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Review

# Road Traffic Pollution, Motor Driving Occupation and Human Reproductive Health- An Overview

Sunil Kumar *

A-10, Radhey Kunj Apartment, B/h Circuit House, Shahi Baugh, Ahmedabad-38 00 04, India; E-Mails: sunilnioh@gmail.com; agarwalsk1955@gmail.com

* **Correspondence:** Sunil Kumar; E-Mails: sunilnioh@gmail.com; agarwalsk1955@gmail.com

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**Abstract**

Generally, automobile vehicles discharge specific contaminants into the surrounding environment during operation, which might affect human and reproductive health. Additionally, a more extended period of sitting while driving is linked with the manifestation of heat in the pelvic region, which might also impact the driver's semen quality. The data on motor traffic pollution, sitting posture duration while driving, and their impact on reproduction was collected by searching various websites. The data on the effects of traffic-related contamination on motorway tollgate workers indicated an alteration in semen quality parameters to some extent compared to control, i.e., nonexposed to traffic-connected pollution. Still, the data on other male reproductive endpoints are scanty. Some data on the impact of traffic-related air pollution (TRAP) on pregnancy outcomes are accessible based on the studies mostly carried out in women residing close to the main road, which suggests the adverse impact of TRAP on pregnancy outcomes. The comprehensive data on drivers with reverence to professional driving i.e., long duration of sitting while driving and reproductive health, is scanty and needs more studies. The available information suggests that vehicular pollution might affect the semen quality and pregnancy outcome to some extent. At the same time, the impact on the other reproductive endpoints of both sexes is inconsistent, and

additional data is needed to conclude these aspects of human reproduction. Further, there is a need to adopt better efficient fuel engines to reduce TRAPs or use alternate technology or fuel to petrol/diesel.

**Keywords**
Road traffic pollution; reproductive health; reproductive hormones; scrotal temperature; semen quality; sitting posture; pregnancy outcome

## 1. Introduction

Generally, motor vehicles emit numerous pollutants during operation, which could affect the quality of the surrounding air that the living beings inhale. The criteria contaminants emissions produced during fuel combustion by the motor vehicle consist of nitrogen oxides ($NO_x$), volatile organic compounds (VOCs), carbon monoxide (CO), and particulate matter (PM) of size <10 microns (µm) and <2.5 µm and black carbon [1]. Motor vehicles are reported to be an essential source of PM (<2.5 µm in diameter), $NO_2$, Polycyclic aromatic hydrocarbons (PAHs), and VOCs exposure [2]. Traffic-related air contamination is ubiquitous in cities and a critical public health issue resulting in premature mortality and the manifestation of various diseases. The importance and relevance of traffic-related air pollution (TRAP) continue to elevate with urbanization, fast population growth, and demand for motorized vehicles for travel and goods transport. Furthermore, the list of unfavorable health consequences related to TRAP exposure continues to grow, which comprises cognitive deterioration, neurodegenerative diseases, and several hostile metabolic disorders like diabetes and obesity [3]. A recent study showed that human health and environmental risks are growing following the manifestation of air pollution connected with vehicular and industrial emissions in which particulate matter (PM) is a significant component, and studies on the health impacts and mortality induced by PM showed a strong positive relationship amid fine and ultra-fine particles exposure and cardio-vascular, hypertension, obesity, type 2 diabetes mellitus, mortality, and cancer risks [4]. People working on roadsides or vehicular congested/traffic areas, such as toll gate operators, workers of roadside restaurants/hotels/motels, wayside shopkeepers and drivers, conductors, etc., are exposed to TRAP. Some of the vulnerable groups of society, i.e., children and pregnant women, are also exposed to TRAP as some of them reside in the vicinity of higher traffic areas. Exposure to traffic-related pollutants may affect the human health or even reproductive health of both sexes to some extent because TRAP contains several potential reproductive toxic components.

A cross-sectional study examined the associations of a duration of driving concerning health behaviors and outcomes using data from the social, economic, and environmental factors and advised that a longer duration of driving was related to higher odds for smoking, inadequate physical activity, petite sleep, obesity, inferior physical or mental health [5]. Professional drivers with long periods of sitting position while driving for a long distance may be related to the hostile impact on the driver's reproductive health. No systematic, comprehensive review is available on road traffic pollution, motor driving, and human reproductive health of both sexes and on pregnancy or

outcome. Thus, an effort has been made to provide a comprehensive overview of human reproduction and outcomes concerning motor vehicle-related pollution or associated variables.

## 2. Materials and Methods

The related literature on the review was collected through the internet using different keywords like motor vehicle emission, traffic-associated pollution or variables and reproductive health of men, women, semen quality, fertility, impact on pregnancy or outcome, menstruation cycle, libido, puberty, erectile dysfunction, reproductive hormones, preterm birth, spontaneous abortion, miscarriage, etc. which were associated with the theme of this overview by searching different websites i.e., Google, Google Scholar, PubMed, PubChem, etc. The data on the impact of traffic-related pollution upon human reproduction were also collected from the people who resided nearer to the highways/ freeways or motor vehicular congested areas as they are subjected to higher doses of traffic-linked pollutants repeatedly for a longer duration of time by using review related different keywords. In addition, the impact of workers' sitting posture or whole-body vibration (wbv) associated with driving a motor vehicle for a longer time on reproductive health was also collected. A total of eighty relevant articles were included in this manuscript.

The data were presented into three broad sections 1) Road traffic-associated pollutants and male reproduction 2) Road traffic-associated pollutants and female reproduction and pregnancy outcome 3) The Impact of professional motor driving variables on reproductive health. The data were further summarized in Table 1, Table 2 and Table 3 for quick understanding or appraisal. The flow diagram of data collection and outlines of the presentation of information in this MS are shown in Figure 1. A few papers on animal studies were also included in the absence or scanty clinical data.

**Table 1** Road traffic pollutants or driving profession and male reproduction.

| Sl No | Exposure | Effects | Reference |
|---|---|---|---|
| 1 | Semen quality of tollgates workers. | Sperm count, serum LH, FSH, & testosterone levels were in the normal range, while sperm motility, functional tests, and kinetics were lower in toll-workers. | De Rosa *et al.* (2003) [6] |
| 2 | Semen quality of toll collectors and control. | Abnormal sperm count, motility, and normal morphology significantly differ among two groups. | Guvan *et al.* (2008) [7] |
| 3 | Sperm chromatin/ DNA integrity in tollgate workers & control | Serum LH, FSH, & testosterone levels are in a normal range. Sperm concentration, total count, motility, and normal forms were notably lower and considerably higher % of sperm with damaged chromatin & DNA fragmentation in toll workers. | Calogero *et al.* (2011) [8] |
| 4 | Semen quality of traffic police officers, Peru where leaded gasoline was in use | Current PbB-related declines in semen parameter (sperm morphology, count & concentration), Sperm motility & viability differed significantly amid < or =40 µg/dL & >40 µg/dL of PbB groups. | Eibenstein-er *et al.* (2005) [9] |

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|---|---|---|---|
| 5 | Effect of gasoline emission, diesel exhaust, PM, heat, noise on reproduction of auto drivers. | A noteworthy reduction in serum testosterone, elevation in cortisol level and more reproductive health problems (declined libido, erectile dysfunction, ejaculatory snags, infertility) noted in rickshaw drivers. | Nabi *et al.* (2014) [10] |
| 6 | Housing distance to roadways, & semen quality. | Not related to sperm features or serum reproductive hormones of men attending a fertility clinic. | Nassan *et al.* (2018) [11] |
| 7 | Taxi drivers compared to the controls | A significantly lower normal sperm (45.8% vs. 64.0%) in driver. Other parameters did not differ. Subjects with poor semen quality showed longer TTP. | Figà-Talamanca *et al.* (1996) [12] |
| 8 | Infertile auto-mobile drivers & infertile non-drivers & normal. | Frequency of abnormal semen quality higher in infertile drivers than infertile non-drivers & normal men. Semen anomalies are higher in drivers with more than 8 yrs of experience. | Zhao *et al.* (2003) [13] |
| 9 | Data on auto-mobile exhaust on male reproductive function & allied proteins reviewed | Hostile effects of VE on male reproduction by affecting organs wt., sperm quality, causing oxidative stress. VE causes noteworthy changes in patterns of protein expression, main components of spermatogenesis & synthesis of testosterone. | Rengaraj *et al.* (2015) [14] |

**PM-**Particulate matter**; PbB-**Blood lead; **Yrs-**Years; **Wby-**Whole body vibration; **VE-**Vehicular exhaust; **TTP-**Time to pregnancy.

