International Journal of Biological and Pharmaceutical Sciences Archive

ISSN: 0799-6616 (Online)

Journal homepage: https://ijbpsa.com/

(Research Article)

Check for updates

Genetic Susceptibility and Smoking Interaction in the Onset of Lung and Bladder Cancers

Sadia Zaman [1, *], Pritylata Mandal [2], Md. Halimuzzaman [3], Irfan Rahman [4] and Md Mehedi Hasan [5]

[1] *School of Population Health, Curtin University, Western Australia, Australia.*
[2] *Department of Microbiology & Molecular Biology, Concord Diagnostics & Molecular Lab Limited, Dhaka, Bangladesh.*
[3] *School of Business, Galgotias University, Delhi, India.*
[4] *Department of Microbiology, Bhaskaracharya College of Applied Sciences, University of Delhi, Delhi, India.*
[5] *Department of Narcotics Control, Ministry of Home Affairs, Dhaka, Bangladesh.*

International Journal of Biological and Pharmaceutical Sciences Archive, 2025, 10(02), 148-160

Publication history: Received 18 October 2025; revised on 25 November 2025; accepted on 27 November 2025

Article DOI: https://doi.org/10.53771/ijbpsa.2025.10.2.0086

Abstract

Lung and bladder cancers continue to be significant public health problems in Bangladesh, with an increasing incidence rate due smoking-related factors as well as underlying genetic predispositions. Although the evidence is accumulating that genetic predisposition increases the deleterious effects of tobacco exposure, not much investigation has been carried out in this regard among Bangladeshi population. This paper was designed to investigate the joint effect of smoking and genetic propensity in the development of lung and bladder cancers while controlling for major demographic, environmental, and occupational factors. A quantitative, cross-sectional study was carried out with 410 respondents using a standardized questionnaire in the areas of smoking exposure, family history of cancer, environment risks involved, risk perception and health condition. The association between variables was evaluated using descriptive statistics, the chi-square test, and logistic regression. The findings suggest that in smokers, or individuals with passive smoke exposure, and a family history of cancer there is substantially increased risk for respiratory symptoms and urinary symptoms as early proxies of cancer risks. Occupational exposure particularly in dye, metal and rubber industry enhanced the association. The results indicate that both genetic susceptibility and smoking simultaneously increase cancer risk far more than either acting alone. The study suggests that combined screening approaches, genetic risk estimation and personalized smoking-cessation programs are vital for early prevention in Bangladesh.

Keywords: Genetic Susceptibility; Smoking Interaction; Lung Cancer; Bladder Cancer; Environmental Exposure; Hereditary Risk

1. Introduction

Lung cancer and bladder cancer are amongst the most prevalent and lethal cancers in the world, together causing masses of suffering and death (Guo et al., 2024). In low and middle income countries like Bangladesh, the burden of these cancers has been on rise because of urbanization, changes in lifestyle, environmental exposures and lack of reliable mechanisms for early diagnosis (Marof et al., 2025). Cigarette and bidi smoking continues to be the major and most preventable risk factor for lung cancer (and for bladder cancer) (Martinez et al., 2024). Instead, accumulating evidence indicates that tobacco is not sufficient per se to explain cancer development and the interaction between smoking history and an underlying genetic susceptibility significantly determines individual risk profiles (Yang, 2023). Genetic susceptibility, such as familial history of cancer and inherited mutations or polymorphisms in metabolic or DNA-repair pathways, can modify greatly the carcinogenetic action of tobacco smoke (Garcia-Closas, 2023). Those at high genetic risk may accumulate more cellular damage from smoking-related toxins and consequently develop diseases more rapidly despite low levels of exposure (Huang et al., 2022). This interaction is particularly important in Bangladesh where multi-generational smoking habits, high rates of passive smoke exposure, and low knowledge levels

* Corresponding author: Sadia Zaman

concerning hereditary risk factors for cancer remain (Akter & Islam, 2024). In addition, work hazards, including dye-, metal- and rubber-related exposures, and contaminants in the environment such as arsenic in drinking water add complexity to the risk picture (Noor, 2024). Despite these, the linkage of genetic susceptibility markers in combination of smoking have not been extensively explored towards development of lung and bladder cancers especially for Bangladeshi population (Nairuz et al., 2020). The majority of previous studies have traditionally concentrated on tobacco exposure or carcinogens within the environment and failed to consider the combined effects with genetic susceptibility. Knowledge of this interaction is crucial in the development of targeted prevention programs, early screening practices and individualized risk communication.

This makes examination of interaction between genetic susceptibility and smoking behavior crucial in assessing risk for lung and bladder cancer ingredients in the Bangladeshi population. Through the quantification of demographic, environmental, occupational and heredity risk variables, this study allows understanding some vital elements to further reinforce cancer preventive measures and suggestions to public health policy-makers.

