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Review Article

Exploring the relationship between genetic polymorphisms and cancer in the Bangladeshi population

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ABSTRACT

Since the completion of the Human Genome Project two decades ago, genetic polymorphisms in human DNA have gained significant attention for their role in diseases, particularly cancer. Cancer susceptibility studies have examined Single Nucleotide Polymorphisms (SNPs) across various genes, but populations such as Bangladeshis have remained underrepresented in this field of research. This review consolidates all 54 studies on cancer-related genetic polymorphisms in the Bangladeshi population, focusing on genetic factors involved in cancer progression, and 5 more studies on treatment toxicity association. A comprehensive search of online literature databases was conducted to identify studies specifically conducted on Bangladeshis, and these studies are organized by cancer type. Various genotyping techniques, including PCR-RFLP, T-ARMS-PCR, TaqMan Assay, and Sanger sequencing, were employed to detect genetic associations, all of which are discussed. Genes most frequently studied include *TP53*, *XRCC1*, *CDH1*, *IL17A*, and *RAD51*, with the *TP53* rs1042522 polymorphism being the most investigated SNP. The review also identifies cancers such as blood, esophageal, bladder, hepatocellular and liver cancer as understudied. Importantly, SNPs in genes such as *MTHFR* and *GSTP1* were found to influence chemotherapy-related toxicities. This review provides a comprehensive overview of cancer polymorphism studies conducted on this population, highlights the current scope of research, and identifies opportunities for further exploration. It emphasizes the potential for discovering unique Bangladeshi polymorphisms, which could lead to more tailored cancer diagnosis and treatment approaches.

Introduction

Cancer has become a global health crisis and a major concern affecting the overall health of populations worldwide. It is the leading or second leading cause of death among individuals under the age of 70 in 112 out of 183 countries. According to GLOBOCAN 2020, there were 19.3 million new cancer cases and 10 million cancer-related deaths reported worldwide in 2020 (Sung et al., 2021). The global cancer burden is projected to rise by 47% by 2040 compared to 2020, with a more significant increase expected in transitioning countries than in transitioned ones (Sung et al., 2021). In South-Central Asia, Bangladesh ranks third in cancer incidence, with 156,775 new cases and 108,990 deaths reported in 2020 (Sung et al., 2021). Among men, esophageal (16.1%) and lung (11.1%) cancers are most common, whereas breast (19%) and cervical (12%) cancers dominate among women (IARC, 2023).

The etiology of cancer is multifactorial, involving interactions between genetic predispositions and environmental exposures. Genetic factors include inherited mutations or polymorphisms that affect the function or expression of genes involved in DNA

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repair, cell cycle regulation, apoptosis, metabolism, inflammation, immune response, angiogenesis, or metastasis (Kou et al., 2016). Among these, Single Nucleotide Polymorphisms (SNPs), the most common type of genetic variation in humans, account for approximately 90% of human genetic diversity. These mutations involve the substitution of a single nucleotide base pair at a specific genomic location and can be classified as either coding (affecting the amino acid sequence of proteins) or non-coding. Coding SNPs that alter protein sequences may change protein structure and function, potentially leading to reduced or lost activity. Non-coding SNPs, although they do not alter protein sequences, can influence regulatory elements such as transcription factor binding sites, splicing mechanisms, microRNA binding, and RNA expression.

Extensive research has investigated the association between SNPs and cancer. Such genetic association studies are generally divided into two categories: susceptibility studies, which examine cancer risk, and outcome studies, which explore factors influencing survival, complications, and treatment response. Most existing studies focus on a limited number of SNPs or genes, occasionally extending to multiple variants within a pathway or related biological process (Erichsen and Chanock, 2004). Although individual genes such as DNA repair enzymes, oncogenes, and tumor suppressor genes, may contain numerous SNPs, not all are clinically relevant to cancer development (Tan, 2017). Therefore, statistical measures like odds ratios (OR), confidence intervals (CI), and p-values are essential for determining the significance of specific SNPs in relation to cancer progression and treatment response. Technological advancements, especially in bioinformatics, have facilitated the study of multiple genes simultaneously (Dong et al., 2008). Nonetheless, replication and validation of findings are essential before clinical application, and such studies should be considered preliminary (Erichsen and Chanock, 2004).