**Table 2** Road traffic-associated pollutants and female reproduction or outcome.

| Sl. No | Exposure | Effects | Reference |
|---|---|---|---|
| 1 | Prenatal TRAP, & home environmental factors | Both outdoor TRAP & indoor mold/ dampness play a role in PTB, & interaction in early pregnancy, raises PTB risk. | Lu *et al.* (2019) [15] |
| 2 | Traffic-related air pollution & PTB | Higher odds of PTB are noted in those exposed to highest quartile of each pollutant from 2$^{nd}$ trimester to end of pregnancy. Links sturdier those living in low socio-economic area. | Padula *et al.* (2014) [16] |
| 3 | Residential exposure to local traffic pollution | Higher risks of pre-eclampsia & PTB from maternal exposure to locally traffic-generated $NO_x$ & $PM_{2.5}$. | Wu *et al.* (2009) [17] |
| 4 | TRAPs exposure judged via ambient station (CO, NO, $NO_2$, & $NO_x$). | Elevated risks for pre-eclampsia, PTB, very PTB with respect to exposures to traffic air pollutants. | Wu *et al*. (2011) [18] |

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|---|---|---|---|
| 5 | Residence during pregnancy, and traffic density & mobility | Risk for premature delivery with community factors (urban/ suburban, traffic density, travel to work in higher vehicle density area). | Kahr *et al.* (2016) [19] |
| 6 | Traffic-related air toxins & PTB | Odds of PTB elevated 6-21% per inter-quartile elevation in exposure to organic carbon, elemental carbon, benzene, diesel, biomass burning, ammonium nitrate $PM_{2.5}$, & 30% per inter-quartile rise in PAHs. | Wilhelm *et al.* (2011) [20] |
| 7 | Elevated exposure to traffic pollution in pregnancy | Higher traffic pollution in pregnancy linked with pregnancy-induced hyper-tension. A 10 μg/$m^3$ elevation of $NO_x$ at home resulted an OR of 1.17. The $2^{nd}$ to $4^{th}$ quartile of $NO_x$ exposure related with SGA birth, and a risk of spontaneous preterm delivery with higher levels of $NO_x$. | Olsson *et al*. (2015) [21] |
| 8 | Pregnancy outcome & fine particulate matter | A significant link amid 10-μg/$m^3$ elevation in $PM_{2.5}$ with SGA birth & reduced birth wt. | Stieb *et al.* (2016) [22] |
| 9 | Maternal residential exposure to TRAP ($NO_2$) in pregnancy. | Examined relations with SGA birth & fetal growth restriction as proportion of optimal birth wt. (POBW) below $10^{th}$ percentile & TRAP exposure in mid to late pregnancy linked to SGA & low POBW. | Pereira *et al.* (2012) [23] |
| 10 | Maternal exposure to TRAP & urban activities | Enhance $PM_{2.5}$ levels and $PM_{2.5}$ absorbance related with lower term birth wt. | Slama *et al.* (2007) [24] |
| 11 | Infants exposed to TRAP in early life | Rapid postnatal weight gain in addition to reduced fetal growth. | Fleisch *et. al.* (2015) [25] |
| 12 | Traffic related prenatal pollution & birth wt. | Linked with LBW, most obvious with males born to obese mothers. | Lakshmanan *et al*. (2015) [26] |
| 13 | Higher traffic-related pollution exposure | Linked with higher cord blood leptin level & high molecular weight adiponectin. These adipokines related with more female infant wt. changes, which may have obesity risk. | Alderete *et al.* (2018) [27] |
| 14 | Maternal exposure to TRAP by home proximity to roadways | Women residing within 250 m of roadway were at 3-5% more odds of LBW, PTB, & late PTB equated to women residing beyond 250 m. | Miranda *et al*. (2013) [28] |
| 15 | Maternal exposure to traffic-linked air pollution | PTB was significantly more in mothers lived within 500 m of freeway than living 500-1,500 m away. | Yang *et al.* (2003) [29] |
| 16 | Residential exposure to vehicular traffic | Living within 50 m from a road with annual average daily traffic of 15,200 or more significantly linked with SAb. | Green *et al.* (2009) [30] |

| | | | |
|---|---|---|---|
| 17 | Links of stillbirth with placental abruption, placental insufficiency & exposure to TRAP. | Mothers living within 50 m of a road had 60% higher odds of stillbirth related to placental abruption equated to mothers living more than 200 m away. | Butler *et al.* (2021) [31] |
| 18 | Traffic distance & number of roads around home. | More freeways & main roads around home related to short gestation. No impacts on birth wt. length, head circumference. | Barnett *et al.* (2011) [32] |
| 19 | Air pollution from road traffic | Negatively affect fetal growth. Little evidence for traffic related noise impact on birth wt. | Smith *et al.* (2017) [33] |
| 20 | Residential proximity to roadway, & fetal growth, placental DNA methylation | Residence close to roadways related to lower fetal growth & placental epigenetic alterations. But epigenetic changes appear to be deficient to explain noted relation amid roadway proximity & fetal growth. | Kingsley *et al.* (2016) [34] |
| 21 | TRAP & pregnancy loss | Related with pregnancy loss; sturdiest effects noted during $10^{th}$ & $20^{th}$ gestational wks. | Kioumourtzog - lou *et al.* (2019) [35] |
| 22 | Residential traffic related exposure & pregnancy outcomes | Mothers exposed to traffic related exposure had no higher risk of birth outcomes or pregnancy complications. | van den Hooven *et al.* (2009) [36] |
| 23 | TRAP on birth wt. term low birth wt. & SGA | No clear relations amid birth-weight-related outcomes & traffic-linked pollution. | Kashima *et al* (2011) [37] |
| 24 | Maternal exposure to TRAP in pregnancy & PTB, fetal growth | No evidence for a hostile effect of maternal exposure to TRAP on pregnancy & outcome i.e., PTB, SGA, term birth wt. | Gehring *et al.* (2011) [38] |
| 25 | Role of high socio-economic position (SEP) & higher vehicular traffic with respect to LBW | Mothers with good SEP living in high vehicular area might not be exposed to higher air pollution. The protection against LBW occurring from a better SEP was stronger than effect of air pollution. | Habermann & Gouveia (2014) [39] |
| 26 | Hydrocarbons i.e. benzene, toluene, ethylbenzene, m/p-xylene, & o-xylene exposure from early pregnancy | Negatively related to growth in biparietal diameter (BPD). Women those spent <2 hr/ day outdoor, stronger effect amid $NO_2$ & head circumference during 12-20 wks & abdominal circumference, BPD, & fetal wt. in 20-32 wks. Concluded that TRAPs exposure in early pregnancy may affect fetal development. | Aguilera *et al.* (2010) [40] |
| 27 | Residential proximity to major roadways & IVF outcome | Lower probability of implantation & live birth after IVF. Live birth of women living ≥400 m from roadway was 46% compared to 33% living in <50 m. | Gaskins *et al.* (2018) [41] |

| | | | |
|---|---|---|---|
| 28 | $PM_{2.5}$ exposure, traffic intensity, distance to major roadway and fertility-assisted births | Not found strong significant adverse link between traffic-connected air pollution exposure and fertility-assisted births. Studies in highly exposed study areas are needed to draw inferences. | Thampy and Vieira (2023) [42] |
| 29 | Traffic metrics & pubertal onset (stage 2 or higher for breast or pubic hair development) [B2+ and PH2+]. | Puberty onset 10.3 yrs for B2+ & 10.9 yrs for PH2+. Girls in highest quintile of either distance-weighted traffic density, annual mean daily traffic, or traffic density reached PH2+ earlier than girls with lowest quintile. | McGuinn *et al.* (2016) [43] |
| 30 | Association amid TAPE & fertility rate (live births/1000 women) | A significant reduction of fertility rates with an elevation in traffic related air pollution, mainly coarse fraction of PM. | Nieuwenhuijsen *et al.* (2014) [44] |
| 31 | Fecundability ratios & residence from road | Fecundability ratios of women lived <50 m equated to ≥400 m from road were 0.88. | Wesselink *et al.* (2020) [45] |
| 32 | Prenatal exposure to traffic & ambient particulate matter with neonatal anthropo-metric measurements (NAPM). | High exposure to $PM_{2.5}$ & $PM_{10}$ linked with lower birth wt. (BW) & birth length (BL). Similar outcomes for street length in a 100 m around maternal home with BW & BL. More distance to traffic linked with higher BW & BL. More distance of residence to traffic, ring roads, bus terminal, linked with more head circumference. | Moghaddam Hosseini *et al*. (2022) [46] |
| 33 | Reviewed data on traffic related metrics & birth outcome | The odds ratio for SGA birth related with per 500 m decline in the distance to roads was 1.01. Analysis showed a significant link amid traffic density and term LBW babies. | Wang *et al*. (2020) [47] |
| 34 | Higher exposure to TRAP in pregnancy | Related with enhanced risk of pre-eclampsia. Impact was highest with higher exposures in third trimester and among younger & older women, aboriginal women, and diabetic women. | Pereira *et al.* (2013) [48] |
| 35 | Miscarriages in women with vehicle traffic air pollution. | The miscarriage prevalence was 15.83% in the higher exposure zone and 6.11% in the lower exposure region. | Silva *et al.* (2022) [49] |

**FR-**Fecundability ratios; **IVF-**In Vitro Fertilization; **m-**meter; **SGA-**Small for Gestation Age; **TRAP-**Traffic-related air pollution; **Wt-**Weight; **PM-**Particulate matter.