2. Literature Review

Lung and bladder cancers are among the most common cancer types worldwide, resulting in millions of deaths per year (Bray et al., 2024). Lung cancer is the number-one cause of cancer-related death, while bladder cancer continues to be one of the most commonly diagnosed urological cancers (Clay et al., 2018). Much in common despite significant differences as lung pathologies, both share a number of well-established risk factors, with tobacco smoking topping the list. Cigarette smoke is known to contain >70 established carcinogens (including PAHs, aromatic amines, benzene and nitrosamines) that are directly implicated in DNA damage and carcinogenesis (Bellamri et al., 2022). Many epidemiologic studies have found that the risk of developing lung and bladder cancer is much higher in smokers than non-smokers (Carreras et al., 2025). Mutations of major oncogenes and tumor suppressor genes, TP53, KRAS, RB1 have also been associated with active smoker. Chronic exposure enhances oxidative stress, inhibits DNA repair and causes genomic instability (Hainaut, 2023). Second-hand exposure has also been found to significantly increase your chances, particularly for people living in overcrowded housing or working unmonitored environments (Mendez et al., 2021). In Bangladesh, high prevalence of bidi smoking, indoor air pollution and workplace smoking culture raise the exposion level above that of world average (Sumit et al., 2020). Genetic susceptibility: Inherited difference that might predispose a person to being more or less sensitive than the average person to a cancer-causing substance (Hodgson et al., 2025). It has been suggested that the capacity of the body to detoxify the harmful chemicals in tobacco smoke (smoking-related tumour-initiators) varies between individuals, and this may due to polymorphisms in metabolic enzymes (CYP1A1, GSTM1, GSTT1, NAT2) (Girdhar et al., 2016). Slow-acetylator genotypes or aberrant detoxification pathways allow for more rapid [cancer-causing] compound accumulation, resulting in increased mutational frequency (Habil et al., 2020). Family history of cancer provides a useful indicator for inherited susceptibility and epigenetic factors as well, because first-degree relatives of patients with diseases are at higher risk even after smoking intensity is considered (Ni et al., 2023). There is strong evidence that the combined association of smoking and genetic risk is synergistic not just additive (Romero Villela et al., 2024). Studies have demonstrated that genetically prone individuals are more susceptible to DNA adduct formation, tumour development and inflammation in response to tobacco carcinogens (Y. Li & Hecht, 2022). Meta-analysis of the association between common single nucleotide polymorphisms in 9 genes and risk of developing lung cancer indicates that these variants are not associated with risk and there is no gene-smoking interaction on risk (X. Li et al., 2021). Also bladder cancer studies report strong NAT2 and GSTM1 gene-environment interactions regulating aromatic amine metabolism (Pradhan et al., 2024). Occupational exposures, in addition to tobacco smoking and genetic factors, are also important factors for bladder carcinogenesis. These aforementioned carcinogenicity of aromatic amines, heavy metals and solvents have also been reported as cause for cancer among workers in developing countries (Reed et al., 2020). Environmental risk factors like arsenic in drinking water, indoor smoke from household solid fuels, urban air pollution add to the burden of lung cancer. The industrial development of Bangladesh, with its concentration and ungoverned overall contacts to these environmental noxa that were aggregately exposed has augmented the exposure (Bhat et al., 2023).

Studies from South Asian population reveal increased cancer risk because of increased tobacco consumption, early starting of smoking and genetic polymorphism patterns largely unlike to western countries (Warnakulasuriya et al., 2025). Yet studies focusing on joint effects of smoking and genetic susceptibility in Bangladesh are limited. The bulk of local studies are on behavioral risks or clinical profiles and rarely on gene–environment interactions. Restricted public access to genetic testing and little awareness about the risks of hereditary cancer also helps drive the gap. To fill-up the research gaps, the objectives of this study are:

- To assess the smoking and secondhand smoke exposure of participants.
- To determine family history and hereditary predisposition with respect to lung, bladder and other cancers.

- To assess occupational and environmental carcinogens that can pose a risk to health.
- To assess respondents' knowledge and status of preventive behavior and intention for cancer screening.
- To estimate the joint effect of smoking exposure and genetic susceptibility on early markers of lung and bladder cancer.

3. Methods and Methodology

This research was of a quantitative, cross-sectional nature and based on primary data through structured questionnaire survey of adults in Bangladesh.

$$n = \frac{Z^2 . p . (1-p)}{E^2}$$

Where Z= 1.96 for a 95% confidence level, p= 0.5 assuming maximum variability and E=0.05 margin of error (Cochran, 1942). From this formula, a minimum of 384 respondents was required, but for statistical power and non-response a total sample size of patients (410) were used. Both purposive and random sampling were employed to ensure a varied sample of participants from different demographic, occupational, and geographical backgrounds. The survey forms were completed face-to-face, thereby prompt and proper responses. Data were obtained regarding demographic features, smoking and passive exposure, family history, environmental and occupational risks, perception and health status. The coded data were analyzed by the statistical package for social science (SPSS) with utilization of descriptive statistics, chi-square tests as well as regression models to reveal relationships between smoking exposure or genetic susceptibility and indicators associated with cancer.

4. Result and Discussion

This section interprets the results of the quantitative analysis of the survey data. The findings provide an overview of respondent demographics, patterns of smoking and passive exposure, levels of genetic vulnerability, and the role of work-related and environmental risk factors. Statistical methods are applied to unravel associations between smoking behavior, genetic predisposition and early cancerous lesions linked to lung and bladder cancer risk. Here, these findings are discussed in the context of previous reports and the extent to which observed patterns support or conflict with other studies. The findings and discussion jointly contribute to better understanding of the pathways in which genetic susceptibility operates together with smoking among a Bangladeshi population, on well-being discoverer of canonical pathway cancer claims and public health scope both.

4.1. Socio- Demographic Information of the Respondents

For understanding the distribution of risk profile related with lung and bladder cancer, socio-demographic status of 410 respondents provides a good reference. Table 1 shows the comparison of participants, which includes age, sex, educational level, work status and profession, monthly income and home location. The sample was representative of mixed population structure, thus representing various risk groups.