In Bangladesh, case-control studies have been conducted to examine the relationship between SNPs and both cancer risk and treatment outcomes. These studies typically focus on candidate genes previously associated with various cancer types in other populations. This review summarizes findings from studies on SNPs linked to cancers of the bladder, lung, cervix, colon, breast, prostate, liver, and others in the Bangladeshi population. Only studies specifically involving Bangladeshi cohorts were included, and they have been categorized by cancer type. Statistical measures of significance have also been reported for each SNP studied.

A comprehensive discussion of cancer-related polymorphisms in the Bangladeshi population will enhance understanding of the region's genetic variability. Furthermore, this information can serve as a valuable reference for developing region-specific biomarkers and predicting treatment outcomes in cancer patients.

Method

A thorough literature search was conducted to identify studies assessing the association between single nucleotide polymorphisms (SNPs) and cancer susceptibility or treatment-related toxicity in the Bangladeshi population. Relevant articles published up to April 2025 were retrieved from PubMed and Google Scholar using search terms such as "SNP," "genetic polymorphism," "cancer," "Bangladeshi population," "cancer treatment toxicity," and "SNP cancer risk." After initial screening, duplicate studies were removed through manual cross-verification. Studies were included if they were original research articles, involved human subjects of Bangladeshi ethnicity, and reported associations between specific SNPs and either cancer risk or treatment outcomes. Studies were excluded if they were reviews, conference abstracts, purely in silico analyses, or focused on non-Bangladeshi populations. Articles lacking genotype or association data were also excluded.

Following the selection process, a total of 54 studies were included that investigated SNP associations with various cancer types, along with 5 studies focused on SNPs associated with cancer treatment toxicity. Fig. 1 presents a summary of the genes involved, highlighting both unique and overlapping genes

across different cancer types. While this review is not a formal systematic review, it takes a thematic approach to organize and interpret the existing literature.

Acute Lymphoblastic Leukemia SNP

The *ARID5B* gene plays a crucial role in epigenetic regulation through chromatin remodeling. A recent study by Jahan et al. (2025) investigated the rs10821936 (T>C) polymorphism in this gene and found that the homozygous mutant C allele (C/C) was significantly associated with an increased risk of acute lymphoblastic leukemia (ALL) in Bangladeshi children. The study reported a 1.35-fold increased risk of childhood ALL associated with the C/C genotype (95% Confidence Interval [CI]: 1.02–1.79; $p = 0.0380$).

Bladder Cancer SNPs

A case-control study investigating the association between *TP53* gene polymorphisms and bladder cancer in the Bangladeshi population analyzed SNPs at codons 72 and 248 (Hosen et al., 2015). Individuals with the Pro/Pro genotype (homozygous mutant) at codon 72 (G→C transversion) were found to have a significantly higher risk of developing bladder cancer compared to those with the Arg/Arg (wild-type) genotype (Odds Ratio [OR] = 3.02; 95% CI: 1.42–6.40; $p < 0.01$). The risk increased further among smokers carrying the Pro/Pro genotype, who had a 3.91-fold higher likelihood of developing bladder cancer (OR = 3.91; CI: 1.33–11.5; $p< 0.05$). However, no significant association was found for the Arg/Pro (heterozygous) genotype at codon 72 or for polymorphisms at codon 248 (Hosen et al., 2015).

The *TP53* gene, located on chromosome 17, regulates critical cellular functions, including cell cycle control, genomic stability, and apoptosis. Mutations in *TP53* often result in truncated or inactive proteins, impairing its tumor-suppressive roles. Additionally, mutant p53 proteins may acquire new functions through interactions with other transcription factors, potentially promoting oncogenesis (Mantovani et al., 2019; Kim and Lozano, 2018).

The *NAT1* and *NAT2* genes encode N-acetyltransferase enzymes involved in the detoxification of carcinogens. Mutations in these genes can lead to the formation of "slow acetylator" genotypes, which are less efficient in detoxifying arylamine carcinogens and may increase cancer risk (Kabir and Rehman, 2018). Specifically, *NAT2* gene mutations result in four known mutant alleles. While wild-type alleles enable faster acetylation, mutant alleles confer slower metabolic activity. A study conducted in Bangladesh found that individuals with slow acetylator genotypes had a significantly higher risk of developing bladder cancer (OR = 4.45; CI: 2.26–8.77; $p< 0.001$). These individuals were also more likely to develop high-grade (OR = 6.63; CI: 1.15–38.13; $p< 0.05$) and invasive (OR = 10.6; CI: 1.00–111.5; $p = 0.05$) bladder tumors. Cigarette smokers with slow acetylator genotypes faced an even higher risk, with a six-fold increased likelihood of developing bladder cancer (OR = 6.05; CI: 2.23–15.82) (Hosen et al., 2015).

