, making women less suitable to be investigated through this tool. BSI is a transcultural method of assessment permitting to exclude abstract language, which makes it easy to handle even in primary care institutions for expressing psychosomatic symptoms [29].

The key emotional insults of our series were represented by stormy family and partner-related situations. Although financial constraint accompanying economic depression is expected to expose to stress [5], it did not appear to matter in its generation. Whether this finding is typically local (in Kinshasa) or African remains to be addressed. Due to cultural differences in low perception of the magnitude of stress situations, African people have been claimed to be less disturbed by certain devastating events [35]. We thus postulate that for less favored women so far immerged in deprivation financial constraints might no more represent a crucial issue, at least in comparison with solidarity's values contained in community and partner-related factors. Rather, instead of having buffering effect high socioeconomic status seemed to be stress enhancer, a finding linkable to fear to lose social advantages and/or inability to solve the stressing situation. Belonging to a new charismatic religion did neither appear to provide social support, conversely to findings from some studies [27] [30] [31]. By the time (2003) Dole *et al.* [4] questioned success of social support in the issue of prematurity, for instance, there has not as yet been evidence of its confirmation. So, meditation and counseling that have been advocated as mental escape to retrieve stressing situations [7] [27] have to be better conceived by the community in order to find accurate forms that could be compensative among stressed women.

A major purpose when launching this research was to seek for maternal morbidity attributable to stress. Actually, organic outcomes such as infection, gastritis, hypertensive disorders and threatened abortion/preterm labor and non-organic ones such as insomnia and depression were significantly predominant among stressed women. Although global understanding of stress has dramatically improved these last ten years, making it evident to expect such consequences among mothers [14] [15] [19] [22] [23] [24], authors used to restrict the issue of stress-induced morbidity to offspring. Dole *et al.* [4] mentioned medical co-morbidities to anxiety in its association with preterm birth and Tegethoff *et al.* [8] included hypertension and diabetes during pregnancy among major potential confounders to explain perinatal outcomes rather than potential maternal outcomes of stress in themselves. Some others [5] excluded mothers with hypertension and/or infection from their study sample. Our study whose results are consistent with suggested pathways is thus the first one supporting that stress-related morbidity during pregnancy involves offspring as well as mother. For those planning to promote maternal health, this finding is expected to redeem prevention or mitigation of maternal adverse outcomes, including hypertensive disorders and infection (generated by stress or not), two of major causes of mortality [13].

Regarding infant's data, our results confirm findings of previous studies so far reporting high incidence of morbidity in terms of shortening of gestational age [3]-[6], [8]-[10], [18], prematurity and restriction of birth weight [3]-[10] due to effect of stress mediators on myometrial irritability and materno-fetal inflammatory climate [20]. Based on our results, perinatal death should be for the first time added to the list. No specific stressor was found significantly linked to perinatal death, an outcome twice as frequent among children born by stressed women. Since there was no significant difference of neonatal distress between children born to stressed and non-stressed, we have come to infer that killing stressors mostly acted during pregnancy. Stillbirth is known to be likely to recur, which strengthens its emotional charge, provided it also is known to separate couples [37].

The retrospective nature of our study is inaccurate for recognizing all maternal medical co-morbidities that are likely to impact on perinatal outcomes. However, those encountered as maternal adverse outcomes showed close relationships with perinatal adverse outcomes, mostly prematurity. Such a finding is in accordance with Dole *et al.* [4] when concluding that the association between pregnancy-related anxiety and preterm birth is reduced but not eliminated when restricted to women without medical co-morbidities.

The main strength of this study addressing the issue of maternal stress during pregnancy is to be the first one considering adverse outcomes in mother-infant pairs. This is important when planning actions for the promotion of maternal well-being beyond the challenge of mortality. It is also the first to address the issue in a deprived environment, making it obvious that the issue of stress actually matters in our setting. Another striking finding

of our study was to illustrate that stress situation's impact on both mother and infant is linked to emotional (relationships) and pregnancy (preciousness) factors rather than poverty in itself. Most studies on relations between stress and offspring have concentrated on prematurity and birth weight. Our study added fetal/neonatal status in order to address the issue of perinatal mortality. Finally, inclusion of all biggest maternities of the town makes it of key value for the generalization of results in our setting.

The main limitations of our study lie in its retrospective nature. In settings where attention is not paid on full completion of patients' records, it is source of missing variables such as cervical status during pregnancy (cervical incompetence being a major risk factor for prematurity), nutrition status, physical activity and certain maternal pathologies happening during just terminated pregnancy (anemia, malaria, intestinal parasitosis and chronic infections). Likewise, underreporting is likely to affect risky behaviors (drugs/alcohol and certain foods) or physician's medications intended to relieve the stress situation. In this field, prospective cohort studies have many advantages, including the possibility to assess laboratory stress markers [37] although searchers have to confront the ethical need to intervene in those persons experiencing stress situation, which is likely to modify direction of the causation between stress and pregnancy outcomes.

