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Original article

The Effect of Maternal Exposure to Textile Industry-Induced Pollution on Pregnancy and Its Outcome

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ABSTRACT

Background: Inside textile mills, pregnant women employees are inevitably exposed to a huge pollution that can result in adverse pregnancy outcomes.

Aim of the work: We aimed to evaluate the potential effect of exposure to textile industry induced pollution, among women textile workers, on pregnancy outcome.

Patients and methods: A case-control study was carried out at Misr Spinning/Weaving Company, El Mahalla El Kubra, Egypt. The exposed and control group consisted of 142, and 143 eligible participants respectively. All underwent full history taking, clinical examination and ultrasound investigations during first, second and third trimesters. Pregnancy outcome was documented.

Results: 64.1% of exposed group' pregnancies were complicated versus 16.1% of control group. Of which pregnancy induced hypertension (PIH, 19.0%), preterm birth (23.2%), term low birth weight (TLBW, 19.7%), and congenital anomalies (2.1%), in contrast to 4.9%, 7.7%, 2.8%, and 0.7% respectively, in the control group.

Conclusion: We concluded from our results that textile induced pollution exposure was significantly associated with adverse pregnancy outcomes (OR=1.652, CI: 1.287-1.954), and this risk was significantly proportional to duration of exposure (OR=2.110, CI: 1.334-3.338).

Keywords: Textile; Pollution; Mahalla Al-Kubra; Congenital anomalies; Low birth weight.

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INTRODUCTION

Textile industry is considered as one of the oldest and most complex industries in the world [1].

In spite of the substantial research and development to minimize pollution potentials of textile processing, the textile industry is one of the biggest polluters on our planet. [2]

It has been estimated that between 1.5 and 6.9 kilogram (kg) of chemicals is needed to produce 1 kg of garment, this implying that the weight of the used chemicals in the textile production process is larger than the weight of the finished garment itself. These toxic chemicals are major sources of pollution[3].

Moreover, the textile manufacturing process contributes significantly as sources of air emissions, which is considered as the second greatest pollution problem in the textile mills. These emissions include dust, acid vapors, oil mists, odors and boiler exhausts. Carbon monoxide (CO), nitrogen dioxides (NO_2), sulfur dioxide (SO_2), ozone (O_3), lead, and particulate matters (PMS) are well identified air emissions from textile process [4].

Large scale of epidemiological studies has reported a critical link between indoor industrial pollution and an increased incidence, and aggravated severity of different diseases. Moreover, occupational noise exposure has been recently emerged as one of the most influential and a harmful physical factor at workplaces, linked with a wide range of negative health effects [5].

The women textile workers are exposed inside textile mills, to high noise levels exceed those recommended by the National Institute of Occupational Safety and Health [6]. So, workers in textile factories are inevitably exposed to huge amount of pollution. Fetuses, in particular, are considered to be highly susceptible to a variety of toxicants, due to their exposure pattern and physiologic immaturity, especially during periods of high cell proliferation, differentiation, and rapid organ development. Indeed, pregnancy outcome is determined by the ability of the fetus to thrive, which depends on a complex combination of genetic, social, and environmental factors [7].

The daily prenatal maternal exposure to the textile emitted pollutants, including different neurotoxic, carcinogenic, and developmental toxic chemicals, might be associated with more serious, permanent damage to the fetuses [7].

AIM OF THE WORK

We aimed to evaluate the potential effect of maternal indoor exposure to textile industry induced pollution, among women textile workers, on pregnancy outcome, at Misr Spinning and Weaving Company in El Mahalla El Kubra, Egypt.

PATIENTS AND METHODS

This case-control study was conducted by Damietta Faculty of Medicine, Al- Azhar University, in corporation with the Egyptian Ministry of Health, at Al-Mahalah Al-Kubra, during the period from June 2018 to June 2019.