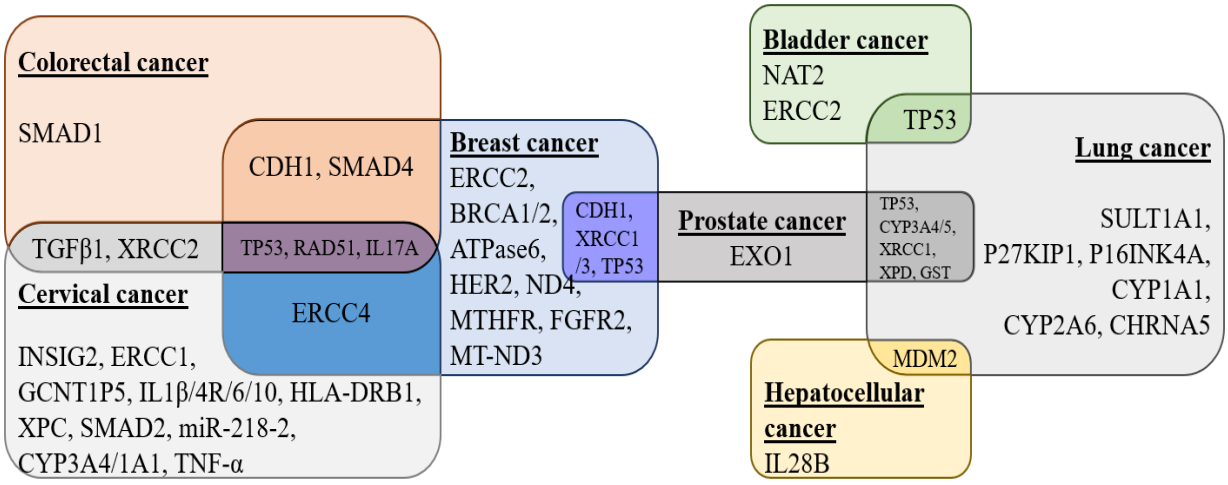

Fig. 1. All the studied genes in a Venn diagram, showing different cancer types and the individual list of genes studied in those types. Different cancer types are color-coded, and the intersections contain the gene names that have been studied in multiple cancer types.

Another study conducted on the Bangladeshi population investigated two polymorphisms in the ERCC2 gene, specifically rs13181and rs1799793, and found both single nucleotide polymorphisms (SNPs) to be significantly associated with an increased risk of bladder cancer (Islam et al., 2024). Individuals with the homozygous mutant genotype of rs13181 had more than a three-fold increased risk of developing bladder cancer (OR = 3.27; 95% CI: 1.19–8.67; $p< 0.05$). Additionally, the heterozygous genotype of rs1799793 was associated with a two-fold increased risk (OR = 2.14; 95% CI: 1.03–4.29; $p< 0.05$). Table 1 shows information on all the Bangladeshi studies for bladder cancer SNPs including gene, chromosome location and reported association.

T10400C—which caused amino acid substitutions. These mutations were found in 75% of breast cancer patients compared to 35% of healthy individuals, a statistically significant association (OR = 5.57; CI: 1.51–20.51; $p = 0.0138$) (Sultana et al., 2011). The same research group identified novel mutations in *ND3*, *ND4*, and the D-loop region of mtDNA. Mutations at positions 16290 and 16293 in the D-loop were found in 95% and 75% of patients, respectively (OR = 6.00; CI: 1.25–28.84; $p = 0.002$ and OR = 5.60; CI: 1.44–21.89; $p = 0.010$). A novel mutation at position 10316 in *ND3* was also detected in 69% of cancer patients, while absent in healthy controls

Table 1. SNPs of genes associated with risk of bladder cancer in Bangladesh.