Further studies should consider diagnosis of stress in terms of continuum rather than as a scaled situation and adapt it to the late or no prenatal care that is common in our setting.

6. Conclusion

More than the half of pregnant women in our setting is exposed to stress, the key risk factors being family and partner-related ones. High socioeconomic and married status are significantly associated with stress while there is no obvious buffering factor. Stress enhances risk for strictly maternal morbidities, whether organic (infections, gastritis and hypertensive disorders) or non organic (insomnia and depression). Infant's morbidity made of shortening of gestational age and restriction of birth weight makes maternal stress an additional factor to explain perinatal death.

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Authors' Contribution

This work was carried out in collaboration between all authors. Author TUB generated the scientific ideas and study design, contributed to results interpretation and revised the manuscript critically. Author DKD participated in generating the scientific ideas and study design, organized the data collection and analysis and revised the manuscript critically. Author MLG participated in generating the scientific ideas and study design and revised the manuscript critically. Author MLG participated in generating the scientific ideas and study design and revised the manuscript critically. All of them certify that they have read and approved the manuscript.

Conflict of Interest

We declare to have no conflict of interest.

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References

- [1] Abou-Zahr, C.L. and Wardlaw, T.M. (2003) Antenatal Care in Developing Countries: Promises, Achievements and Missed Opportunities: An Analysis of Trends, Levels and Differentials, 1990-2001.World Health Organization.
- [2] Khan, K.S., Wojdyla, D., Say, L., Gülmezoglu, A.M., Paul, F.A., Van Look, P.F.A., et al. (2006) WHO Analysis of Causes of Maternal Death: A Systematic Review. *The Lancet*, **367**, 1066-1074. http://dx.doi.org/10.1016/S0140-6736(06)68397-9
- [3] Class, Q.A., Lichtenstein, P., Långström, N. and D'Onofrio, B.M. (2011) Timing of Prenatal Maternal Exposure to Severe Life Events and Adverse Pregnancy Outcomes: A Population Study of 2.6 Million Pregnancies. *Psychosomatic Medicine*, 73, 234-241. <u>http://dx.doi.org/10.1097/PSY.0b013e31820a62ce</u>