Three hundred pregnant women were included and classified into two groups. **Group 1 (Exposed group):** included 150 pregnant women working at Misr Spinning and Weaving Company in Al-Mahalla Al-Kubra. Eight pregnancies were excluded (three were complicated by gestational diabetes, three complicated by abortion, while other two were complicated by intrauterine fetal deaths). **Group II (Non-exposed, Control group):** included 150 pregnant women work in non- textile polluted area. Seven pregnancies were excluded (two underwent abortion, two of them did not complete antenatal follow up during the study and three delivered at their village).

Inclusion criteria: Among women textile workers, in Al-Mahalla Al-Kubra, Egypt, had a history of indoor-exposure to occupational pollution during pregnancy, singleton intrauterine gestation, at 18-35 years old.

Exclusion criteria*:* Presence of consanguinity, multiple pregnancies, and women with history of chronic medical disorder, obstetric history show past or recent history of a pregnancy complication, pervious history of infant with congenital malformation to exclude other factors that may affect pregnancy outcome, history of drug medication intake during present pregnancy.

An informed consent was signed by each participant, then, the following was done to each participant: History taking, general and local examination. In addition, ultrasound was carried out

at first, second and third trimesters.

First-trimester ultrasound scan: It was performed at (11–14 weeks) by trans-abdominal ultrasound, soft markers plus the fetal nuchal translucency, with Screening for chromosomal anomalies.

Second and third trimester ultrasound scans. Fetal anomaly scan at 18- 22 week. Uterine artery Doppler was performed at 20-24 weeks of gestation. Doppler on umbilical artery to all suspected cases. Screening for gestational diabetes and maternal body mass index (BMI) were performed between 24 –30 weeks of gestation.

Statistical Analysis: The collected data was organized, tabulated and statistically analyzed using statistical package for social science (SPSS) version 22 (SPSS INC, Chicago, USA) Running on IBM compatible computer. For qualitative data, the frequency and percent distributions were calculated, while mean, standard deviation (SD) were calculated for quantitative data. For comparison between two groups, the independent samples (t) test was used. Pearson correlation co-efficient (r-test) was used for correlating different variable. For all tests p value ≤0.05 were considered significant.

RESULTS

Table (1) revealed non-significant differences in mean intra-pregnancy BMI, and age between exposed and control groups. Mean duration of exposure to textile pollution in the exposed group was 5±1.6 years; 21.8 % exposed less than 3 years; 66.9 % exposed between 3 and 9 years and 11.3% exposed more than 9 years.

Longer maternal hospital stay was significantly associated with exposed group (35.4±11.6 hour) when compared to control group (28.9±7.2 hour). Otherwise, no significant differences were found regarding gravidity, and delivery mode between studied groups (Table 2).

Regarding the gestational age, our results revealed that the exposed group was significantly associated with younger gestational age (36.8±2.8) completed weeks of gestation when compared to control group (38.2±1.3). Similarly, birth weight (g) data analysis was revealed significantly lowered birth weight in the exposed group (2835.9±491.4g), compared to control group (3204.3±322.6 g).Where 28.9% of textile workers delivered babies with LBW (< 2.500g) in contrast to 4.9% of the age matched control. 71.1% of exposed group had babies weights ranged (2.500-4.000g), compared to 95.1% in the control group (Table 3).

The textile workers in the exposed group were significantly associated with maternal and neonatal complications when compared to their controls. 35.9% of textile workers exhibited normal maternal and neonatal outcomes, while 64.1% revealed complications with their pregnancies, in contrast to 83.9% and 16.1% in the control group respectively. PIH was recorded as outcome in 19.0% of pregnancies in the textile workers versus 4.9% in the control group. 23.2% of pregnancies in the exposed group were terminated preterm in contrast to 7.7% of controls. 19.7% of pregnancies in the exposed group yielded LBW babies at their term, compared to only 2.8% of the control group. 2.1% of exposed group' pregnancies were complicated by congenital anomalies; anencephaly, hydrocephaly, and atrio-ventricular septal defect (Figures 1, 2, and 4), while only a case of omphalocele (Figure 3) with a percent of 0.7% of pregnancy outcomes, was reported among the control group (Table 4).