Gene	Location in Chromosome	SNP	Sample Size (Patients/ Controls)	Reported Association	Ref.
TP53	17p13.1	rs1042522 G>C	102/140	Significant association found	Hosen et al., 2015
TP53	17p13.1	Arg248Trp C>T	102/140	No association found	Hosen et al., 2015
NAT2	8p22	rs1799929 C>T	102/140	Significant association found in M1 genotype	Kabir and Rehman, 2018
NAT2	8p22	rs1799930 G>A	102/140	Significant association found in M2 genotype	Kabir and Rehman, 2018
NAT2	8p22	rs1799931 G>A	102/140	Significant association found in M3 genotype	Kabir and Rehman, 2018
ERCC2	19q13.32	rs13181 T>G	121/130	Significant association found in homozygous mutant	Islam et al., 2024
ERCC2	19q13.32	rs1799793 G>A	121/130	Significant association found in heterozygous mutant	Islam et al., 2024

All genotyping was performed using the PCR-RFLP (Polymerase Chain Reaction-Restriction Fragment Length Polymorphism) method.

Breast Cancer SNPs

Breast cancer is the most common cancer among women in Bangladesh. Consequently, numerous studies have investigated associations between specific gene polymorphisms and breast cancer susceptibility in this population (Table 2).

Several studies have focused on mitochondrial DNA (mtDNA) polymorphisms. One such study reported two SNPs in the NADH dehydrogenase subunit 3 (*ND3*) of mitochondrial complex I—G10398A and (Sultana et al., 2012). These polymorphisms are believed to increase reactive oxygen species (ROS) production, contributing to oxidative damage and carcinogenesis (Harman, 1988; Ames and Shigenaga, 1992).

Another study found SNPs in the mitochondrial *ATPase6* gene, part of ATP synthase complex V. Two variants—A8812 and A8701G—were present in 50% (OR = 22.37; CI: 1.15–437.91; $p = 0.008$) and 56.3% (OR = 14.14; CI: 1.46–137.30; $p = 0.016$) of patients, respectively (Islam et al., 2021).

Polymorphisms in nuclear DNA repair genes have also been implicated in breast cancer risk. A study by Howlader et al. (2020) reported significant associations for *XRCC1* (Arg399Gln) and *XRCC3* (Thr241Met) polymorphisms. The Arg/Gln and Gln/Gln genotypes of *XRCC1* were associated with increased breast cancer risk (OR = 1.78; CI: 1.01–3.14; p = 0.047 and OR = 2.41; CI: 1.03–5.59; p = 0.041). Similarly, the *XRCC3* Thr/Met genotype showed a 1.86-fold increased risk (OR = 1.86; CI: 1.12–3.19; p = 0.017), while the Met/Met genotype was not significantly associated (Howlader et al., 2020). These genes are critical for single- and double-strand DNA break repair, and their mutations can impair DNA repair capacity (Shen et al., 2004).

In the Nucleotide Excision Repair (NER) pathway, *ERCC2* and *ERCC4* genes play essential roles. A study on Bangladeshi women reported that the homozygous mutant allele of *ERCC2* rs13181, like in bladder cancer, was also significantly associated with breast cancer risk (OR = 4.00; p = 0.001), while *ERCC4* rs2276466 showed no significant correlation (p> 0.05) (Sahaba et al., 2022).

Other critical genes studied include *BRCA1/2*, *RAD51*, and *HER2* (*ERBB2*), which have well-established links to breast cancer. *BRCA1/2* are tumor suppressor genes involved in DNA repair and cell cycle regulation (Hall et al., 1990). *RAD51* assists in homologous recombination during DNA double-strand repair (Richardson, 2005), while *HER2* promotes cell proliferation and is a proto-oncogene (Xie et al., 2000). A study showed that while combined *BRCA1/2* mutations were significantly associated with breast cancer (OR = 3.27; CI: 1.13–9.52; p = 0.029), individual *BRCA1* rs80357713, rs80357906, and *BRCA2* rs11571653 variants were not. However, *RAD51* rs1801320 heterozygous (GC) and combined mutant (GC+CC) genotypes were significantly associated with breast cancer (OR = 1.72; CI: 1.13–2.62; p = 0.012 and OR = 1.70; CI: 1.14–2.53; p = 0.009, respectively). Similarly, *HER2* rs1136201 in AG and AG+GG genotypes was also linked to elevated risk (OR = 1.49; CI: 1.00–2.22; p = 0.050 and OR = 1.52; CI: 1.04–2.24; p = 0.031) (Parvin et al., 2017).