- [4] Dole, N., Savitz, D.A., Hertz-Picciotto, I., Siega-Riz, A.M., McMahon, M.J. and Buekens, P. (2003) Maternal Stress and Preterm Birth. American Journal of Epidemiology, 157, 14-24. <u>http://dx.doi.org/10.1093/aje/kwf176</u>
- [5] Latendresse, G. (2009) The Interaction between Chronic Stress and Pregnancy: Preterm Birth from a Biobehavioral Perspective. *Journal of Midwifery and Women's Health*, 54, 8-17. <u>http://dx.doi.org/10.1016/j.jmwh.2008.08.001</u>
- [6] Khasan, A.S., McNamee, R., Abel, K.M., Pedersen, M.G., Webb, R.T., Kenny, L.C., Mortensen, P.B. and Baker, N.P. (2008) Reduced Infant Birth Weight Consequent upon Maternal Exposure to Severe Life Events. *Psychosomatic Medicine*, **70**, 688-694. <u>http://dx.doi.org/10.1097/PSY.0b013e318177940d</u>
- [7] Hobel, C., Goldstein, A. and Barrett, E.S. (2008) Psychosocial Stress and Pregnancy Outcome. *Clinical Obstetrics and Gynecology*, **51**, 333-348. <u>http://dx.doi.org/10.1097/GRF.0b013e31816f2709</u>
- [8] Tegethoff, M., Greene, N., Olsen, J., Meyer, A.H. and Meinlschmidt, G. (2010) Maternal Psychosocial Adversity during Pregnancy Is Associated with Length of Gestation and Offspring Size at Birth: Evidence from a Population-Based Cohort Study. *Psychosomatic Medicine*, **72**, 419-426. <u>http://dx.doi.org/10.1097/PSY.0b013e3181d2f0b0</u>
- Latendresse, G., Ruiz, R.J. and Wong, B. (2013) Psychological Distress and SSRI Use Predict Variation in Inflammatory Cytokines during Pregnancy. *Open Journal of Obstetrics and Gynecology*, 3, 184-191. http://dx.doi.org/10.4236/ojog.2013.31A034
- [10] Lee, S.Y., Park, K.H., Jeong, E.H., Oh, K.J., Ryu, A. and Park, K.U. (2012) Relationship between Maternal Serum C-Reactive Protein, Funisitis and Early-Onset Neonatal Sepsis. *Journal of Korean Medical Sciences*, 27, 674-680. <u>http://dx.doi.org/10.3346/jkms.2012.27.6.674</u>
- [11] Sorokin, Y., Romero, R., Mele, L., Wapner, R.J., Iams, J.D., *et al.* (2010) Maternal Serum Interleukin-6, C-Reactive Protein, and Matrix Metalloproteinase-9 Concentrations as Risk Factors for Preterm Birth <32 Weeks and Adverse Neonatal Outcomes. *American Journal of Perinatology*, 27, 631-640. <u>http://dx.doi.org/10.1055/s-0030-1249366</u>
- [12] Sykes, L., MacIntyre, D.A., Yap, X.J., Teoh, T.G. and Bennett, P.R. (2012) The Th1:Th2 Dichotomy of Pregnancy and Preterm Labour. *Mediators of Inflammation*, 2012, Article ID: 967629. <u>http://dx.doi.org/10.1155/2012/967629</u>
- [13] Firoz, T., Chou, D., von Dadelszen, P., Agrawal, P., Vanderkruik, R., Tunçalp, O., et al. (2013) Measuring Maternal Health: Focus on Maternal Morbidity. Bulletin of the World Health Organization, 91, 794-796.
- [14] Elenkov, I.J. and Chrousos, G.P. (1999) Stress Hormones, Th1/Th2 Patterns, Pro/Anti-Inflammatory Cytokines and Susceptibility to Disease. *Trends in Endocrinology & Metabolism*, **10**, 359-368. http://dx.doi.org/10.1016/S1043-2760(99)00188-5
- [15] Raison, C.L. and Miller, A.H. (2003) When Not Enough Is Too Much: The Role of Insufficient Glucocorticoid Signaling in the Pathophysiology of Stress-Related Disorders. *American Journal of Psychiatry*, 160, 1554-1565. <u>http://dx.doi.org/10.1176/appi.ajp.160.9.1554</u>
- [16] Lazarus, R.S. and Folkman, S. (1984) Stress, Appraisal and Coping. Springer Publishing Company, New York, 1-21.
- [17] Challis, J.R., Lockwood, C.J., Myatt, L., Norman, J.E., Strauss 3rd, J.F. and Petraglia, F. (2009) Inflammation and Pregnancy. *Reproductive Sciences*, 16, 206-215. <u>http://dx.doi.org/10.1177/1933719108329095</u>
- [18] Lobo, V., Patil, A., Phatak, A. and Chandra, N. (2010) Free Radicals, Antioxidants and Functional Foods: Impact on Human Health. *Pharmacognosy Review*, 4, 118-126. <u>http://dx.doi.org/10.4103/0973-7847.70902</u>
- [19] Casanueva, E. and Viteri, F.E. (2003) Iron and Oxidative Stress in Pregnancy. Journal of Nutrition, 133, 1700S-1708S.
- [20] Zaretsky, M.V., Alexander, J.M., Byrd, W. and Bawdon, R.E. (2004) Transfer of Inflammatory Cytokines across the Placenta. *Obstetrics & Gynecology*, **103**, 546-550. <u>http://dx.doi.org/10.1097/01.AOG.0000114980.40445.83</u>
- [21] Gupta, S., Agarwal, A., Banerjee, J. and Alvarez, J.G. (2007) The Role of Oxidative Stress in Spontaneous Abortion and Recurrent Pregnancy Loss: A Systematic Review. *Obstetrical & Gynecological Survey*, 62, 335-347. <u>http://dx.doi.org/10.1097/01.ogx.0000261644.89300.df</u>
- [22] Marsland, A.L., Bachen, E.A., Cohen, S., Rabin, B. and Manuck, S.B. (2002) Stress, Immune Reactivity and Susceptibility to Infectious Disease. *Physiology and Behavior*, **77**, 711-716. <u>http://dx.doi.org/10.1016/S0031-9384(02)00923-X</u>
- [23] Luebke, R.W. (2004) Suppression of Immune Function and Susceptibility to Infections in Humans: Association of Immune Function with Clinical Disease. *Journal of Immunotoxicology*, 1, 15-24. http://dx.doi.org/10.1080/15476910490438342
- [24] Fariss, M.W., Chan, C.B., Patel, M., Van Houten, B. and Orrenius, S. (2005) Role of Mitochondria in Toxic Oxidative Stress. *Molecular Interventions*, 5, 94-111. <u>http://dx.doi.org/10.1124/mi.5.2.7</u>
- [25] Davies, P.F. (2009) Hemodynamic Shear Stress and the Endothelium in Cardiovascular Pathophysiology. Nature Clinical Practice Cardiovascular Medicine, 6, 16-26. <u>http://dx.doi.org/10.1038/ncpcardio1397</u>
- [26] Lobel, M. (1994) Conceptualizations, Measurement, and Effects of Prenatal Maternal Stress on Birth Outcomes. Journal of Behavioral Medicine, 17, 225-272. <u>http://dx.doi.org/10.1038/ncpcardio1397</u>
- [27] Folkman, S., Lazarus, R.S., Gruen, R.J. and DeLongis, A. (1986) Appraisal, Coping, Health Status, and Psychological