**Table 3** Impact of professional motor driving variables on reproductive health.

| Sl. No | Exposure | Effects | Reference |
|---|---|---|---|
| 1 | Prolonged urban driving | A risk features for hostile sperm quality, mainly sperm morphology, but further confirmation is needed. | Figà-Talamanca |

| | | | |
|---|---|---|---|
| | | | *et al.*(1996) [12] |
| 2 | Duration of sitting while working | Positively related to daytime higher scrotal temperatures, which was negatively relates with semen quality. But presumed negative correlation amid sitting duration with semen quality was not shown. However, fertility parameters of drivers with longer periods of sitting were impaired, mostly of vans, trucks, industrial machinery drivers. | Jung and Schuppe (2007) [50] |
| 3 | Relationship amid male' work-related heat exposure & time to pregnancy (TTP) | TTP for the subcategories 'exposure to heat' & 'professional driving >3 h/day' was substantially more than controls. | Thonneau *et al.* (1997) [51] |
| 4 | Impact of long-duration of driving on spermiogenesis | Pathospermia is substantially more in drivers equated to other professionals. Impairment of spermiogenesis was lesser in car drivers but more in agricultural-industrial hard machinery drivers. | Sas and Szollosi (1979) [52] |
| 5 | Reviewed data on long-term whole-body vibration (wbv) & female reproduction. | A long-term wbv can possibly impact pathogenesis of female reproductive health (menstrual disturbances, etc.) & pregnancy (abortions, stillbirths) disorders. | Seidel (1993) [53] |
| 6 | Reviewed data on the effects of Wbv on the driver's health | A few reports existed on the hostile effects of wbv on female reproduction, fetal health, and even lesser reports on male reproduction. | Joubert (2009) [54] |
| 7 | Pregnancy problems in higher and lower wbv exposure groups | More risk of pregnancy complications in highest wbv group, compared with lowest wbv group. The adjusted ORs were 1.76, 1.55 & 1.62 for pre-eclampsia, gestational hypertension, gestational diabetes, respectively. | Skröder *et al.* (2020) [55] |
| 8 | Examined the effect of wbv exposure on sperm quality. | A statistically significant difference in total sperm count, progressive, non-progressive, total motility was observed among the taxi drivers and the office employees. Difficult to draw firm conclusions about the effects of wbv exposure on semen quality as several confounding factors are also involved. | Zarei *et al*. (2022) [56] |
| 9 | Male-mediated outcome in wives of males exposed to traffic pollutants | There is a significant elevation in neonatal deaths & abortions in the spouses of traffic police & a decline in live birth. | Sree Devi *et al.* (2006) [57] |

**TTP-**Time to pregnancy; **Wbv-**Whole body vibration.

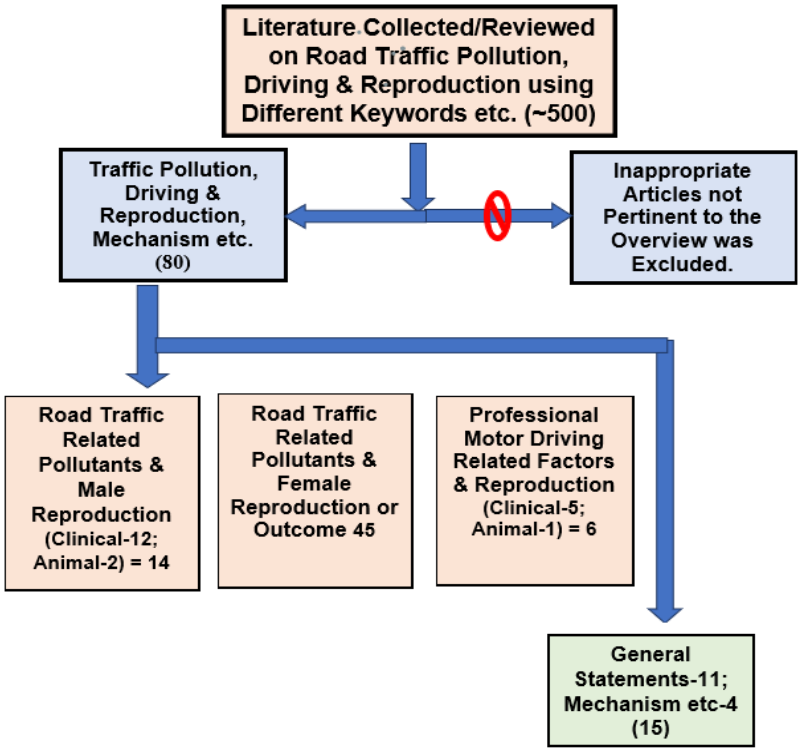


**Figure 1** Flow diagram of literature collection and presentation on road traffic pollution, motor driving occupation, and reproductive health.

## 3. Results and Discussion

People working or residing in traffic-congested areas or near key highways/ motorways are chronically exposed to travel-connected organic and inorganic pollutants. Traffic congestion increases the toxins in the surrounding environment. It degrades the adjoining air quality, and data showed higher morbidity and mortality of drivers, travelers, and persons residing close to roadways. Human health risks linked with traffic congestion must be examined regarding travel duration, rush-hour duration, congestion-specific emissions from automobile vehicles, etc. [58]. A higher manifestation of central nervous system ailments (headaches, irritability, anxiety, unusual tiredness), irritation of mucous membranes (nasal congestion, eye irritation, dry throat), and musculoskeletal complications (back and joint pains) were noted among highway toll booth workers. These signs are contemplative of the severe irritant and central nervous system effects of exposure to motor vehicle exhaust, and the musculoskeletal complaints are likely due to the cause of bending, reaching, and leaning outside of the toll booth cubicle regularly during working [59].

Air pollutants emitted from automobile vehicles are higher at locations closer to major roads/ highways/congested areas, etc. People who are living, working, or attending school adjacent to significant roads seem to have elevated health problems like asthma, cardiovascular illness, impaired lung growth among children, pre-term and low-birth-weight babies, premature death, and childhood leukemia (US, EPA) [60]. It is rational to consider that road traffic pollution might have a hostile influence on reproduction because motor vehicle driving is associated with the emission of several toxins, and some of them are reported to have reproductive toxic potential.

### *3.1 Road Traffic Associated Pollutants and Male Reproduction*

A few published reports are available on road traffic-associated toxins exposure on male reproduction, especially on semen quality, reproductive hormone levels, etc. (Table 1). However, data on erectile dysfunction, libido, etc., on human male reproduction with reverence to TRAP exposure are not available or scanty. A recent study assessed the impact of gasoline vehicle exhaust (VE) on the function of penile erectile in rat models. The results indicated that VE exposure induces a significant decline in erectile function, as assessed by the measurement of intra-cavernous pressure. They observed notable elevations of serum inflammatory factors, reduction in total lung capability and chord compliance, thickened alveoli septum, ruined alveoli, induced pulmonary fibrosis, and down-regulation of the messenger RNA and protein expression of endothelial and neuronal nitric oxide synthase in the penile tissue of VE exposed rats compared to control [61]. Another study investigated the impact of fine particulate matter ($PM_{2.5}$) on the erectile function of rats and noted that penile erection was impaired by fine particulate matter [62]. However, clinical data with regards to the VE exposure on libido or erectile function, etc., are not available or scanty, and even data on animal models are insufficient. Tallon *et al.* explored an association between ED and air pollution in older men. They found an association between $PM_{2.5}$, $NO_2$, and $O_3$ exposures and odds of developing ED, which was statistically insignificant, even though exposures to each pollutant were consistently connected with higher odds of developing ED and more data is needed on this aspect [63].

Reports are available on the influence of traffic-related pollutants on the health of Tollgates workers, including a few reports on the reproductive health of tollgate employees. Tollgate workers are exposed to traffic automobile-linked toxins while working at tollgate. The exposure intensity to traffic-related toxins depends upon the number of vehicles passed through and the duration of the tollgate workers' working hours. A study examined the semen characteristic of tollgates workers compared to age-matched control and found that sperm count, serum luteinizing hormone (LH), follicle-stimulating hormone (FSH), and testosterone concentrations were in normal range in both groups. Meanwhile, sperm motility, forward progression, functional tests, and kinetics were considerably lower in tollgate workers. Among tollgate personnel with below normal sperm motility, methemoglobin was inversely related to viability, motility, hypo-osmotic swelling, the acridine orange, and the cervical mucus penetration evaluations tests, etc. and blood Pb level were negatively linked with sperm viability and count. They concluded that blood methemoglobin and Pb levels were adversely linked with sperm quality, which indicates that nitrogen oxide (NO) and Pb might affect semen quality [6]. Another study investigated the impacts of traffic contaminants, mainly diesel exhaust, on the semen quality of toll workers and office employees (control). The abnormal sperm number and motility significantly differed among the two groups. Similarly, the number of normal morphological sperms was substantially lower in the exposed group [7].