In a separate study, three novel mutations in exon 11 of *BRCA1* were identified through Sanger sequencing (nucleotide positions 709 [G>A], 711 [A>G], and 852 [G>C]) in one out of 130 samples (Nishat et al., 2019).

The *TP53* gene, frequently mutated in various cancers, was also linked to breast cancer in Bangladesh. The Pro/Pro genotype at codon 72 showed a 2.52-fold increased risk (OR = 2.52; CI: 1.19–5.33; p = 0.0157) (Hossain et al., 2017). Another study confirmed similar findings for the TP53 Arg72Pro mutation (Shabnaz et al., 2016), along with *CDH1* rs16260, where Pro/Pro and A/A genotypes were associated with increased risk (OR = 1.83; p = 0.018 and OR = 1.52; p = 0.0058, respectively).

A novel association was also reported for the *SMAD4* gene, a regulator of cell proliferation and differentiation (Massagué, 2012). The rs10502913 SNP in its A/A genotype was significantly linked to increased breast cancer risk (OR = 4.96; CI: 1.99–12.33; p = 0.006), as was the combined G/A + A/A genotype (OR = 3.56; CI: 1.69–7.51; p = 0.008) (Rahman et al., 2023).

A recent study by Aziz et al. (2024) found no significant association between the *IL17A* rs3748067 polymorphism and the development of breast cancer. In contrast, the *MTHFR* gene, critical for folate metabolism and DNA methylation, was reported to be significantly associated with increased breast cancer risk in a study by Alam et al. (2024). Specifically, the rs1801131 polymorphism in its heterozygous form was linked to a 3.85-fold increased risk of breast cancer (OR=3.85; 95% CI: 2.06–7.25; p< 0.001). However, the rs1801133 variant showed no significant association.

Similarly, the *FGFR2* gene, which plays a key role in cell development regulation, was found to be significantly associated with breast cancer risk in a study on the Bangladeshi population (Jahan et al., 2023). The rs1219648 polymorphism showed strong associations with breast malignancy across multiple models, with risk increases of 2.87-fold and higher. The rs2981582variant also demonstrated a significant association with breast cancer development, while the rs2420946 polymorphism showed no significant link.

Table 2. SNPs of genes associated with risk of breast cancer in Bangladesh.

Gene	Location in Chromosome	SNP	Sample Size (Patients/Controls)	Reported Association	Ref.
$ND3	MT-DNA	G10398A	24/20	Significant association found	Sultana et al., 2011
$ND3	MT-DNA	T10400C	24/20	Significant association found	Sultana et al., 2011
$D-loop	MT-DNA	16290T-ins	24/20	Significant association found	Sultana et al., 2012
$D-loop	MT-DNA	16293A-del	24/20	Significant association found	Sultana et al., 2012
$ND3	MT-DNA	10316 A>G	24/20	Significant association found	Sultana et al., 2012
$ATPase6	MT-DNA	T96P A>C	16/12	Significant association found	Islam et al., 2021
$ATPase6	MT-DNA	T59A A>G	16/12	Significant association found	Islam et al., 2021
*XRCC1	19q13.31	rs7997821 G>A	121/133	Significant association found	Howlader et al., 2020
*XRCC3	14q32.33	rs861539 C>T	121/133	Significant association found in heterozygous mutant	Howlader et al., 2020
*ERCC2	19q13.32	rs13181 T>A	140/111	Significant association found	Sahaba et al., 2022
*ERCC4	16p13.12	rs2276466 C>A	140/111	No significant association found	Sahaba et al., 2022
*BRCA1	17q21.31	rs80357713 ->T	310/250	No significant association found	Parvin et al., 2017
*BRCA1	17q21.31	rs80357906 ->G	310/250	No significant association found	Parvin et al., 2017
*BRCA2	13q13.1	rs80357906 ->G	310/250	No significant association found	Parvin et al., 2017
*RAD51	15q15.1	rs1801320 G>C	310/250	Significant association found	Parvin et al., 2017
*HER2	17q21.1	rs1136201 A>C	310/250	Significant association found	Parvin et al., 2017
#BRCA1	17q21.31	852 G>C	65	No significant association found	Nishat et al., 2019
#BRAC1	17q21.31	711 A>G	65	No significant association found	Nishat et al., 2019
#BRAC1	17q21.31	709 G>A	65	No significant association found	Nishat et al., 2019
*TP53	17p13.1	rs1042522 G > C	125/125	Significant association found	Hossain et al., 2017
*TP53	17p13.1	rs1042522 G>C	310/250	Significant association found	Shabnaz et al., 2016
*CDH1	16q22.1	rs16260 C>A	310/250	Significant association found	Shabnaz et al., 2016
*SMAD4	18q21.2	rs10502913 G>A	70/60	Significant association found	Rahman et al., 2023
¥IL17A	6p12.2	rs3748067 G>A	156/156	No significant association found	Aziz et al., 2024
*MTHFR	1p36.6	rs1801131 A>C	202/104	Significant association found in heterozygous mutant	Alam et al., 2024
*MTHFR	1p36.6	rs1801133 C>T	202/104	No significant association found	Alam et al., 2024
*FGFR2	10q26.13	rs1219648 A>G	226/220	Significant association found	Jahan et al., 2023
*FGFR2	10q26.13	rs2981582 C>T	226/220	Significant association found	Jahan et al., 2023
*FGFR2	10q26.13	rs2420946 C>T	226/220	No significant association found	Jahan et al., 2023