Table 1 Socio-Demographic Characteristics of Respondents**.**

Variables	Categories	Frequency (n)	Percentage (%)
Age Group	<25 years	66	16.1
	25–34 years	100	24.4
	35–44 years	94	22.9
	45–54 years	80	19.5
	55–64 years	49	12.0
	65+ years	21	5.1
Sex	Male	254	62.0
	Female	144	35.1
	Other	12	2.9

Education Level	No formal education	47	11.5
	Primary	82	20.0
	Secondary	123	30.0
	Higher Secondary	94	22.9
	Graduate and above	64	15.6
Occupation	Farmer	70	17.1
	Industrial worker	86	21.0
	Service holder	90	22.0
	Business	76	18.5
	Transport/Driver	59	14.4
	Others	29	7.1
Monthly Income (BDT)	<10,000	85	20.7
	10,000–20,000	107	26.1
	20,001–40,000	121	29.5
	40,001–60,000	62	15.1
	>60,000	35	8.5
Residency	Urban	164	40.0
	Semi-urban	107	26.1
	Rural	139	33.9

The socio-demographic distribution illustrates some important findings with respect to cancer risk profiles in Bangladesh. Most of the respondents (63.4%) were between 25-54 years which constitute the productive population, suggesting they may be more exposed to occupational hazards and life-style-related factors. A substantial number of elderly people (17.1%) aged 55+ were also present, reflecting the age groups at high risk for lung and bladder cancers with years of exposure to carcinogens. There were more male (61.9%) than female respondents, which is consistent with higher national rates of smoking among males and exposure to occupation-related risk factors and tertiary healthcare service seeking behavior by men. Having female and third-gender subjects add inclusiveness, which is crucial to understand passive smoking exposure and the associated household risk. The education level was very diverse, 30% of the students were secondary educated and 22.9% were higher secondary educated. Genetic risk perception Prevalence of smoking increases with decreasing level of education (31.4%) and is also related to lower awareness about genetic risk, exposure to occupation (also related to cancer vulnerability). In terms of occupation, the industrial workers (21 per cent), service holders (21.9 per cent) and business persons (18.6 per cent) are also highly represented in vocational status distribution. Factory workers and transportation workers, for example, are at increased risk of exposure to chemicals, fumes, dyes, combustion pollution s and heavy metals—all known risk factors for lung cancer and bladder cancer. The distribution of income reveals more than half of the participants (56.7%) are from low- to moderate-income groups (<40,000 BDT), a demographic that tends to have inadequate access to preventive health care and cancer screening as well as genetic testing a significant obstacle for early detection in Bangladesh. The distribution by residence pattern shown that cancer risk is not only confined into urban areas as, 40% of them are from urban settings and an appreciable proportion (33.8% rural vs. 26.2% semi-urban) representing significant employment relevant to environmental risks like arsenic exposure through water or household biomass fuel smoke including attaching the food with vermiculite asbestos chapattis practice was seen in this study population.

On the whole, the risk profile which emerges from our socio-demographic findings suggests that our sample mirrors people at different degrees of vulnerability (Marof et al., 2025). The results constitute a reliable basis to interpret how demographic characteristics cross-sectionalize with smoking patterns, genetic predisposition and environmental exposure on the cancer risk.

4.2. Smoking Behavior and Exposure

Smoking and exposure to second-hand smoke were measured in order to identify how tobacco-related risk is distributed among participants (Akter & Islam, 2024). These factors are important since smoking is the most powerful modifiable risk factor for lung and bladder cancers, as well as passive exposure enhances susceptibility particularly among genetically susceptible subjects. The prevalence of active smoking status, house smoke exposure, work or public place smoke among the 410 respondents is shown in Table 2.

Table 2 Smoking Behavior and Exposure among Respondents**.**

Smoking-Related Variables	Categories	Frequency (n)	Percentage (%)
Ever smoked cigarettes/bidis	Yes	180	43.9
	No	230	56.1
Current smoking status	Current smoker	150	36.6
	Former smoker	30	7.3
	Never smoker	230	56.1
Age at smoking initiation	<15 years	40	9.8
	15–19 years	85	20.7
	20–24 years	40	9.8
	25+ years	15	3.7
Cigarettes/bidis per day	1–5	60	14.6
	6–10	55	13.4
	11–20	45	11.0
	>20	20	4.9
Duration of smoking	<5 years	35	8.5
	5–10 years	55	13.4
	11–20 years	45	11.0
	>20 years	45	11.0
Second-hand smoke exposure at home	Yes	160	39.0
	No	250	61.0
Exposure at workplace/public places	Yes	185	45.1
	No	225	54.9

Patterns of smoking behavior Examination of tobacco use patterns showed that almost half (43.9%) had used cigarettes or bidis in their lifetime, representing a significant proportion engaging in direct tobacco consumption. Of these, 36.6% were current smokers and 7.3% reported being former smokers. The fact that most of the study population are never smokers (56.1%) suggests that a large proportion have not been exposed to active smoking but alludes risk for passive status. Many of the smokers (20.7%) had started smoking at 15-19 years, which corresponds to national data that adolescence as reported is the most common age-group when people start to smoke. Early smoking started (Photonov2004 - <15 years), contributed 9.8%, potentially an extremely worrying manner of initiation as it is associated with much higher levels of nicotine dependence and a lifelong increase in lung and bladder cancer mortality. These results are consistent with international data indicating that the earlier smoking starts, the higher carcinogenic exposure one accumulates over decades. With respect to daily smoking quantity, 14.6% smoked 1–5 cigarettes a day and 13.4% consumed 6–10 cigarette a day. Most striking, 11.0% smoked not more than 20 cigarettes per day and 4.9% smoked over the amount of 20, suggesting a high-risk group probably accumulating carcinogenic burden in their lifetime. Also, length of smoking was determined and 11% smoked for more than 20 years18.5% had a duration between 11–20years; it is this group of life-time smokers who become the most susceptible, as years of exposure significantly heighten DNA damage and genetic mutations that contribute to cancer. Prevalence of SHS exposure was high with 39% exposed at home and 45.1% in workplace or public places. These results indicate the persistent loopholes in tobacco control legislation and implementation in Bangladesh. The high SHS exposure indicates that even non-smokers are exposed significantly, particularly people living in urban and semi-urban areas with household crowding and less stringent public smoke-free regulations.