Symptoms. Journal of Personality and Social Psychology, 50, 571-579. http://dx.doi.org/10.1037/0022-3514.50.3.571

- [28] Cohen, S., Kamarck, T. and Mermelstein, R. (1983) A Global Measure of Perceived Stress. Journal of Health and Social Behavior, 24, 385-396. <u>http://dx.doi.org/10.2307/2136404</u>
- [29] Mumford, D.B., Bavington, J.T., Bhatnagar, K.S., Hussain, Y., Mirza, S. and Naraghi, M.M. (1991) The Bradford Somatic Inventory. A Multi-Ethnic Inventory of Somatic Symptoms Reported by Anxious and Depressed Patients in Britain and the Indo-Pakistan Subcontinent. *British Journal of Psychology*, **158**, 379-386.
- [30] Mandina, N.M., Longo-Mbenza, B., Wumba, R., Tandu, U.B., Buassa-bu-Tsumbu, B., Mbula, M.M., et al. (2012) Nadir CD4+, Religion, Antiretroviral Therapy, Incidence of Type 2 Diabetes Mellitus, and Increasing Rates of Obesity among Black Africans with HIV Disease. International Journal of General Medicine, 2012, 983-990. http://dx.doi.org/10.2147/IJGM.S32167
- [31] Mananga, L.G., Mampunza, M.M.S., Longo-Mbenza, B., Verbanck, P., Nyirenda, S. and Yassa, P. (2012) Psycho-Social Determinants of Progression to AIDS among Black Africans in Kinshasa, Democratic Republic of Congo. *Psychiatry & Mental Health*, 1, 2.
- [32] Houyoux, J. (1973) Housholds Budgets, Nutrition and way of Life in Kinshasa, Republic of Zaïre. University Press of Zaïre, Kinshasa, 304 p.
- [33] Dancause, K.N., Laplante, D.P., Oremus, C., Fraser, S., Brunet, A. and King, S. (2011) Disaster-Related Prenatal Maternal Stress Influences Birth Outcomes: Project Ice Storm. *Early Human Development*, 87, 813-820. <u>http://dx.doi.org/10.1016/j.earlhumdev.2011.06.007</u>
- [34] Hogue, C.J.R., Parker, C.B., Willinger, M., Temple, M.J.R., Bann, C.M., Silver, R.M., Dudley, D.J., *et al.* (2013) A Population-Based Case-Control Study of Stillbirth: The Relationship of Significant Life Events to the Racial Disparity for African Americans. *American Journal of Epidemiology*, **177**, 755-767. <u>http://dx.doi.org/10.1093/aje/kws381</u>
- [35] Sarfati, Y. (1998) Somatizations. Encyclopedia of Medicine and Surgery. Elsevier, Paris, 6 p.
- [36] Yali, A.M. and Lobel, M. (1999) Coping and Distress in Pregnancy: An Investigation of Medically High Risk Women. Journal of Psychosomatic in Obstetrics and Gynaecology, 20, 39-52. <u>http://dx.doi.org/10.3109/01674829909075575</u>
- [37] Rondó, P.H.C., Ferreira, R.F., Nogueira, F., Ribeiro, M.C.N., Lobert, H. and Artes, R. (2003) Maternal Psychological Stress and Distress as Predictors of Low Birth Weight, Prematurity and Intrauterine Growth Retardation. *European Journal of Clinical Nutrition*, 57, 266-272. <u>http://dx.doi.org/10.1038/sj.ejcn.1601526</u>
- [38] Shaikh, K., Premji, S., Khowaja, K., Tough, S., Kazi, A. and Khowaja, S. (2013) The Relationship between Prenatal Stress, Depression, Cortisol and Preterm Birth: A Review. *Open Journal of Depression*, 2, 24-31. http://dx.doi.org/10.4236/ojd.2013.23006
- [39] Grover, S., Avasthi, A., Kalita, K., Dalal, P.K., Rao, G.P., Chadda, R.K., et al. (2013) IPS Multicentric Study: Functional Somatic Symptoms in Depression. Indian Journal of Psychiatry, 55, 31-40. http://dx.doi.org/10.4103/0019-5545.105502
- [40] Dunkel-Schetter, C. and Glynn, L. (2011) Stress in Pregnancy: Empirical Evidence and Theoretical Issues to Guide Interdisciplinary Researchers. In; Contrada, R. and Baum, A., Eds., *The Handbook of Stress Science: Biology, Psychology, and Health*, Springer Publishing Company, New York, 321-343.