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Effect of Environmental Factors on Reproductive Health: A Prospective Comparative Study

Sarita Nehra[1], Deepika Jhuriya[2], Shushil Kudi[3], Charu Taneja[4]

[1]Senior Resident, Department of Obstetrics and Gynaecology, ESIC Medical College and Hospital, Jaipur, Rajasthan, India
[2]Senior Resident, Department of Obstetrics and Gynaecology, ESIC Medical College and Hospital, Jaipur, Rajasthan, India
[3]Senior Resident, Department of Community Medicine, Government Medical College, Nagaur, Rajasthan, India
[4]Associate Professor, Department of Anatomy, Government Medical College, Nagaur, Rajasthan, IndiaORCID ID:- (0000-0001-5982-4539)

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Corresponding Author:

Dr Sarita Nehra
Senior Resident, Department of Obstetrics and Gynaecology, ESIC Medical College and Hospital, Jaipur, Rajasthan, India
Email:
saritanehra411@gmail.com

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ABSTRACT

Background and Aim: Environmental and occupational exposures may adversely affect female fertility and pregnancy outcomes through endocrine, oxidative, inflammatory, and immunological mechanisms. The present study was conducted to identify and compare the impact of environmental factors on reproductive health among factory workers and non-working women. **Materials and Methods**: This comparative prospective observational study included 500 women aged 18-40 years presenting to the Department of Obstetrics and Gynaecology, ESIC Model Hospital, Jaipur. Group 1 consisted of 250 factory workers and Group 2 consisted of 250 household women. The outcomes studied were infertility, early pregnancy loss, preterm labour, and other medical disorders. **Results**: The age distribution in the two groups was comparable, with no statistically significant difference ($p>0.05$). Among factory workers, the largest proportions were employed in chemical factories (34.0%) and textile industries (31.2%). Adverse reproductive outcomes were significantly more frequent among factory workers than among household women: infertility 6.4% vs 2.0% ($p=0.014$), early pregnancy loss 8.4% vs 2.8% ($p=0.009$), preterm labour 6.8% vs 1.6% ($p=0.004$), and other medical disorders 14.8% vs 6.0% ($p=0.001$). **Conclusion**: Factory-based environmental exposure was associated with a higher burden of adverse reproductive outcomes. Larger exposure-specific studies are needed to clarify the responsible toxicants and the mechanisms by which they affect female reproductive health.

Keywords*: environmental exposure, reproductive health, factory workers, infertility, early pregnancy loss, preterm labour.*

INTRODUCTION

Female reproductive function is influenced by multiple biological and environmental determinants. In recent years, growing attention has focused on the role of occupational and environmental toxicants in altering fertility, menstrual function, conception, implantation, and pregnancy outcome.[1]

Environmental chemicals may disrupt reproduction directly by interfering with endocrine signaling and ovarian function or indirectly by inducing oxidative stress, immune dysregulation, inflammation, and altered cellular repair pathways.[2]

Reviews of the available literature indicate that endocrine-disrupting chemicals, solvents, pesticides, heavy metals, plastics-related compounds, and air pollutants may all contribute to female reproductive dysfunction and poorer pregnancy outcomes.[3]

Among the commonly discussed toxicants, bisphenol A and related compounds have been associated with altered hypothalamic-pituitary-ovarian activity, reduced oocyte quality, impaired uterine receptivity, and infertility-related disorders.[4]

Similarly, broader reviews on environmental pollution have highlighted associations with reduced fertility, miscarriage, preterm birth, and other adverse obstetric outcomes, especially in women with repeated workplace exposure.[5]

The present study was undertaken to compare reproductive outcomes in women working in factories with those in non-working household women attending a tertiary obstetrics and gynaecology department.[6]

MATERIALS AND METHODS

2.1 Study design and participants

This was a comparative prospective observational study conducted in the Department of Obstetrics and Gynaecology, ESIC Model Hospital, Jaipur. A total of 500 women between 18 and 40 years of age were included. Group 1 comprised 250 women working in factories, while Group 2 comprised 250 household women who were not engaged in factory work.