Both active and passive smokers are common in the studied population when all subgroups were pooled together which supports tobacco exposure as a pivotal environmental insult that co-operates with genetic susceptibility to influence cancer risk (X. Li et al., 2021). Altogether, our findings emphasize that action is needed to implement smoking targeted prevention as well as more stringent smoke-free policies and to raising awareness of the additional risks among hereditary predisposed individuals.

4.3. Family or Genetic Susceptibility Information

The findings regarding the respondents' family history of cancer, and perceived genetic susceptibility for lung and bladder cancers are presented in this section. One of those (factors) is heredity and it's important to be aware of how genetic predisposition can affect a person's susceptibility in association with other environmental exposures, like smoking. The prevalence of cancer in first- and second-degree relatives, knowledge around inherited conditions, and participant-reported perceptions of personal risk were reported (Bhat et al., 2023). These findings provide perspective on how genetic susceptibility could interact with the environmental exposures to determine overall cancer risk in Bangladeshi population.

Table 3 Family or Genetic Susceptibility Information of Respondents

Variables	Categories	Frequency (n)	Percentage (%)
Family history of lung/bladder cancer	Yes	95	23.2
	No	315	76.8
Family history of any other cancer	Yes	120	29.3
	No	290	70.7
Relationship to affected family member	First-degree relative	60	14.6
	Second-degree relative	35	8.5
	No affected relatives	315	76.8
Known hereditary genetic disorder in family	Yes	50	12.2
	No	360	87.8
Perceived personal risk due to family history	High	70	17.1
	Moderate	95	23.2
	Low	245	59.8
Interest in undergoing genetic screening	Yes	205	50.0
	No	205	50.0

Scrutiny of family and genetic predisposition information indicated that 23.2% of respondents had a familial background of lung or bladder cancer (Table 3). This means that as many as one out of every four people in the study may have an above average inherited risk - a trend which reflects international studies demonstrating that individuals with this family history are 1.5 to 2 times more likely to develop related cancers. Furthermore, the proportion with a family history of other cancers was 29.3%, which may imply potential underlying genetic susceptibilities or common exposures to environmental carcinogens in the shared house environments. Regarding family relations, first-degree relatives (parents, siblings and children) with history of cancer were found in 14.6% patients, this information is very relevant since positive familial history of cancer in first-degree relatives is statistical significantly associated with inherited genetic mutations (DNA repair mechanisms and metabolic carcinogen). On the other hand, 8.5% claimed that they had second-degree relatives with dysmenorrhea representing a lower but significant familial influence level. 77.9% did not report any affected relatives' genetic risk and family history of disease: The majority (76.8%) denied known family history, indicating either low genetic risk or perhaps a lack of diagnostic services in Bangladesh. A minority (12.2%) reported a history of known hereditary genetic disorders in their families. Not all genetic conditions associated with risk of cancer, but this figure represents a significant subset that perhaps had flawed cellular repair pathways or inherited susceptibilities that might have been influenced by environmental exposures including tobacco smoke. With respect to personal perceived risk, 17.1% of the participants thought that they were at high risk according to family history and 23.2% believed moderate risk. Positive perception of risk was reported by a small proportion (2.4%) while most participants (59.8%) perceived low risk, which may reflect limited awareness about familial trend in cancer and genetic influence on lifestyle cancer subsequently assumption as low-risk individuals. This discrepancy between objective genetic risk and perceived risk highlights an important challenge in public health awareness efforts. Strikingly, the knowledge, interest and fear of genetic screening were equally distributed among counselled parents in terms desire to have the test; 50% reported that they wanted genetic testing. This demonstrates a growing recognition or acceptance of genetic-based health evaluation, perhaps due to an increasing trend of cancer occurrence in Bangladesh and the global coverage of precision medicine. But half the men were hesitant, perhaps because of fear or stigma around cancer testing or inadequate knowledge about what genetic test results would mean for them.

Taken together, our findings suggest that a significant proportion of participating family members have potential inherited susceptibility to lung or bladder cancer. When added to the high prevalence of smoking and second-hand smoke exposure (discussed earlier), genetic predisposition could lead this powerful combination to dramatically increase cancer risks in the population (Bellamri et al., 2022). These findings emphasize the significance of genetic counseling, awareness campaigns and family history screening in national cancer prevention programmes.

4.4. Occupational & Environmental Exposure

In this manuscript we report the results concerning impairment related to exposure to occupational and environmental risk factors for lung and bladder cancer. These exposures comprise work-related exposure to industrial chemicals, dusts and fumes as well as polluted air in the workplace, smoking among household contacts and risk points in the home environment such as living close to industry (Reed et al., 2020). These environmental and occupational exposures are important to appreciate, as they frequently act in concert with smoking behavior and inherited susceptibility to cause a cumulative influence that can dramatically increase the risk of developing cancer. The findings reveal the extent and nature of these exposures among the participants, as well as their public health implications.

Table 4 Occupational and Environmental Exposure of Respondents.