Furthermore, the impacts of traffic contaminants on sperm chromatin/DNA integrity in tollgates and unexposed workers were evaluated. Serum concentrations of FSH, LH, and testosterone were in the normal range in both control and tollgate workers. Sperm concentration, count, total and progressive motility, and standard sperms were substantially lower in tollgate workers. Tollgate employees had a considerably higher fraction of sperm with deteriorated chromatin and DNA disintegration. This showed that exposure to car exhaust has a genotoxic impact on spermatozoa and might potentially have a hostile effect on offspring health [8]. Furthermore, a study examined

exposure to Pb and semen characteristics of traffic police personnel of Peru, where leaded petrol was in use, and the mean PbB level was seen to be 48.5 µg/dL. The current PbB level was associated with the deterioration of semen quality parameters (sperm morphology, concentration, and total count), while sperm viability and motility varied significantly amid the < or =40 µg/dL and >40 µg/dL PbB categories [9]. Furthermore, Nabi *et al.* examined the effects of gasoline emission, diesel exhaust, particulate matter, heat, and noise on the reproductive well-being of auto-rickshaw drivers. They observed a noteworthy reduction in serum testosterone, elevation in cortisol level, and more reproductive health complications (declined libido, erectile dysfunction, ejaculatory snags, infertility) in rickshaw drivers than in control [10]. An association of residential closeness to vital roads, as a proxy for TRAP exposures, with reverence to sperm characteristics and reproductive hormones, was also examined. It was found that it was not related to the deterioration of sperm characteristics, serum reproductive hormone levels among men attending the clinic [11]. However, additional data are needed with reference to the distance of residence from the motorway and the semen quality of inhabitants.

A few reports on professional motor driving and reproductive health are accessible. A study showed that taxi drivers equated to the controls, had a substantially lower occurrence of standard forms of sperms (45.8% vs. 64.0%). But other sperm parameters did not vary considerably among the two groups. Among the lifestyle factors, smoking was related to inferior sperm morphology. Subjects with low semen quality were more frequently exhibited to be associated with a longer time to pregnancy (TTP) [12]. Furthermore, the abnormal semen characteristics in infertile automobile drivers were significantly more compared to infertile non-drivers and ordinary men. Moreover, semen anomalies were considerably more common among drivers with more than 8 years of driving experience than <8 years of experience. They inferred that the driving profession can produce abnormal semen quality [13]. In addition, the data on the impacts of automobile exhaust exposure on male reproductive function and allied proteins were reviewed and stated that VE might cause hostile impacts on male reproduction by affecting organs' weight, lowering sperm quality, and causing oxidative stress. Notably, motor VE exposure causes noteworthy alterations in the patterns of protein expression, which are the main components of spermatogenesis and synthesis of testosterone [14].

An earlier study assessed the effect of working posture, nitrogen oxide exposure on semen quality. Substantially inferior sperm motility was observed in workers exposed to $NO_2$ than control. Among the workers with the mandatory requirement of sitting working posture, low sperm motility was found compared to the workforce adopting free working posture. Variations in sperm characteristic were stronger when chemical and postural factors were combined. The study established a harmful effect of $NO_2$ as an indicator of traffic contaminants and suggested the probable interaction between chemical exposure and obligated sitting postural in the occurrence of hostile sperm motility [64]. Furthermore, the long duration of driving posture might raise the scrotal temperature. A study was carried out to monitor the scrotal temperature while driving a car, and a significant scrotal temperature elevation was observed after 2 hours of driving [65]. These working conditions i.e., more time to sit for driving and related heat in the pelvic region, might be linked with deterioration of semen quality. Thus, a few minutes of rest in between driving might be beneficial to combat these factors. Moreover, De Fleurian *et al.* investigated the relationship between self-reported physical or chemical work-related exposures and semen quality and reported that physical factors, such as mechanical vibrations, were associated with oligospermia and terato-

spermia and excess heat and extended periods of sitting duration were related with impaired motility [66].

The accessible clinical data on male reproduction, especially on reproductive hormone, semen quality, and traffic-associated pollutants, on tollgate workers indicated that exposure to Traffic Associated Pollutants Exposure (TAPE) might affect sperm quality to some extent, and data on other male reproductive endpoints are scanty or not available or inconsistent. Studies on professional drivers with reverence to the profession on reproductive health are inadequate and need more data.

### *3.2 Road Traffic Linked Pollutants and Female Reproduction or Pregnancy Outcome*

The data pertaining to the effects of Traffic Associated Pollutants Exposure (TAPE) are less documented on female reproduction. No comprehensive study can be located among female workers/personnel who were directly exposed to TAPE occupationally while working on highways/ motorways/traffic-congested areas to recognize the impact of TAPE on menstruation cycle, menarche age, hormonal changes, ovarian function, the occurrence of menopause, etc. However, reasonable clinical data are accessible on the influence of TAPE on pregnancy and its outcome, especially in females residing in traffic/roadways congested areas or inhabiting adjacent to key traffic areas. Earlier, an association between TAPE and humans' fertility rates was studied, and a significant reduction in fertility rates was noted with an elevation of TRAP concentrations, especially for the coarse portion of particulate matter [44].

Furthermore, a study investigated perturbations of the maternal serum metabolome with respect to traffic-related air pollution exposure during pregnancy. The metabolomic characteristics and pathways affected by the TRAP exposure suggested that maternal exposure to TRAP in pregnancy causes oxidative stress and affects inflammation pathways, which are implicated in pregnancy complications and hostile outcomes [67]. Further, a study assessed relationships amid prenatal ambient air pollution, traffic-related exposure, and baby weight and adiposity. A limited sign of association was found amid prenatal traffic or ambient air contamination exposure with baby body configuration. While, a suggestive relationship was noted between prenatal exposure to ozone and initial postnatal variations in body configuration, which is worth further investigation [68]. The impact of air contamination on the span of the menstrual cycle and its stages (follicular and luteal) was evaluated. Municipal data was utilized to assess air pollution exposure during the menstrual cycle, and critical factor analyzed were group pollutants ($PM_{10}$, $SO_2$, CO, and $NO_x$). They stated that CO and $NO_x$ analyzed either separately or combined as a traffic emission were not connected with alteration of menstrual cycle length or cycle phases. Luteal phase lessening might be resulted from exposure to fossil fuel combustion [69]. Further, a study analyzed traffic-related metrics data and pubertal commencement (defined as stage 2 or above for breast or pubic hair growth) [B2+ and PH2+]. The commencement of median age was 10.3 years (yrs) for B2+ and 10.9 yrs for PH2+. Girls in the maximum quintile of either distance-weighted traffic intensity, a yearly average of daily traffic, and/or traffic intensity attained PH2+ earlier than girls of the lowermost quintile. They concluded that girls with higher exposure touched one pubertal landmark a few months prior than those with lower exposer [43].

A few reports exist on TRAPs exposure and preterm birth (PTB). The risk of PTB from prenatal TRAP exposure and household environmental factors in pregnancy indicated that both traffic-linked air contaminant and indoor mold/humid environments play a crucial role in the occurrence of PTB,

and interaction of these during early pregnancy elevates the risk of PTB considerably [15]. An elevated odds of early PTB were also observed in women who were exposed to the highest quartile of each TRAP contaminant from the 2nd trimester of pregnancy to the culmination of pregnancy. Associations were much more robust in mothers inhabiting low socioeconomic status zones [16]. Moreover, a study scrutinized the impacts of exposure to local traffic air contamination on pre-eclampsia, PTB, and a higher risk of pre-eclampsia, PTB, was observed from maternal exposure to traffic-generated $NO_x$ and $PM_{2.5}$ [17]. Elevated risks for pre-eclampsia, PTB, and very PTB were also reported from maternal exposures to TRAPs as assessed at ambient stations (CO, NO, $NO_2$, and $NO_x$). Furthermore, elevated PTB risk and very PTB were positively related to $PM_{10}$ and $PM_{2.5}$ air contamination exposure estimated at ambient stations [18]. They concluded that TRAP was associated with adverse reproductive outcomes irrespective of the exposure assessment method was utilized.