The study compared reproductive outcomes and related morbidity in the two groups. The principal outcomes evaluated were infertility, early pregnancy losses, preterm labour, and other medical disorders. Within the factory-worker group, participants were further classified according to the type of industry in which they were employed.

2.2 Statistical analysis

Data were summarized as frequencies and percentages. Group-wise comparisons were interpreted using the p values available from the study dataset. A p value of less than 0.05 was considered statistically significant.

RESULTS

Table 1. Age-wise distribution of study participants

Age group (years)	Group 1 Factory workers n (%)	Group 2 Household women n (%)
18-25	38 (15.2)	33 (13.2)
26-30	102 (40.8)	107 (42.8)
31-35	86 (34.4)	82 (32.8)
36-40	24 (9.6)	28 (11.2)

The two groups were comparable with respect to age distribution. The majority of women in both groups belonged to the 26-30 years age group, followed by the 31-35 years age group. No statistically significant intergroup difference was observed ($p>0.05$).

Table 2. Distribution of factory workers according to type of industry

Type of industry	Number	Percentage
Mining industry	54	21.6
Chemical factory	85	34.0
Paper industry	33	13.2
Textile industry	78	31.2

Among factory workers, the highest proportion was employed in chemical factories (34.0%), followed closely by textile industries (31.2%). Women from mining and paper industries comprised 21.6% and 13.2% of the exposed group, respectively.

Table 3. Comparison of reproductive outcomes between the two groups

Outcome	Group 1 n (%)	Group 2 n (%)	P value
Infertility	16 (6.4)	5 (2.0)	0.014
Early pregnancy losses	21 (8.4)	7 (2.8)	0.009
Preterm labour	17 (6.8)	4 (1.6)	0.004
Other medical	37 (14.8)	15 (6.0)	0.001

disorders			

All adverse reproductive outcomes studied were more common in factory workers than in household women. Statistically significant differences were observed for infertility, early pregnancy losses, preterm labour, and other medical disorders, indicating a consistently higher burden of reproductive morbidity among women with workplace exposure.

DISCUSSION

The present study demonstrated a higher frequency of adverse reproductive outcomes among factory workers as compared with household women. This pattern is biologically plausible and is consistent with published reviews showing that occupational and environmental exposures can adversely influence female reproductive function and pregnancy outcome.[1]

Occupational reproductive hazards may operate through endocrine disruption, oxidative injury, altered ovarian steroidogenesis, inflammatory changes, and impaired implantation or placental function. These mechanisms may translate clinically into infertility, early pregnancy loss, and preterm birth.[2]

Our finding of a greater burden of infertility among factory workers is in line with earlier evidence linking occupational chemical and physical exposures with an increased risk of female infertility.[7]

The observed excess of early pregnancy losses in exposed women is also noteworthy. Previous occupational studies, including investigations among textile workers, have suggested higher miscarriage risk in women with dust and chemical exposures, supporting the direction of the association seen in our study.[8]

The large representation of chemical and textile industry workers in the exposed group is important because these work environments may involve repeated contact with solvents, dyes, synthetic fibers, particulate matter, plasticizers, and other endocrine-disrupting compounds. Such contaminants have been implicated in altered ovarian function, impaired oocyte quality, uterine dysfunction, and reduced fertility potential.[6]

Environmental pollution more broadly has also been linked to poorer reproductive performance in women, and the adverse effects are believed to be cumulative, especially when exposure is recurrent and long term.[5]

Bisphenol A and related chemicals have received particular attention because of their ability to interfere with hormonal signaling and uterine receptivity, which may contribute to infertility and abnormal pregnancy outcomes.[4]

The present study is strengthened by its comparative design and equal group size. However, it also has important limitations. Specific exposure assessment, duration of work, dose-response relationships, and potential confounders such as nutritional status, socioeconomic variables, and pre-existing medical conditions were not stratified in the available dataset. Therefore, the findings should be interpreted as evidence of association rather than proof of causation.

CONCLUSION

Women working in factory settings had higher rates of infertility, early pregnancy loss, preterm labour, and other medical disorders than non-working household women. These findings suggest that environmental and occupational exposure may play an important role in female reproductive health. Further studies with detailed toxicant profiling and exposure quantification are needed to identify the specific agents involved and to inform preventive workplace strategies.

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