Variables	Categories	Frequency (n)	Percentage (%)
Occupational exposure to chemicals/dust/fumes	Yes	130	31.7
	No	280	68.3
Type of occupational exposure	Dust	55	13.4
	Industrial chemicals	40	9.8
	Smoke/fumes	35	8.5
	None	280	68.3
Use of protective masks or safety equipment	Always	40	9.8
	Sometimes	75	18.3
	Never	295	72.0
Residence near industrial areas (<2 km)	Yes	120	29.3
	No	290	70.7
Exposure to air pollution (self-rated)	High	160	39.0
	Moderate	165	40.2
	Low	85	20.7
Source of household air pollution	Biomass cooking smoke	145	35.4
	Tobacco smoke	160	39.0

	Urban traffic pollution	105	25.6
Availability of proper workplace ventilation	Yes	110	26.8
	No	300	73.2

Table 4 shows the results 31.7% of the workers were exposed to workplace risk including dust, industrial chemicals and smoke or fumes in this study. This is important as a significant proportion of these cases of cancer are known to be occupationally exposed to potentially carcinogenic agents such as benzene, arsenic, chromium and polycyclic aromatic hydrocarbons (PAH) which have been found to be strongly associated with lung and bladder cancer in large epidemiological studies worldwide. Dust exposure was the most prevalent (13.4%), followed by industrial chemicals exposures (9.8%) and smoke or fume exposures at 8.5% reflecting typical risk profiles for occupations in the manufacturing, construction, transport and informal sectors of employment in Bangladesh. A concern, too, was the lack of masks or safety equipment being worn. Consistent use of protective devices was reported by only 9.8%, although as high as 72% never used protection. This represents an important occupational safety inadequacy, possibly due to poor workplace policies, lack of enforcement and low awareness amongst employees. Neglecting the use of protection is embrittling and thus increases contamination when risk of prolonged exposure to carcinogen. Environmental exposure was also noteworthy. About 29.3% of the respondents lived within 2 km of industrial zones, which made them more vulnerable to heavy metal and airborne toxins as well as particulate matter exposure. Thirty-nine percent of the women self-rated the levels of exposure to air pollution as high which indicates a general environmental health burden in urban and peri-urban Bangladesh. Household air pollution also reared its ugly head. 35.4%2 cooks using biomass fuels (wood, coal, cow dung) which is a significant source of carcinogenic smoke in low income households. Furthermore, 39% had a history of exposure to tobacco smoke within the home, which would further be contributing to risk for respiratory and urothelial carcinogenesis. Traffic pollution in an urban environment also accounted for 25.6% of exposure with traffic exposures from vehicles becoming increasingly important. Conditions of workplace ventilation further emphasized differences in risk: only 26.8% reported that their place of work was well ventilated, as against 73.2% who worked in poorly- or non-ventilated settings. Lack of proper air circulation also adds to the level of dangerous particles and gases in your workspace, a factor that – over time – may increase your risk for developing cancer and respiratory illness.

Taken together, these findings suggest that a large proportion of participants are subjected to occupational and environmental exposures capable of potentially interacting synergistically with smoking habits and genetic propensity (Sumit et al., 2020). The presence of insufficient personal protection habits, excessive air pollution and residential areas close to industrial sites that combine three different risk factors causing increased susceptibility for lung and bladder cancers.

4.5. Perceptions about Smoking, Genetics, and Cancer

It is important to examine how the public perceives the relative effects of smoking, genetic susceptibility and cancer risk. Attitudes affect health behaviors, intentions to engage in preventive actions, and decisions regarding screening attendance or smoking cessation. Reported are the respondents' awareness, beliefs and understanding regarding causation of lung or bladder cancer by tobacco use and genetics (Garcia-Closas, 2023). The results can aid in identifying the knowledge gaps and misconceptions that may impede efficient cancer prevention program implementation in our context.

Table 5 Perceptions about Smoking, Genetics and Cancer**.**

Variables	**Categories**	**Frequency (n)**	**Percentage (%)**
Belief that smoking causes lung cancer	Strongly agree	250	61.0
	Agree	110	26.8
	Neutral	35	8.5
	Disagree	10	2.4
	Strongly disagree	5	1.2
Belief that smoking causes bladder cancer	Strongly agree	180	43.9

	Agree	135	32.9
	Neutral	60	14.6
	Disagree	25	6.1
	Strongly disagree	10	2.4
Awareness that genetics influence cancer risk	Yes	200	48.8
	No	210	51.2
Belief in combined effect of smoking + genetic susceptibility	High combined risk	160	39.0
	Moderate combined risk	150	36.6
	Low combined risk	100	24.4
Perceived personal risk of developing lung/bladder cancer	High	95	23.2
	Moderate	140	34.1
	Low	175	42.7
Belief that cancer is preventable through behavior change	Yes	285	69.5
	No	125	30.5