The most potent variables that were associated with significant increased risk of abnormal pregnancy outcomes in univariable analysis, were younger maternal age; OR 0.867 (95% CI, 0.823-0.912), gestational age OR 0.352 (95% CI, 0.248-0.499), textile exposure OR 1.434 (95% CI, 1.161-1.771) and more evidently longer duration of textile exposure OR 0.894 (95% CI, 0.830-0.964) (Table 5).

Younger maternal age OR 0.522 (95% CI, 0.386-0.707); gestational age, OR 0.356 (95% CI, 0.232-0.547); textile exposure OR 1.652 (95% CI, 1.287-1.954); and longer duration of textile exposure OR 2.110 (95% CI, 1.334-3.338) were considered independent prognostic factors for abnormal pregnancy outcomes (Table 6).

Table (1): Maternal age, body mass index, and duration of exposure (years), in the studied groups

Characteristics		Control N=143		Exposed N=142		*p*
Age (years)	mean±SD	24.6 ± 4.6		23.8 ± 4.6		0.282[T]
		N	%	N	%	
	< 20	19	13.3	53	37.3	<0.001[C]
	20-30	102	71.3	74	52.1	
	> 30	22	15.4	15	10.6	
Pregnancy BMI	mean±SD	30.5 ± 5.1		29.6 ± 5.8		0.153[T]
		N	%	N	%	
	Underweight	3	2.1	17	12	0.336[F]
	Normal	37	25.9	28	19.7	
	Overweight / Obese	103	72	97	68.3	
Duration of exposure (Years)	Up to 3			31	21.8	
	3-9			95	66.9	
	> 9			16	11.3	
	mean±SD			5±1.6		

SD: standard deviation, T: student t test, C: Chi square, F: Fisher exact test. The data was represented as mean±SD, number (N), percent (%).

Table (2): Obstetric data in the studied groups (represented as number, and percent, unless otherwise mentioned).

		Control N=143		Exposed N=142		p
Parity	**Primigravida**	**N**	**%**	**N**	**%**	
		59	41.3	73	51.4	0.086[C]
	Multigravida	84	49.7	69	48.6	
Mode of delivery	**CS**	64	44.8	63	44.4	1.000[C]
	Vaginal	79	55.2	79	55.6	
Maternal hospital stay (hours, mean±SD)		28.9±7.2		35.4±11.6*		**<0.001[T]**

SD: standard deviation, T: student t test, C: Chi square. *Denotes statistical significance P≤0.05 compared to the control group.

Table (3). Neonatal data in the studied groups.

Parameters			Control N=143		Exposed N=142		*p*
Gestational age (Completed weeks)		mean±SD	38.2±1.3		36.8±2.8*		<0.001[T]
Birth weight (g)	mean±SD		3204.3±322.6		2835.9±491.4*		<0.001[T]
	< 2,500	N, %	7	4.9	41	28.9	<0.001[T]
	2,500–4,000	N, %	136	95.1	101	71.1	

SD, standard deviation; the data was represented as mean ± SD, number (N), percent (%).T, student t test. *Denotes statistical significance P≤0.05 compared to the control group.

Table (4): Maternal and neonatal outcomes (number, percent; N, %) in the studied groups.

Outcome		**Control N=143**		**Exposed N=142**		***p***
		N	%	N	%	
Normal outcome		120	83.9	51	35.9	**<0.001[F]**
PIH		7	4.9	27	19.0	
Preterm birth		11	7.7	33	23.2	
TLBW	**SGA**	4	2.8	11	7.7	
	IUGR	0	0	17	12	
Congenital anomalies		1	0.7	3	2.1	

PIH, Pregnancy induced hypertension; TLBW, Term Low birth weight; F; Fisher exact test. The data was represented as number (N), percent (%).

Table (5): Univariable regression analysis for prediction of abnormal pregnancy outcome.