Genotyping methods used were *PCR-RFLP,#Sequencing, $PCR+Sequencing and ¥T-ARMS-PCR

Hepatocellular Cancer SNPs

Interleukin-28B (*IL28B*) is involved in antiviral defense, and its polymorphisms have been associated with disease prognosis in hepatocellular carcinoma (HCC), particularly among patients infected with Hepatitis B or C viruses (HBV/HCV) (Qin et al., 2019). A study conducted in Bangladesh found that individuals carrying the T/T (homozygous mutant) or C/T (heterozygous) genotypes of *IL28B* had a significantly higher risk of developing HBV-related HCC. Carriers of the minor T allele were more likely to suffer from HCC ($p = 0.002$) (Al-Mahtab, 2016).

Another Bangladeshi study investigated the *MDM2* (T309G) polymorphism in HCC patients. The G/G genotype was associated with a significantly higher risk of HCC compared to the T/T genotype (OR = 3.6; CI: 1.64–7.80; $p< 0.01$). Individuals with either G/G or T/G genotypes also showed a two-fold increased risk of developing HCC (OR = 2.20; CI: 1.21–4.14; $p< 0.05$) (Hosen et al., 2021). *MDM2* is a key regulator of *TP53*, maintaining its cellular levels and influencing tumor suppression.

Colorectal Cancer SNPs

The *SMAD1* gene plays a central role in regulating biological functions such as cell growth and apoptosis. A study on Bangladeshi colorectal cancer (CRC) patients found significant associations with two *SMAD1* SNPs: rs11100883 and rs7661162. The rs11100883 heterozygous G/A genotype was linked to a 1.55-fold increased risk of CRC (OR = 1.55; CI: 1.09–2.20; $p = 0.014$), while the A/A genotype showed a 1.82-fold increase, though not statistically significant. For rs7661162, the A/G genotype showed a 1.78-fold increased risk (OR = 1.78; CI: 1.24–2.56; $p = 0.002$) (Karmokar et al., 2020).

Conversely, a separate study reported a protective effect for the *SMAD4* rs10502913 SNP. The G/A and A/A genotypes were associated with significantly lower CRC risk (OR = 0.24; CI: 0.12–0.45; $p< 0.001$ and OR = 0.06; CI: 0.02–0.21; $p< 0.001$, respectively). In contrast, *TGFβ1* rs1800469 was associated with a higher risk, though this result was not statistically significant ($p> 0.05$) (Sultana et al., 2022).