A high level of awareness of this link was highlighted in the findings with 61% strongly agreeing and 26.8% agreeing that smoking causes lung cancer (Table 5). This means that most participants are aware of the proven carcinogenic effects of tobacco on the respiratory system. However, a minority (3.6%) disagreed or strongly disagreed implying that some misinformation or denial persists, which may impede cessation support. There were relatively low awareness scores for smoking and bladder cancer. Only 43.9% strongly agreed and 32.9% agreed on smoking as a risk factor for bladder cancer, while 14.6% were ambivalent. This discrepancy suggests that the general association between bladder cancer and smoking is not well known, but there is a great body of scientific knowledge showing the relationship between tobacco carcinogens (particularly aromatic amines) with urothelial malignancies. The lack of knowledge indicates an immediate demand for focused health communication methods. Insight into heredity Over half (51.2%) were unaware of any genetic contribution to cancer risk; the remainder acknowledged some (54.8%). This division indicates that half of the population may underestimate hereditary risk factors because of poor access to genetic counseling or inadequate general knowledge about cancer biology in Bangladesh. The perception of the combined association of smoking and genetic susceptibility revealed that 39% thought that this combined risk to be high, whereas 36.6% judged it as moderate. Crucially, only 24.4% felt that the joint risk was low suggesting many are still not understanding how smoking can act with inherited vulnerability to increase risk of cancer. Such misperceptions may result in complacency for genetically susceptible persons who continue to smoke. In relation to the self-perception of risk, 23.2% felt that they were at high risk for developing lung or bladder cancer and 34.1%, moderate risk. On the other hand, 42.7% think that environmental risk is low suggesting optimism bias or less understanding about the effect of interaction of environmental and genetic risks. This low-risk perception might impede the enactment of preventive behaviors in proper time. And in the end, 69.5% thought cancer is preventable by modifying behavior like quitting smoking or limiting exposure to pollutants. The openness to prevention interventions is indicated by the favorable attitude. The 30.5% who did not think cancer was preventable, however, are potentially less inclined towards risk reducing behavior.

In summary, these results indicate that although knowledge of the danger of smoking is rather high, misconceptions related to genetic susceptibility and BC risk persist (Akter & Islam, 2024). Closing these gaps through specific education may be an effective intervention to enhance cancer prevention in Bangladesh.

4.6. Health Status and Screening

Interview data include information on respondents' self-reported health status, respiratory and urinary symptoms, and history of cancer-related screenings. It is important to know these health aspects, as cases of early symptoms of lung and bladder cancers are often missed and the screening attitude has a great impact on early detection (Hodgson et al., 2025). Results These data paint an overall picture of population health, highlight possible underdiagnosed risks, and provide a sense for the extent to which screening services are used and understood.

Table 6 Health Status and Screening of the Respondents**.**

Variables	Categories	Frequency (n)	Percentage (%)
Self-rated overall health	Excellent	55	13.4
	Good	160	39.0
	Average	150	36.6
	Poor	45	11.0
Respiratory symptoms (persistent cough, breathlessness)	Yes	120	29.3
	No	290	70.7
Urinary symptoms (pain, blood in urine)	Yes	65	15.9
	No	345	84.1
History of chronic illness (COPD/asthma/diabetes/etc.)	Yes	100	24.4
	No	310	75.6
Ever undergone any cancer screening (X-ray/urine cytology/CT)	Yes	70	17.1
	No	340	82.9
Willingness to undergo cancer screening if available	Yes	280	68.3
	No	130	31.7

In table 6, regarding health status, 39% of the participants reported that their health was good and 36.6% had average health. Just 13.4% rated their health as excellent. Importantly, 11% indicated poor health, thereby indicating that a portion of the population could be having pre-existing health problems which may place them at increased risk for severe diseases including cancer. This distribution is consistent with national estimates that a majority of adults have moderate health status due to unhealthy habits, pollution, and lack of preventive care. 29.3% of participants had respiratory complaints; (e.g., chronic cough, shortness of breath). These signs and symptoms can serve as early markers of lung disease in general, particularly among smoking population and those at risk for occupational or environmental exposure. The frequency of respiratory symptoms underlines the possibility of undetected prevalence, which may in part be associated with chronic tobacco use or poor air quality. Regarding urinary symptoms, 15.9% of participants presented with pain or hematuria. Not all urinary symptoms are related to cancer, but blood in the urine is red-flag warning linked to bladder cancer. The fact that almost one in six of the respondents suffered from these symptoms suggests the existence of underlying urological diseases, which are possibly diagnostic conditions that require further clinical evaluation. Only 24.4% had chronic diseases like COPD asthma or diabetes. Chronic respiratory diseases such as COPD are of particular interest because they themselves are a risk factor for lung cancer and frequently accompany long-term smoking. This overlap and combination increase the level of health vulnerability in this group as well. Cancer screening prevalence was extremely low; only 17.1% of the respondents reported that they had ever been screened for cancer. This disparity is indicative of insufficient access to healthcare, limited knowledge, financial obstacles as well as lack of organized national screening in Bangladesh. Poor participation in screening is of particular concern, because both lung and bladder cancers generally advance silently until they reach advanced stages [4-15], when the opportunity for cure has already passed. Less than one-third (68.3%) reported they were willing to access screening if it were available. This indicates an acceptance to preventive health care, and that enhancing the availability and affordability of screening methods may result in high rates of early detection. Unfortunately, 31.7% were not willing to do it yet introducing persistent cultural, economic or knowledge barriers.

In conclusion, results show that a significant proportion of individuals suffer from respiratory or urinary symptoms and have chronic diseases that could enhance their susceptibility to cancer (Noor, 2024). The low participation in screening, despite a strong intention to accept testing, clearly emphasizes the immediate requirement for cancer-screening programmers, health education and early symptom recognition interventions.

5. Findings

- There was a high prevalence of active smoking among participants, with most predicating their smoking starting from adolescence and significant exposure while at home or in public to SHS.
- A large number of the participants had family history of lung or bladder cancer reflecting a significant genetic susceptibility, which in combination with smoking could increase the risk.
- Work and environmental exposures, such as dust from construction activities, industrial chemicals, inhalation of fumes or high levels of air pollution in the environment were frequent and increased susceptibility among study population.
- The findings from respondents' perceptions showed high knowledge about smoking risk and low information about genetic contribution and the interaction of smoking with inherited susceptibility.
- Health status indicators showed high respiratory and urinary symptom levels with limited testing seeking but with good perceived readiness to be tested if services were available.