Furthermore, a geospatial analysis was conducted to find out whether home during pregnancy, concerning traffic density and mobility or movement in urban or suburban area, might be a risk issue for PTB and suggested that community-related risk factors (urban/suburban location, traffic density, and traveling to work in high-vehicle density area) might be a risk factor for PTB [19]. Furthermore, a study investigated the role of traffic-related air toxin and PTB. Odds of PTB was augmented to 6-21% per inter-quartile range elevation of TRAP during whole pregnancy exposure to organic carbon, elemental carbon, benzene, and diesel, biomass burning, and ammonium nitrate $PM_{2.5}$, and 30% per inter-quartile elevation in PAHs. The analyses provided additional evidence of TRAPs exposure and risk of PTB and stated that PAHs is a pollutant of concern and should be focus for future studies [20].

An association between elevated traffic-connected pollution exposure in pregnancy and pregnancy-related hypertensive disorders was reported, and a 10 μg/m$^3$ elevation in mean level of NOx in pregnancy at home resulted in an OR of 1.17. The 2nd to 4th quartile of NOx exposure were related with an elevated risk for small for gestation age (SGA) birth and a tendency of a risk of spontaneous preterm delivery with respect to higher levels of NOx [21]. A Canadian study included ~3 million births and found a significant relationship between a 10-μg/m$^3$ elevation in $PM_{2.5}$ with SGA birth and reduced term birth weight, providing evidence of $PM_{2.5}$ exposure and hostile pregnancy outcomes [22]. A study assessed maternal housing exposure to a marker of TRAP ($NO_2$) during pregnancy and birth outcome. Logistic regression was used to examine the association for SGA birth and fetal growth restriction as a proportion of optimal birth weight (POBW) lower than the 10th percentile level and stated that exposure to TRAP during mid to late pregnancy was related to an SGA birth and low POBW babies [23]. Furthermore, elevated exposure to TRAP in pregnancy was linked with an enhanced risk of pre-eclampsia. The impact dimensions were highest with higher exposures to TRAP in the third trimester of pregnancy and among younger and older women, aboriginal women, and diabetic women [48].

Traffic-related pollution exposure may also be associated with the birth weight of the offspring. The impact of maternal exposure to air contaminants owing to road traffic and metropolitan activities on term birth weight indicated that elevation in $PM_{2.5}$ concentrations and absorbance were related to the deterioration of the offspring's term birth weight [24]. Moreover, a study revealed that infants exposed to higher traffic-related contamination in early life showed speedy postnatal weight gain and reduced fetal growth [25]. Furthermore, a study examined links between prenatal exposure to air pollution and birth weight with reverence to offspring sex, maternal BMI

and stated a relationship amid prenatal exposure to TRAP and shortened birth weight, which were apparent in males of obese mothers [26]. Also, a higher TRAP exposure was related with an elevation of cord blood leptin level and a higher molecular weight of adiponectin. These adipokines were linked with higher infant weight changes in female babies, which might have consequences of future obesity risk [27]. Recently, prenatal exposure to traffic and ambient particulate matter indicators on neonatal anthropo-metric measurements (NAPM) was studied. The data exhibited that considerable exposure to $PM_{2.5}$ and $PM_{10}$ was considerably connected with reduced birth weight (BW) and birth length (BL). Similar outcomes were noted for total street distance in a 100 m zone across a maternal home for BW and BL. More distance to heavy traffic was substantially connected with higher BW and BL. The elevation in the distance from residence to heavy traffic, ring roads, bus terminals, and transportation was related to higher head circumference [46]. Furthermore, Wang *et al*. reviewed data on traffic-related metrics on birth outcomes. The odds ratio for the change in SGA birth was related to the per 500 m decline in the distance to roads was 1.01, and further analyses showed a significant positive relationship amid traffic density and term low birth weight babies [47].

Recently, Zhao *et al*. examined the impact of prenatal fine particulate matter ($PM_{2.5}$) exposure on fetal growth and the underlying placental epigenetic mechanism in Chinese women. Increased $PM_{2.5}$ exposure was significantly linked with a reduced biparietal diameter (BPD), head circumference (HC), femur length (FL), and abdominal circumference in the 2$^{nd}$ trimester and birth length. Prenatal $PM_{2.5}$ exposure may also cause undesirable impacts on fetal growth by altering placental DNA methylation [70]. Furthermore, first trimester and entire pregnancy $PM_{2.5}$ levels were significantly related to the risk of elevated Intrauterine Fetal Death (IUFD) risk among Jewish women. In a multi-variable model, for every 10 $\mu g/m^3$ unit increase in $PM_{2.5}$ exposure, the risk for IUFD increases by 2.98 and the 3.61 during the first trimester and the entire pregnancy, respectively [71]. Wang *et al.* stated that $PM_{2.5}$ exposure can break the respiratory mucosal barrier and enter the human body, causing pathological effects on various body' systems, and $PM_{2.5}$ can accumulate in the reproductive organs through blood-testis, placental, epithelial, and other barriers. $PM_{2.5}$ exposure can disrupt hormone levels, ultimately affecting fertility. Also, oxidative stress, inflammation, apoptosis, and the breakdown of barrier structures contribute to reproductive toxicity [72].

It is rational to consider that closeness to significant roadways/traffic captures a variety of traffic-linked air pollutants in addition to allied pollution such as traffic' noise, work posture, long periods of sitting while driving etc., and drivers are subjected to chronic exposure to these pollutants/factors. An earlier study categorized maternal TRAP exposure in pregnancy using housing proximity to vital roadways. It noted that women inhabiting within 250 meters of a key road were at 3-5% elevated odds of low birth weight (LBW), PTB, and late PTB, which was significant compared to women inhabiting beyond 250 meters of roadways. They stated that road proximity represents a straight forward approach to maternal risk estimation related to traffic-related pollution [28]. PTB babies were substantially higher in mothers residing within 500 meters of the freeway than mothers living over 500-1,500 meters from the highway [29]. Furthermore, a study determined the impact of housing exposure related to vehicular traffic to spontaneous abortion (SAb). It stated that residing within 50 meters of the road with annual mean daily traffic of 15,200 or more vehicles was significantly related to the occurrence of SAb among African Americans and nonsmokers [30].

A case-control study was carried out to assess the relationship between stillbirth and placental abruption, placental insufficiency, or maternal exposure to TRAP. No overall meaningful rise in the odds of placental-associated stillbirths was observed. However, mothers residing within 50 meters of a road had 60% elevated odds of stillbirth related to placental abruption, equated to mothers living more than 200 meters away from roadways. This indicative finding might be imprecise owing to the lower number of subjects in the uppermost exposure group, and studies on placental abruption with more precise exposure assessments are required [31]. An earlier study examined the two measures of traffic: 1) residential distance to adjacent road and 2) number of roads around the home. More freeways and key roads closer to the home were related to a shorter gestation period, while no adverse effects were observed on birth length, weight, and head circumference. Furthermore, the adverse impacts on gestation were noted mainly due to vital roads within 400 meters of the residence. For each of the 10 additional extra vital roads within 400 meters of the residence, gestation time declined by 1.1%. They concluded that the observed effect due to the chemical toxins in traffic contaminants or disturbed sleep owed to traffic noise [32]. Furthermore, a study advised that air contamination from road transport in London was related to a negative impact on fetal growth. However, little indication of an independent exposure-effect relationship between traffic-linked noise contamination and birth weight was observed [33]. An association between residential closeness to main roadways and fetal development, placental DNA methylation was studied, and it was stated that residing adjacent to key roadways is linked to lower fetal development and a significant placental epigenetic alteration. However, the noted epigenetic variations appear to be inadequate to explain the observed relationship amid closeness to roadways and fetal growth [34].

Furthermore, a report indicated that air pollution is hazardous to pregnant women and newborns, and several of them are exposed to higher doses of traffic-related pollutants in urban areas. Such exposure can cause the occurrence of LBW babies and hostile effects like respiratory ailments and premature death [73]. Another study analyzed the association between air pollution exposure and pregnancy loss during different gestation periods. The data obtained, with striking similarity at two independent locations, suggest that higher traffic-connected air pollution is related to more risk of pregnancy loss, with the highest risk between the $10^{th}$ to $20^{th}$ weeks of gestation [35].

A few studies reported no significant impact of TAPE on birth outcome parameters. In a study, an association between housing proximity to traffic and pregnancy outcomes was investigated, and it was observed that mothers exposed to the proximity of housing to traffic had no more risk of hostile birth outcomes or pregnancy complications [36]. A study evaluated the relationship of air pollution with birth weight, term low birth weight, and SGA birth. They did not observe a clear association between birth-weight-related outcomes and the three markers of traffic-linked air pollution [37]. In addition, the effect of maternal TRAP exposure during different periods of pregnancy on PTB, fetal growth was evaluated, and there was no sign of a harmful effect of maternal TRAP exposure during pregnancy on PTB, SGA birth, and term birth weight [38]. Furthermore, the impact of higher socioeconomic position and more vehicular traffic concerning LBW was studied, and it was stated that mothers with privileged socioeconomic position (SEP), even though they inhabit in higher traffic areas, might not be exposed to higher air pollution. It can be noted that the defense against LBW is occurring from a superior SEP, which was stronger than the consequence of air pollution, and the pollution exposure level may not be adequate to elevate the hazard of LBW babies [39]. Exposure to benzene, ethylbenzene, toluene, m/p-xylene, and o-xylene (BTEX) from

early pregnancy was adversely related with growth in biparietal diameter (BPD). The air contamination impacted no other fetal developmental parameters during pregnancy. When looking at women who spent <2 hr/day at nonresidential sites, the impact was more vital for an association amid $NO_2$ and head circumference during 12-20 weeks and growth in abdominal circumference, BPD, and estimated fetal weight during 20-32 weeks of gestation. They concluded that data support the impact of TRAPs exposure in early pregnancy on fetal development during mid-pregnancy [40].