Maternal variable	*p*	OR	95% CI	
Maternal age	**<0.001**	0.867	0.823	0.912
BMI	0.078	0.966	0.929	1.004
Gestational age	**<0.001**	0.352	0.248	0.499
Gravidity	0.068	0.673	0.440	1.030
Delivery mode	0.119	0.595	0.385	1.920
Textile exposure	**0.001**	1.434	1.161	1.771
Duration of exposure	**0.003**	0.894	0.830	0.964

OR, odds ratio; CI, confidence interval; BMI, Body mass index. Statistical significance was considered at P≤0.05.

Table (6): Multivariable regression analysis for prediction of abnormal pregnancy outcomes.

Variables	*p*	OR	95% CI	
Maternal age	**<0.001**	0.522	0.386	0.707
Gestational age	**<0.001**	0.356	0.232	0.547
Textile exposure	0.002	1.652	1.287	1.954
Duration of exposure	0.001	2.110	1.334	3.338

OR, odds ratio; CI, confidence interval. Statistical significance was considered at P≤0.05.

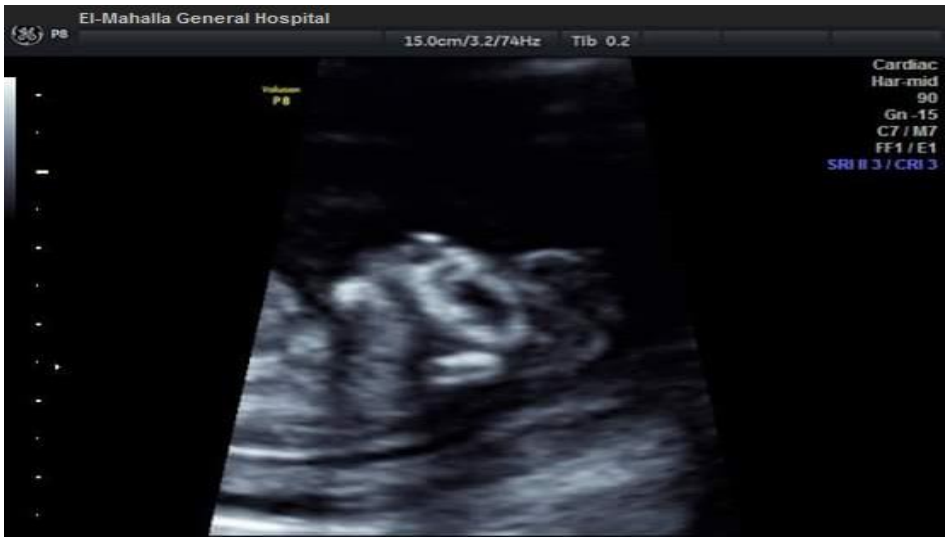

Figure (1): Trans abdominal Ultrasound of anencephaly at gestational age 18 weeks.

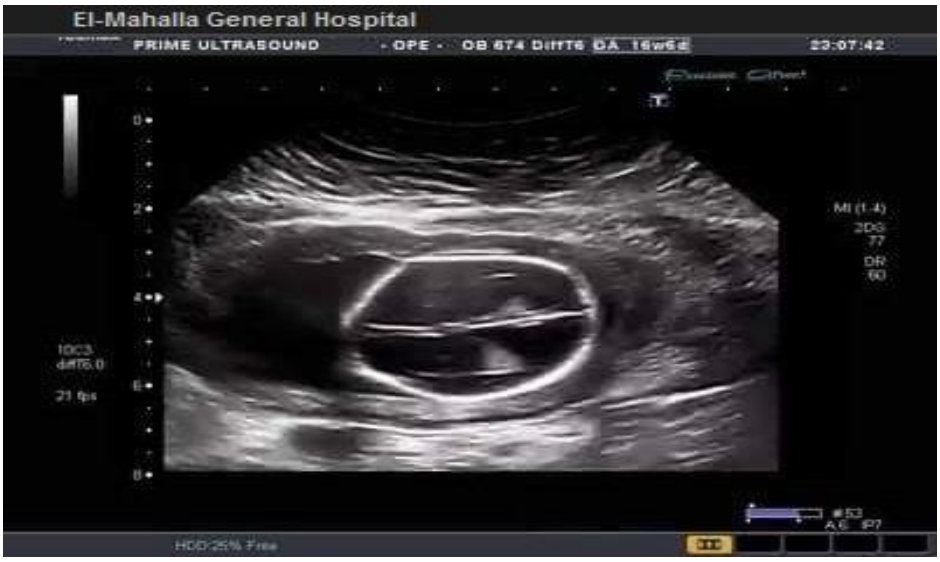

Figure (2): Trans abdominal Ultrasound of hydrocephaly at gestational age 16w$^{+6d.}$