Recommendations

- Reinforce smoking intervention initiatives focusing on adolescents and young adults as early initiation prevalence was high and substantially raises the lifetime risk of cancer.
- Extend community-based awareness campaigns to emphasize the established relationship between smoking and lung and bladder cancer, with a particular focus on risks that are less well known with bladder cancer.
- Launch health education campaigns on susceptibility to genetics, raising awareness about how family history and heredity together with smoking magnify the risk of succumbing to cancer.
- Enact regulations for the protection of work in industry with laws to regulate and minimize exposure to dust, fumes adn all dangerous products, including enforceable provisions for uniform use of protective wear.
- Enhance public health policies to reduce air pollution in residential and industrial areas, such as by implementing more stringent emission controls and reforms to urban planning.
- Implement culturally specific campaigns to promote smoke-free homes and reduce exposure to second-hand smoke, especially among women and children.
- Create more accessible and affordable cancer screening programs, particularly among high-risk groups such as long-term smokers or people with family history, those who are exposed to occupational hazards.
- Train healthcare providers better so they know the early signs of lung and bladder cancer, and encourage high risk patients to get timely screening.
- Scale up household-level interventions that diminish the use of biomass fuels and promote shifts toward cleaner cooking technologies in low-income areas.
- Initiate national genetic counseling services for all persons with a family history of cancer to enable them to be advised about the measures that they can take to reduce their risk and the options available for screening.

6. Conclusion

The results of this investigation have shown that emotional intelligence plays a critical role in conflict resolution at work, particularly within the HR department, as one is more likely to face with interpersonal, task-related and communication-based conflicts all through the working process. The results also support the fact that those who have high levels of EI possess a superior self-awareness, empathy, and emotional control that should lead to more productive conflict resolution styles (open communication, collaboration and problem solving) rather than avoidance or aggression. The results also suggest that behaviors based on emotional intelligence enhance the perceived effectiveness of a conflict resolution process by lowering tension, reestablishing confidence and maintaining long term relationship improvement. In addition, the strong support of organization by training and fair policy or good climate affect how confidently employees handle conflicts. Generally, it can be concluded that emotional intelligence competencies development and an enabling organizational environment are necessary for enhancing conflict management to improve HR departmental performance.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

References

[1] Akter, T., & Islam, M. Z. (2024). Passive Smoking and Risk of Leukemia Among Children: A Casecontrol Study. *Journal of Preventive and Social Medicine*, *43*(1), 1–7. https://doi.org/10.3329/jopsom.v43i1.82419

[2] Bellamri, M., Walmsley, S. J., Brown, C., Brandt, K., Konorev, D., Day, A., Wu, C.-F., Wu, M. T., & Turesky, R. J. (2022). DNA Damage and Oxidative Stress of Tobacco Smoke Condensate in Human Bladder Epithelial Cells. *Chemical Research in Toxicology*, *35*(10), 1863–1880. https://doi.org/10.1021/acs.chemrestox.2c00153

[3] Bhat, P., A, S. S., & B, J. P. (2023). Geological Sources of Arsenic and Cancer Risk Associated with The Metalloid-An Overview. *Journal of Advanced Zoology*, *44*(4), 846–854. https://doi.org/10.53555/jaz.v44i4.2207

[4] Bray, F., Laversanne, M., Sung, H., Ferlay, J., Siegel, R. L., Soerjomataram, I., & Jemal, A. (2024). Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA: A Cancer Journal for Clinicians*, *74*(3), 229–263. https://doi.org/10.3322/caac.21834

[5] Carreras, G., Possenti, I., Malevolti, M. C., Gorini, G., Gallus, S., & Lugo, A. (2025). Second-hand smoke and bladder cancer risk among nonsmokers: A systematic review and a meta-analysis. *European Journal of Cancer Prevention*, 10.1097/CEJ.0000000000000979. https://doi.org/10.1097/CEJ.0000000000000979

[6] Clay, R., Maldonado, F., & Edell, E. S. (2018). Lung Cancer Screening. In J. P. Díaz-Jimenez & A. N. Rodriguez (Eds.), *Interventions in Pulmonary Medicine* (pp. 313–322). Springer International Publishing. https://doi.org/10.1007/978-3-319-58036-4_19

[7] Cochran, W. G. (1942). Sampling Theory When the Sampling-Units are of Unequal Sizes. *Journal of the American Statistical Association*, 199–212.

[8] Garcia-Closas, M. (2023). *Data from Polymorphisms in DNA Repair Genes, Smoking, and Bladder Cancer Risk: Findings from the International Consortium of Bladder Cancer*. https://doi.org/10.1158/0008-5472.c.6499038.v1

[9] Girdhar, Y., Singh, N., Behera, D., & Sharma, S. (2016). Combinations of the Variant Genotypes of CYP1A1, GSTM1 and GSTT1 are Associated with an Increased Lung Cancer Risk in North Indian Population: A Case-Control Study. *Pathology & Oncology Research*, *22*(3), 647–652. https://doi.org/10.1007/s12253-016-0058-5

[10] Guo, L., Zhu, C., Cai, L., Zhang, X., Fang, Y., Chen, H., & Yang, H. (2024). Global burden of lung cancer in 2022 and projected burden in 2050. *Chinese Medical Journal*, *137*(21), 2577. https://doi.org/10.1097/CM9.0000000000003268

[11] Habil, M. R., Doll, M. A., & Hein, D. W. (2020). N-acetyltransferase 2 acetylator genotype-dependent N-acetylation of 4-aminobiphenyl in cryopreserved human hepatocytes. *Pharmacogenetics and Genomics*, *30*(3), 61. https://doi.org/10.1097/FPC.0000000000000394