A recent report indicated that fecundability ratios (FR) of women residing <50 meters equated to those living ≥400 meters away from the major road were 0.88 in United States. The relationship in Canadian women was also similar but less precise (FR = 0.93). Likewise, distance of main roads inside buffers of 50 and 100 meters was related to lesser fecundability in both countries. They concluded that TRAP or other adjacent-road exposures might negatively affect fecundability [45]. Furthermore, studies on exposure to TRAPs advise a risk for undesirable birth outcomes. Still, additional studies are required to estimate the exposure more precisely in terms of both pollution components and timing of exposure [74]. Very recently, a cross-sectional study was conducted to evaluate the occurrence of miscarriages in women belonging to high and low vehicular traffic air pollution. The miscarriage prevalence was 15.83% in the higher vehicle traffic air pollution exposure zone, while 6.11% was in the low pollution exposure region [49].

In addition, exposure to TAPE might affect the assisted reproductive technique (ART) related to pregnancy or its outcome, but the data remains scanty on this issue. An article reported that residence close to key roadways was significantly connected with a lower likelihood of occurrence of implantation and live birth after IVF treatment. The adjusted % of IVF cycles ensuing to live birth for women residing ≥400 meters away from a main roadway was 46%, equating to 33% for women residing <50 meters from roadways. A suggestive relationship existed between living near main roadways, marginally higher estradiol concentrations, and reduced endometrial thickness. They concluded that lower residential nearness to main roads was linked to reduced manifestation of live births following IVF treatment [41]. Additional studies are required on this issue. Later, they assessed whether folate consumption altered the relationship between TRAP exposure and live births among women undergoing ART procedures. They also stated that supplementary folate consumption significantly changed the relationship between exposure to $NO_2$ and live birth. Higher folate consumption might defend against the hostile reproductive outcome of traffic-related air pollution [75]. A recent study assessed the relationship between TRAP exposure and fertility-assisted childbirth. They investigated $PM_{2.5}$ exposure, traffic intensity, and distance to a major roadway and fertility-assisted births. They found no significant solid adverse association between traffic-connected air pollution exposure and fertility-assisted births. Further studies in more highly exposed study areas are needed to draw inferences [42].

### *3.3 Impact of Professional Motor Driving Variables on Reproductive Health*

A longer duration of sitting while driving for a long distance might be related to the generation of excess heat in the pelvic region, which may have a hostile impact on reproductive health. However, data on this issue of human reproduction are not available or are very scanty. An earlier study mentioned that prolonged urban driving might be a risk feature for hostile sperm quality, particularly sperm morphology, but the finding requires further confirmation [12]. Furthermore, the duration of sitting during work was reported to be positively related to daytime higher scrotal

temperatures, which was negatively associated with semen characteristics. However, the presumed negative correlation between sitting duration and semen quality was not shown in the accessible studies. Nevertheless, they mentioned that the fertility parameters of drivers with more extended periods of sitting, mostly vans, trucks, and industrial heavy machinery drivers, were predominantly impaired [50] . Also, a relationship between male work-related heat exposure and time to pregnancy (TTP) was investigated. The TTP for the subcategories 'exposure to heat' and 'professional driver >3 h/day' was substantially more than controls [51]. Furthermore, a report indicated that scrotal temperature was significantly higher after two hours of driving posture than scrotal temperature while walking [65]. These data suggest that longer sitting time might be accountable for excessive heat generation, which may affect human reproduction, but the data is still needed to substantiate the reported observations. The impact of long-duration driving on spermiogenesis was examined. Pathospermia was substantially more frequent among drivers than among other professionals, which was duration dependent. The impairment of spermiogenesis was lesser in car drivers but more in agricultural-industrial hard machinery-farm equipment drivers [52].

In addition, drivers are subjected to whole-body vibrations (wbv) while driving. An earlier review reported that long-term wbv exposure could probably contribute to the pathogenesis of female reproductive disorders (menstrual disturbances, etc.) and pregnancy disorders (abortions, stillbirths) [53]. Later, a review on the effects of wbv on the health of drivers stated that only a few reports existed on the hostile impacts of wbv on female reproduction and fetal health, and fewer reports existed on male reproduction [54]. Recently, Skröder *et al.* reported an elevated risk of pregnancy complications in the highest wbv exposure group, compared with the lowest wbv exposure group. The adjusted ORs were found to be 1.76, 1.55, and 1.62 for pre-eclampsia, gestational hypertension, and gestational diabetes, respectively [55]. Furthermore, a study examined the effect of wbv exposure on sperm quality. A statistically significant difference in total sperm count, progressive, non-progressive, and total motility was observed among the taxi drivers and the office employees. According to the uni-variate analysis of variance, exposure to wbv had a hostile effect on sperm parameters. It is difficult to draw firm conclusions about the effects of wbv exposure on semen quality as several confounding factors are also involved [56]. A recent study examined the impact of wbv on reproductive health in a rat model. WBV exposure caused a significant reduction in developing follicles and a decline in circulating estradiol levels, ovarian luteinizing hormone receptor protein levels, and marked changes in transcript levels, cell cycle, and steroidogenesis. In males, wbv resulted in a significant decrease in spermatids and circulating prolactin levels, elevation in testosterone levels, and marked changes in levels of transcripts related to oxidative stress, inflammation, and factors involved in regulating the cell cycle [76].

Telomere length might be considered a molecular indicator of spermatogenesis and sperm quality linked with male fertility potential. A few promising evidence are also accessible for oocyte, female fertility, and embryo quality. Telomere length might have a prognostic potential in the future for couple infertility, mainly to choose the best germ cells with the highest fertilization potential [77]. Furthermore, short telomere length results in meiotic arrest, segregation anomalies, and disjunction, which causes an elevation of aneuploid germ cells. In addition, reduced telomere length in men results in apoptosis of germ cells, while in women, it results in meiotic arrest [78]. Further, a short telomere length in sperm might be a contributing factor for male infertility, but comprehensive studies are desirable to prove the findings [79]. Furthermore, telomere attrition is reported to be fast in the early years of life, whereas during maturity, lifestyle factors slightly impact

telomere attrition. Hence, early life is vital for estimating telomere length (Bijnens *et al*.) [80]. Thus, telomere length in placental tissues and buccal cells in young adulthood was examined to housing exposure to traffic-associated pollution. Maternal residential traffic-related exposure was linked inversely with telomere length at birth, and enhanced telomere reduction was observed in the initial two decades of life. Telomere status at birth and young adulthood was negatively significantly linked with residential traffic-linked exposure at the birthplace. While traffic exposure in adulthood was unrelated to telomere length [80]. It can be said from the information available that a reduction in telomere length may hurt fertility potential. Additional studies are desirable regarding shortening of telomere length by traffic-related exposure and infertility.

In addition, a report on male-mediated hostile reproductive outcomes among wives of males exposed to traffic-related pollutants is also available, which showed a significant elevation in neonatal deaths and abortions in the spouses of traffic police and a decline in live births. The results indicated that exposure to VE induces adverse reproductive consequences in the spouses of traffic police personnel [57]. However, more studies are necessary on this facet of human reproduction to substantiate the findings.

## 4. Conclusions

Based upon the accessible information on the impact of TAPE on the human reproductive health of both sexes, pregnancy, or its outcome, one can infer that exposure to traffic-associated pollution might relate to deterioration of semen characteristics, pregnancy outcome to some extent and data on other reproductive outcome/endpoints to TAPE exposure are scanty or inconsistent. However, more data on the impact of TAPE on human reproduction and pregnancy outcomes are needed to demonstrate the reported findings with additional data.

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## Competing Interests

The author has declared that no competing interests exist.

## References

1. Winkler SL, Anderson JE, Garza L, Ruona WC, Vogt R, Wallington TJ. Vehicle criteria pollutant (PM, $NO_x$, CO, HCs) emissions: How low should we go? NPJ Clim Atmos Sci. 2018; 1: 26.
2. NSW Health. TP03: Health Effects of Traffic-Related Air Pollution [Internet]. New South Wales, Australia: NSW Government; 2014. Available from:

https://www.chiefscientist.nsw.gov.au/data/assets/pdf_file/0004/52987/Road-Tunnels_TP03_Health_effects_of_traffic_related_air_pollution.pdf.