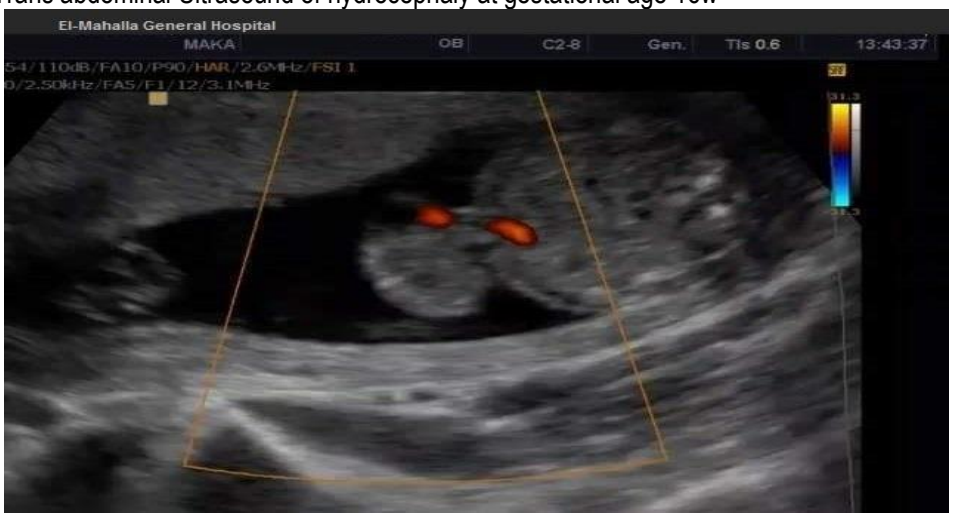

Figure (3): Trans abdominal Ultrasound of omphalocele at gestational age 22 weeks.

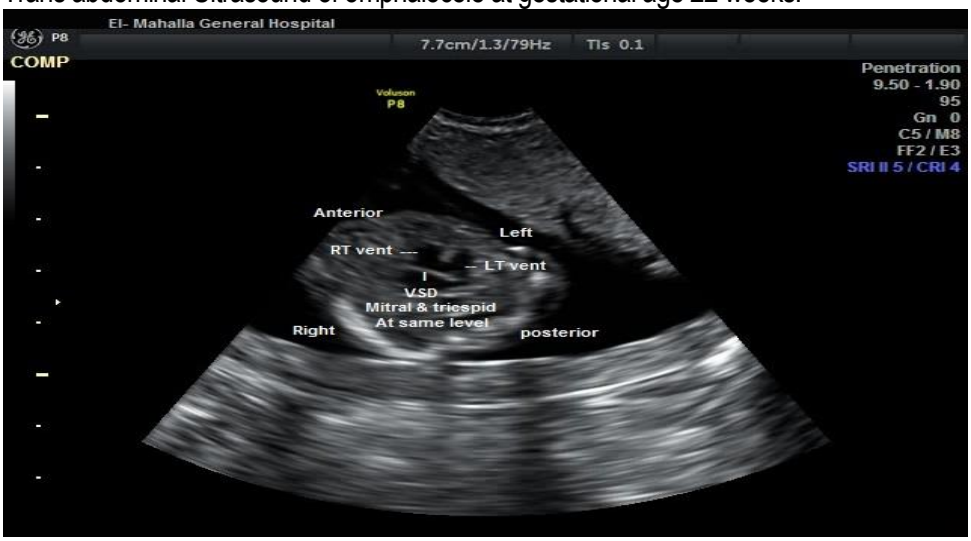

Figure (4 A): Trans vaginal Ultrasound of atrio ventricular septal defect at gestational age 17 week.