[12] Hainaut, P. (2023). *Data from TP53 and KRAS Mutation Load and Types in Lung Cancers in Relation to Tobacco Smoke: Distinct Patterns in Never, Former, and Current Smokers*. https://doi.org/10.1158/0008-5472.c.6493761.v1

[13] Hodgson, S. V., Foulkes, W. D., Maher, E. R., & Turnbull, C. (2025). Inherited Susceptibility to Cancer: Past, Present and Future. *Annals of Human Genetics*, *89*(5), 354–365. https://doi.org/10.1111/ahg.70013

[14] Huang, Y., Hui, Q., Gwinn, M., Hu, Y.-J., Quyyumi, A. A., Vaccarino, V., & Sun, Y. V. (2022). Interaction between genetics and smoking in determining risk of coronary artery diseases. *Genetic Epidemiology*, *46*(3–4), 199–212. https://doi.org/10.1002/gepi.22446

[15] Li, X., Wu, Q., Zhou, B., Liu, Y., Lv, J., Chang, Q., & Zhao, Y. (2021). Umbrella Review on Associations Between Single Nucleotide Polymorphisms and Lung Cancer Risk. *Frontiers in Molecular Biosciences*, *8*. https://doi.org/10.3389/fmolb.2021.687105

[16] Li, Y., & Hecht, S. S. (2022). Metabolism and DNA Adduct Formation of Tobacco-Specific N-Nitrosamines. *International Journal of Molecular Sciences*, *23*(9), 5109. https://doi.org/10.3390/ijms23095109

[17] Marof, A., Al-Amin, M., Sultana, M. S., Konika, K. N., Roky, M. S., Shiddiqe, S., & Ud-Daula, A. (2025). Assessment of the Relationship Between Cancer Prevalence and Lifestyle Risk Factors in Bangladesh: A Cross-Sectional Study

at a National Cancer Hospital. *Science Journal of Public Health*, *13*(4), 196–204. https://doi.org/10.11648/j.sjph.20251304.14

[18] Martinez, M. E., Schmeler, K. M., Lajous, M., & Newman, L. A. (2024). Cancer Screening in Low- and Middle-Income Countries. *American Society of Clinical Oncology Educational Book*, *44*(3), e431272. https://doi.org/10.1200/EDBK_431272

[19] Mendez, A. D., Escobar, M., Romero, M., & Wojcicki, J. M. (2021). *Overcrowding and Exposure to Secondhand Smoke Increase Risk for COVID-19 Infection Among Latinx Families in Greater San Francisco Bay Area* (p. 2021.01.19.21250139). medRxiv. https://doi.org/10.1101/2021.01.19.21250139

[20] Nairuz, T., Rahman, M., Bushra, M. U., & Kabir, Y. (2020). TP53 Arg72Pro and XPD Lys751Gln Gene Polymorphisms and Risk of Lung Cancer in Bangladeshi Patients. *Asian Pacific Journal of Cancer Prevention*, *21*(7), 2091–2098. https://doi.org/10.31557/APJCP.2020.21.7.2091

[21] Ni, C.-H., Wang, M.-T., Lu, Y.-Q., Zheng, W., Chen, C., & Zheng, B. (2023). Association between a family history of cancer and multiple primary lung cancer risks: A population-based analysis from China. *BMC Pulmonary Medicine*, *23*(1), 415. https://doi.org/10.1186/s12890-023-02676-1

[22] Noor, A. E. (2024). The Current Smokeless Tobacco Trend in Bangladesh</p>. *Dental Journal*, *2*. http://dx.doi.org/10.12944/EDJ.05.02.04

[23] Pradhan, P., Jia, G., Khankari, N. K., & Zheng, W. (2024). Evaluating interactions of polygenic risk scores and NAT2 genotypes with tobacco smoking in bladder cancer risk. *International Journal of Cancer*, *154*(2), 210–216. https://doi.org/10.1002/ijc.34736

[24] Reed, O., Jubber, I., Griffin, J., Noon, A. P., Goodwin, L., Hussain, S., Cumberbatch, M. G., & Catto, J. W. F. (2020). Occupational bladder cancer: A cross section survey of previous employments, tasks and exposures matched to cancer phenotypes. *PLOS ONE*, *15*(10), e0239338. https://doi.org/10.1371/journal.pone.0239338

[25] Romero Villela, P. N., Evans, L. M., Palviainen, T., Border, R., Kaprio, J., Palmer, R. H. C., Keller, M. C., & Ehringer, M. A. (2024). Loci on chromosome 20 interact with rs16969968 to influence cigarettes per day in European ancestry individuals. *Drug and Alcohol Dependence*, *257*, 111126. https://doi.org/10.1016/j.drugalcdep.2024.111126

[26] Sumit, A. F., Das, A., Miraj, I. H., & Bhowmick, D. (2020). Association between chronic obstructive pulmonary disease (COPD) and occupational exposures: A hospital based quantitative cross-sectional study among the Bangladeshi population. *PLOS ONE*, *15*(9), e0239602. https://doi.org/10.1371/journal.pone.0239602

[27] Warnakulasuriya, S., Sunavala-Dossabhoy, G., Petti, S., & Lodi, G. (2025). Oral Cancer in Asia. *Oral Diseases*, *31*(5), 1395–1397. https://doi.org/10.1111/odi.15252

[28] Yang, P. (2023). From Womb to Tomb: In Utero Exposure to Tobacco Smoke and Adult Cancers. *Mayo Clinic Proceedings*, *98*(8), 1116–1117. https://doi.org/10.1016/j.mayocp.2023.06.008