3. Khreis H. Traffic, air pollution, and health. In: Advances in transportation and health. Amsterdam, Netherlands: Elsevier; 2020. pp. 59-104. doi: 10.1016/B978-0-12-819136-1.00003-6.
4. Alemayehu YA, Asfaw SL, Terfie TA. Exposure to urban particulate matter and its association with human health risks. Environ Sci Pollut Res. 2020; 27: 27491-27506.
5. Ding D, Gebel K, Phongsavan P, Bauman AE, Merom D. Driving: A road to unhealthy lifestyles and poor health outcomes. PLoS One. 2014; 9: e94602.
6. Rosa MD, Zarrilli S, Paesano L, Carbone U, Boggia B, Petretta M, et al. Traffic pollutants affect fertility in men. Hum Reprod. 2003; 18: 1055-1061.
7. Guven A, Kayikci A, Cam K, Arbak P, Balbay O, Cam M. Alterations in semen parameters of toll collectors working at motorways: Does diesel exposure induce detrimental effects on semen? Andrologia. 2008; 40: 346-351.
8. Calogero AE, La Vignera S, Condorelli RA, Perdichizzi A, Valenti D, Asero P, et al. Environmental car exhaust pollution damages human sperm chromatin and DNA. J Endocrinol Invest. 2011; 34: e139-e143.
9. Eibensteiner L, Sanz AD, Frumkin H, Gonzales C, Gonzales GF. Lead exposure and semen quality among traffic police in Arequipa, Peru. Int J Occup Environ Health. 2005; 11: 161-166.
10. Nabi G, Amin M, Khan AA. Reproductive health in rickshaw drivers: Occupational exposure to environmental stressor. Bali Med J. 2014; 3: 78-84.
11. Nassan FL, Chavarro JE, Mínguez Alarcón L, Williams PL, Tanrikut C, Ford JB, et al. Residential distance to major roadways and semen quality, sperm DNA integrity, chromosomal disomy, and serum reproductive hormones among men attending a fertility clinic. Int J Hyg Environ Health. 2018; 221: 830-837.
12. Figà Talamanca I, Cini C, Varricchio GC, Dondero F, Gandini L, Lenzi A, et al. Effects of prolonged autovehicle driving on male reproductive function: A study among taxi drivers. Am J Ind Med. 1996; 30: 750-758.
13. Zhao RP, Yang SQ, Huang L, Chang XX, Zhang X, Xu ZS, et al. Analysis of sterile male semen of occupational drivers. Zhonghua Nan Ke Xue. 2003; 9: 515-516, 519.
14. Rengaraj D, Kwon WS, Pang MG. Effects of motor vehicle exhaust on male reproductive function and associated proteins. J Proteome Res. 2015; 14: 22-37.
15. Lu C, Cao L, Norbäck D, Li Y, Chen J, Deng Q. Combined effects of traffic air pollution and home environmental factors on preterm birth in China. Ecotoxicol Environ Saf. 2019; 184: 109639.
16. Padula AM, Mortimer KM, Tager IB, Hammond SK, Lurmann FW, Yang W, et al. Traffic-related air pollution and risk of preterm birth in the San Joaquin Valley of California. Ann Epidemiol. 2014; 24: 888-895.e4.
17. Wu J, Ren C, Delfino RJ, Chung J, Wilhelm M, Ritz B. Association between local traffic-generated air pollution and preeclampsia and preterm delivery in the south coast air basin of California. Environ Health Perspect. 2009; 117: 1773-1779.
18. Wu J, Wilhelm M, Chung J, Ritz B. Comparing exposure assessment methods for traffic-related air pollution in an adverse pregnancy outcome study. Environ Res. 2011; 111: 685-692.

19. Kahr MK, Suter MA, Ballas J, Ramphul R, Lubertino G, Hamilton WJ, et al. Preterm birth and its associations with residence and ambient vehicular traffic exposure. Am J Obstet Gynecol. 2016; 215: 111.e1-111.e10.
20. Wilhelm M, Ghosh JK, Su J, Cockburn M, Jerrett M, Ritz B. Traffic-related air toxics and preterm birth: A population-based case-control study in Los Angeles County, California. Environ Health. 2011; 10: 89.
21. Olsson D, Mogren I, Eneroth K, Forsberg B. Traffic pollution at the home address and pregnancy outcomes in Stockholm, Sweden. BMJ Open. 2015; 5: e007034.
22. Stieb DM, Chen L, Beckerman BS, Jerrett M, Crouse DL, Omariba DW, et al. Associations of pregnancy outcomes and $PM_{2.5}$ in a national Canadian study. Environ Health Perspect. 2016; 124: 243-249.
23. Pereira G, Cook AG, Haggar F, Bower C, Nassar N. Locally derived traffic-related air pollution and fetal growth restriction: A retrospective cohort study. Occup Environ Med. 2012; 69: 815-822.
24. Slama R, Morgenstern V, Cyrys J, Zutavern A, Herbarth O, Wichmann HE, et al. Traffic-related atmospheric pollutants levels during pregnancy and offspring's term birth weight: A study relying on a land-use regression exposure model. Environ Health Perspect. 2007; 115: 1283-1292.
25. Fleisch AF, Rifas Shiman SL, Koutrakis P, Schwartz JD, Kloog I, Melly S, et al. Prenatal exposure to traffic pollution: Associations with reduced fetal growth and rapid infant weight gain. Epidemiology. 2015; 26: 43-50.
26. Lakshmanan A, Chiu YH, Coull BA, Just AC, Maxwell SL, Schwartz J, et al. Associations between prenatal traffic-related air pollution exposure and birth weight: Modification by sex and maternal pre-pregnancy body mass index. Environ Res. 2015; 137: 268-277.
27. Alderete TL, Song AY, Bastain T, Habre R, Toledo Corral CM, Salam MT, et al. Prenatal traffic-related air pollution exposures, cord blood adipokines and infant weight. Pediatr Obes. 2018; 13: 348-356.
28. Miranda ML, Edwards SE, Chang HH, Auten RL. Proximity to roadways and pregnancy outcomes. J Expo Sci Environ Epidemiol. 2013; 23: 32-38.
29. Yang CY, Chang CC, Chuang HY, Ho CK, Wu TN, Tsai SS. Evidence for increased risks of preterm delivery in a population residing near a freeway in Taiwan. Arch Environ Health. 2003; 58: 649-654.
30. Green RS, Malig B, Windham GC, Fenster L, Ostro B, Swan S. Residential exposure to traffic and spontaneous abortion. Environ Health Perspect. 2009; 117: 1939-1944.
31. Butler L, Gallagher L, Winter M, Fabian MP, Wesselink A, Aschengrau A. Residential proximity to roadways and placental-associated stillbirth: A case-control study. Int J Environ Health Res. 2021; 31: 465-474.
32. Barnett AG, Plonka K, Seow WK, Wilson LA, Hansen C. Increased traffic exposure and negative birth outcomes: A prospective cohort in Australia. Environ Health. 2011; 10: 26.
33. Smith RB, Fecht D, Gulliver J, Beevers SD, Dajnak D, Blangiardo M, et al. Impact of London's road traffic air and noise pollution on birth weight: Retrospective population based cohort study. BMJ. 2017; 359: j5299.