Figure (4 B): A color Doppler of atrio ventricular septal defect at gestational age 17 week.

DISCUSSION

The results of our study revealed significant association between exposures to the textile induced pollution among the textile pregnant women and our adverse pregnancy outcomes, PIH, preterm birth, TLBW, and congenital anomalies.

Consistent with our results, **Mobashera et al.**[8], documented first trimester CO exposure to pregnant women increased the odds of developing PIH. A link between CO exposure and PIH has been further supported by **Rudra et al.**[9], who ensured a positive strong association between CO and the odds of PIH. **Vigeh et al.** [10] reported twice the rate of PIH in mothers exposed to higher CO levels than control mothers (OR= 2.02, 95% CI= 1.35, 3.03).

Earlier studies confirmed increased risk of developing preeclampsia by about 42%, in women exposed in the highest $PM_{2.5}$ during their pregnancy[9]**.** Similarly**,** in a prospective cohort study in the Netherlands, **van den Hooven**[11], explored a positive association between risk of PIH and PM_{10} concentrations (OR 1.72 (95% CI 1.12 to 2.63).

In contrast to the strong association between PIH and first trimester exposure to PM2.5, and PM_{10}, other lines of evidence addressed significant link between PIH, and IUFR and ozone (O_3) exposure in the second trimester [8].

A well-documented potential mechanism whereby pollutant components can increase BP is superoxide-mediated inhibition of the actions of nitrous oxide in inducing vasodilatation [12]**.**

Previous reports documented a close relation between air pollution, and PIH risk, mostly through systemic/ vascular inflammation. Especially during the first trimester that represents a critical window of susceptibility PIH, during which trophoblast invasion into the maternal decidua takes places to establish efficient fetal blood supply [13]**.**

The dysregulated autonomic nervous system (ANS) with an activated sympathetic tone, may better explain the combined effects of air pollution and noise on pathogenesis of hypertension [14]**.**

Previous convincing evidence pointed out that chronic occupational exposure to ≥ 80 – 85 dB, as typically occurs during daily activities in textile mill, is associated with significantly higher risk for LBW and SGA[15]**.**

Yiming et al.[16] in a prevalence study of hypertension in a group of 1101 female workers in a textile mill, reported by logistic regression that exposure to noise is a significant determinant of prevalence of hypertension, but third in order of importance behind family history of hypertension and use of salt.

Our study revealed a significant risk regarding textile industry exposure and the incidence of preterm birth, where 23.2% of exposed group, their pregnancies complicated by preterm birth.

Ritz, and Wilhelm[17] lend support to the concept of air pollution is a risk preterm birth factor. Increased risk of preterm birth has been previously

recorded to be associated with exposure to air pollutants particulate matters (PM_{10}) and CO [18] and (PM2.5), and So_2 [19].

Previous work has indicated that pollutants absorption may induce several pathophysiological circuits including inflammation, oxidative stress, cell apoptosis, endothelial dysfunction and hemo-dynamic responses, which predispose to preterm birth [20].

In a large study in China suggested that rotating shift-work and working in a squatting position may increase the risk of preterm or LBW deliveries [21].

Salam et al. [22]; found that first trimester exposure to CO was associated with a 20% increased risk of IUGR. Similarly **Liu et al.**[23] reported statistically significant increase in the risk of IUGR with increased CO exposure in the 1st trimester.

Previous associations have reported more consistently the first and third trimesters. Exposures during first trimester may result in disruption of placental formation and its function leading to IUGR while exposures during later pregnancy may interfere with the fastest period the body mass accumulation of fetus [24].

During his a cohort on 14000 pregnant women, **Farrow et al.**[25] reported working in the textile trade, was recorded among the major job groups with the LBWS. The oxidative stress induced by pollution could result DNA damage, disrupting DNA transcription, resulting in decreased capacity of the feto–placental exchange of nutrients and oxygen and compromised fetal growth [26].

Ritz et al. [24] suggested a possible gene–environment interaction enhancing risk of congenital malformation. The continuous exposure to multiple air pollutants especially $PM_{2.5, 10}$, was associated with immediate vasoconstriction and endothelial functions could be considered as an intervening pathway in subsequent impact on fetal growth [12].

Our results revealed higher prevalence of anomalies among new-borns to textile worker, with higher proportions of malformations in the nervous and circulatory systems.

A significant association was detected among cases with congenital birth defects whose mothers had been exposed textile occupation [27].

A significant risk of multiple fetal anomalies was closely linked with textile dye workers with hydrocephaly, ventricular septal defect and congenital heart diseases, among the most frequently encountered defects of the twenty cases with multiple birth defects recorded in the textile dye workers, through registry-based case-control study carried out by [28].

Shi and Chia[29] identified a significant risk between textile dye workers and multiple anomalies (adjusted OR 1.9, 99% CI 1.0–3.8). **Khattak et al.**[30], ranked working women in textile and clothing industries among the most important women-dominated occupations with potential chemical exposures, involving exposure to organic solvents, with its deleterious health effects, including the well documented teratogenicity and an increased risk of major fetal anomalies.

Pregnant women working under persistent occupational exposure to organic solvents, as did the textile workers [31], have been reported to be at high risk for delivering baby with congenital malformations, most frequently, central nervous system, coronary disorders, and congenital deafness [32].

McMartin et al.[33] documented an overall average value (OR=1.64 95% CI=1.16-2.30) during evaluating maternal occupational organic solvents exposure and the associated risk of major congenital anomalies. Higher risk of anencephaly, NTDs and spina bifida subtypes was reported in pregnant women from counties with textile industrial development than in those from other counties, with released solvents have been accused [32, 34].

Castilla et al.[34] declared that the textile industry was ranked as industry uses diverse potentially teratogenic pollutants with increased risk of congenital anomalies; especially anomalies related the central nervous system on top of which anencephaly and microcephaly.

Bianchi et al.[28], reported hydrocephaly , cleft palate and lip , absent diaphragm, oesophageal atresia, absent auditory canal, spina bifida, low set

ears, and ventricular septal defect, among the multiple congenital anomalies that have been previously recorded in textile dye-workers.

Unfortunately, the chemical substances could be transferred directly to fetal circulation if they are not metabolized either by placental or maternal metabolism. The fetus attempt to metabolize these chemical pollutants, by enzyme activity, mainly through fetal liver, however other organs such as kidneys, adrenal glands, lungs, and brain may also be involved [39].

The lowered capacity of the fetus for detoxification and excretion with much weaker enzyme system, may lead to excessively higher levels of these harmful pollutants in fetal blood as compared to those in the maternal circulation. The condition is much worsened by the fact that fetal blood-brain barrier is also immature, leading to enhanced vulnerability of the fetal brain to damaging effects of these toxic chemicals [36].

Maternal exposure to dyes has been reported to be a significant risk factor for congenital septal defects [37]. **Khattak** [30] declared in his study that pregnant women exposed occupationally to organic solvents, especially those in the textile industries, had a 13-fold risk of major malformations as well increased risk for miscarriages in their previous pregnancies while working with organic solvents.

In a previous study performed in China on 10 542 women between 2010–2012, **Jin et al.**[38] documented a positive associations for congenital malformations and the maternal exposures to (PM_{10}), (NO_2), and (SO_2) (OR $_{1st\ trimester}$ 3.96, (CI): 1.36 - 11.53; OR $_{2nd\ trimester}$ 3.59, CI: 1.57, 8.22; OR $_{entire\ pregnancy}$ 2.09, 95% CI: 1.21- 3.62)

Conclusion: The findings reported in this study indicated that pregnant women exposed to textile induced pollutions inside Misr Spinning and Weaving Company at Al Mahalla Al Kubra, at increased risk of the adverse pregnancy outcomes with a particular TLBW, preterm birth, hypertension, and congenital anomalies.

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