34. Kingsley SL, Eliot MN, Whitsel EA, Huang YT, Kelsey KT, Marsit CJ, et al. Maternal residential proximity to major roadways, birth weight, and placental DNA methylation. Environ Int. 2016; 92: 43-49.
35. Kioumourtzoglou MA, Raz R, Wilson A, Fluss R, Nirel R, Broday DM, et al. Traffic-related air pollution and pregnancy loss. Epidemiology. 2019; 30: 4-10.
36. van den Hooven EH, Jaddoe VW, de Kluizenaar Y, Hofman A, Mackenbach JP, Steegers EA, et al. Residential traffic exposure and pregnancy-related outcomes: A prospective birth cohort study. Environ Health. 2009; 8: 59.
37. Kashima S, Naruse H, Yorifuji T, Ohki S, Murakoshi T, Takao S, et al. Residential proximity to heavy traffic and birth weight in Shizuoka, Japan. Environ Res. 2011; 111: 377-387.
38. Gehring U, Van Eijsden M, Dijkema MB, Van der Wal MF, Fischer P, Brunekreef B. Traffic-related air pollution and pregnancy outcomes in the Dutch ABCD birth cohort study. Occup Environ Med. 2011; 68: 36-43.
39. Habermann M, Gouveia N. Socioeconomic position and low birth weight among mothers exposed to traffic-related air pollution. PLoS One. 2014; 9: e113900.
40. Aguilera I, Garcia Esteban R, Iñiguez C, Nieuwenhuijsen MJ, Rodríguez À, Paez M, et al. Prenatal exposure to traffic-related air pollution and ultrasound measures of fetal growth in the INMA Sabadell cohort. Environ Health Perspect. 2010; 118: 705-711.
41. Gaskins AJ, Hart JE, Mínguez Alarcón L, Chavarro JE, Laden F, Coull BA, et al. Residential proximity to major roadways and traffic in relation to outcomes of in vitro fertilization. Environ Int. 2018; 115: 239-246.
42. Thampy D, Vieira VM. Association between traffic-related air pollution exposure and fertility-assisted births. Environ Res. 2023; 1: 021005.
43. McGuinn LA, Voss RW, Laurent CA, Greenspan LC, Kushi LH, Windham GC. Residential proximity to traffic and female pubertal development. Environ Int. 2016; 94: 635-641.
44. Nieuwenhuijsen MJ, Basagaña X, Dadvand P, Martinez D, Cirach M, Beelen R, et al. Air pollution and human fertility rates. Environ Int. 2014; 70: 9-14.
45. Wesselink AK, Kirwa K, Hatch EE, Hystad P, Szpiro AA, Kaufman JD, et al. Residential proximity to major roads and fecundability in a preconception cohort. Environ Epidemiol. 2020; 4: e112.
46. MoghaddamHosseini V, Dowlatabadi A, Najafi ML, Ghalenovi M, Pajohanfar NS, Ghezi S, et al. Association of traffic-related air pollution with Newborn's anthropometric indexes at birth. Environ Res. 2022; 204: 112000.
47. Wang L, Guo P, Tong H, Wang A, Chang Y, Guo X, et al. Traffic-related metrics and adverse birth outcomes: A systematic review and meta-analysis. Environ Res. 2020; 188: 109752.
48. Pereira G, Haggar F, Shand AW, Bower C, Cook A, Nassar N. Association between pre-eclampsia and locally derived traffic-related air pollution: A retrospective cohort study. J Epidemiol Community Health. 2013; 67: 147-152.
49. Silva VC, Pires RC, Silva HE, Lopes ÉM, Lira ÁH, Pestana AL, et al. Exposure to pollution during pregnancy and occurrence of miscarriage. Ambient Soc. 2022; 25. doi: 10.1590/1809-4422asoc 20200134r2vu2022L2OA.
50. Jung A, Schuppe HC. Influence of genital heat stress on semen quality in humans. Andrologia. 2007; 39: 203-215.
51. Thonneau P, Ducot B, Bujan L, Mieusset R, Spira A. Effect of male occupational heat exposure on time to pregnancy. Int J Androl. 1997; 20: 274-278.

52. Sas M, Szöllosi J. Impaired spermiogenesis as a common finding among professional drivers. Arch Androl. 1979; 3: 57-60.
53. Seidel H. Selected health risks caused by long-term, whole-body vibration. Am J Ind Med. 1993; 23: 589-604.
54. Joubert DM. Professional driving and adverse reproductive outcomes: The evidence to date and research challenges. Open Occup Health Saf J. 2010; 1: 1-6.
55. Skröder H, Pettersson H, Albin M, Gustavsson P, Rylander L, Norlén F, et al. Occupational exposure to whole-body vibrations and pregnancy complications: A nationwide cohort study in Sweden. Occup Environ Med. 2020; 77: 691-698.
56. Zarei S, Dehghan SF, Vaziri MH, Gilani MA, Ardakani SK. Assessment of semen quality of taxi drivers exposed to whole body vibration. J Occup Med Toxicol. 2022; 17: 16.
57. Sree Devi VS, Prasad MH, Rao VD, Devi GS, Reddy PP. Reproductive outcome in the wives of traffic policemen exposed to automobile exhaust. J Hum Ecol. 2006; 20: 77-82.
58. Zhang K, Batterman S. Air pollution and health risks due to vehicle traffic. Sci Total Environ. 2013; 450-451: 307-316.
59. Strauss P, Orris P, Buckley L. A health survey of toll booth workers. Am J Ind Med. 1992; 22: 379-384.
60. United States Environmental Protection Agency (EPA). Near Roadway Air Pollution and Health: Frequently Asked Questions [Internet]. Office of Transportation and Air Quality EPA-420-F-14-044. Washington, DC, US: United States Environmental Protection Agency (EPA); 2014. Available from: https://www.epa.gov/sites/default/files/2015-11/documents/420f14044 0.pdf.
61. Zhao S, Wang J, Xie Q, Luo L, Zhu Z, Liu Y, et al. Elucidating mechanisms of long-term gasoline vehicle exhaust exposure-induced erectile dysfunction in a rat model. J Sex Med. 2019; 16: 155-167.
62. Wang X, Yang Y, Li J, Bai Y, Tang Y, Han P. Effects of fine particulate matter on erectile function and its potential mechanism in rats. Urology. 2017; 102: 265.e9-265.e16.
63. Tallon LA, Manjourides J, Pun VC, Mittleman MA, Kioumourtzoglou MA, Coull B, et al. Erectile dysfunction and exposure to ambient air pollution in a nationally representative cohort of older men. Environ Health. 2017; 16: 12.
64. Boggia B, Carbone UM, Farinaro ED, Zarrilli ST, Lombardi G, Colao A, et al. Effects of working posture and exposure to traffic pollutants on sperm quality. J Endocrinol Invest. 2009; 32: 430-434.
65. Bujan L, Daudin M, Charlet JP, Thonneau P, Mieusset R. Increase in scrotal temperature in car drivers. Hum Reprod. 2000; 15: 1355-1357.
66. De Fleurian G, Perrin J, Ecochard R, Dantony E, Lanteaume A, Achard V, et al. Occupational exposures obtained by questionnaire in clinical practice and their association with semen quality. J Androl. 2009; 30: 566-579.
67. Yan Q, Liew Z, Uppal K, Cui X, Ling C, Heck JE, et al. Maternal serum metabolome and traffic-related air pollution exposure in pregnancy. Environ Int. 2019; 130: 104872.
68. Starling AP, Moore BF, Thomas DS, Peel JL, Zhang W, Adgate JL, et al. Prenatal exposure to traffic and ambient air pollution and infant weight and adiposity: The Healthy Start study. Environ Res. 2020; 182: 109130.

69. Merklinger Gruchala A, Jasienska G, Kapiszewska M. Effect of air pollution on menstrual cycle length-a prognostic factor of women's reproductive health. Int J Environ Res Public Health. 2017; 14: 816.
70. Zhao Y, Wang P, Zhou Y, Xia B, Zhu Q, Ge W, et al. Prenatal fine particulate matter exposure, placental DNA methylation changes, and fetal growth. Environ Int. 2021; 147: 106313.
71. Wainstock T, Yoles I, Sergienko R, Kloog I, Sheiner E. Prenatal particulate matter exposure and intrauterine fetal death. Int J Hyg Environ Health. 2021; 234: 113720.
72. Wang L, Luo D, Liu X, Zhu J, Wang F, Li B, et al. Effects of $PM_{2.5}$ exposure on reproductive system and its mechanisms. Chemosphere. 2021; 264: 128436.
73. Slovic AD, Diniz CS, Ribeiro H. Clean air matters: An overview of traffic-related air pollution and pregnancy. Rev Saúde Pública. 2017; 51. doi: 10.1590/S1518-8787.2017051006652.
74. Heinrich J, Schwarze PE, Stilianakis N, Momas I, Medina S, Totlandsdal A, et al. Studies on health effects of transport-related air pollution. In: Health effects of transport-related air pollution. Copenhagen, Denmark: WHO Regional Office Europe; 2005. pp. 173.
75. Gaskins AJ, Mínguez Alarcón L, Fong KC, Abu Awad Y, Di Q, Chavarro JE, et al. Supplemental folate and the relationship between traffic-related air pollution and livebirth among women undergoing assisted reproduction. Am J Epidemiol. 2019; 188: 1595-1604.
76. Krajnak K, Waugh S, Welcome D, Xu XS, Warren C, McKinney W, et al. Effects of whole-body vibration on reproductive physiology in a rat model of whole-body vibration. J Toxicol Environ Health A. 2022; 85: 953-971.
77. Rocca MS, Foresta C, Ferlin A. Telomere length: Lights and shadows on their role in human reproduction. Biol Reprod. 2019; 100: 305-317.
78. Thilagavathi J, Venkatesh S, Dada R. Telomere length in reproduction. Andrologia. 2013; 45: 289-304.
79. Thilagavathi J, Kumar M, Mishra SS, Venkatesh S, Kumar R, Dada R. Analysis of sperm telomere length in men with idiopathic infertility. Arch Gynecol Obstet. 2013; 287: 803-807.
80. Bijnens EM, Zeegers MP, Derom C, Martens DS, Gielen M, Hageman GJ, et al. Telomere tracking from birth to adulthood and residential traffic exposure. BMC Med. 2017; 15: 205.