Another study investigated *TP53* and *CDH1* polymorphisms in CRC. The *TP53* Arg/Pro genotype at codon 72 increased CRC risk by 2.58-fold (OR = 2.58; CI: 1.77–3.77; $p< 0.05$), while the Pro/Pro genotype nearly tripled the risk (OR = 2.92; CI: 1.78–4.78; $p< 0.05$) (Rivu et al., 2017). *CDH1* rs16260 SNP also demonstrated a strong association: the heterozygous C/A genotype carried a 1.94-fold increased risk (OR = 1.94; CI: 1.34–2.81; $p< 0.05$), and the homozygous A/A genotype carried a 2.63-fold increased risk (OR = 2.63; CI: 1.15–6.01; $p< 0.05$).

Interleukin-17A (*IL17A*), important for immune defense and tissue remodeling, was also implicated in CRC risk in a Bangladeshi study. The rs10484879 SNP conferred a 2.44-fold risk in the A/C genotype ($p = 0.0008$) and a 3.27-fold risk in the A/A genotype ($p = 0.0133$). Another SNP, rs3748067, showed similar associations: the G/A genotype conferred a 2.44-fold risk ($p = 0.005$) and the A/A genotype a 2.45-fold risk ($p = 0.031$) (Islam et al., 2022), as opposed to showing no association with bladder cancer as stated before.

Genetic variations in DNA repair genes *RAD51* and *XRCC2* have also been studied. *RAD51* rs1801320 was significantly associated with CRC in the G/C genotype (OR = 1.64; CI: 1.03–2.60; $p = 0.037$), but not in the G/G genotype ($p = 0.423$). *XRCC2* rs3218536 showed significant association in the C/T genotype (OR = 1.60; CI: 1.04–2.46; $p = 0.033$), while the T/T genotype was not significantly associated ($p = 0.237$) (Hridy et al., 2020). Table 3 shows information on all the Bangladeshi studies for colorectal cancer SNPs including gene, chromosome location and reported association.

Table 3. SNPs of genes associated with risk of colorectal cancer in Bangladesh.

Gene	Location in Chromosome	SNP	Sample Size (Patients/ Controls)	Reported Association	Ref.
SMAD1	4q31.21	rs11100883 G>A	275/300	Significant association found in heterozygous mutant	Karmokar et al., 2020
SMAD1	4q31.21	rs7661162 A>G	275/300	Significant association found in heterozygous mutant	Karmokar et al., 2020
TGFβ1	19q13.2	rs1800469 C>T	167/162	No significant association found	Sultana et al., 2022
SMAD4	18q21.2	rs10502913 G>A	167/162	Significant association found	Sultana et al., 2022
TP53	17p13.1	rs1042522 G>C	288/295	Significant association found	Rivu et al., 2017
CDH1	16q22.1	rs16260 C>A	288/295	Significant association found	Rivu et al., 2017
IL17A	6p12.2	rs10484879 C>A	292/288	Significant association found	Islam et al., 2022
IL17A	6p12.2	rs3748067 G>A	292/288	Significant association found	Islam et al., 2022
RAD51	15q15.1	rs1801320 G>C	200/200	Significant association found in heterozygous mutant	Hridy et al., 2020
XRCC2	7q36.1	rs3218536 C>T	200/200	Significant association found in heterozygous mutant	Hridy et al., 2020

All genotyping was performed using the PCR-RFLP method.

Prostate Cancer SNPs

A study on the Bangladeshi population reported a significant association between the *CDH1* rs16260 polymorphism and prostate cancer. Individuals with the heterozygous C/A genotype had a 2.1-fold increased risk compared to those with the C/C (wild-type) genotype (OR = 2.10; CI: 1.17–3.78; p = 0.0135) (Imtiaz et al., 2019). The same study also investigated the K589E SNP in the *EXO1* gene, which is involved in DNA repair and genomic stability. The G/A (heterozygous) genotype showed a 2.3-fold increased risk (OR = 2.30; CI: 1.30–4.09; p = 0.0031), while the A/A (homozygous mutant) genotype exhibited a 4.85-fold increased risk (OR = 4.85; CI: 1.02–23.03; p = 0.0291).

Another study examined *CYP3A41B* (rs2740574) and *CYP3A5*3 (rs776746) gene polymorphisms, which are involved in drug metabolism and hormone regulation. The *CYP3A4**1B heterozygous (*1A/*1B) genotype was associated with a 3.52-fold increased risk (OR = 3.52; CI: 1.38–9.02; p = 0.0086), and the homozygous (*1B/*1B*) genotype showed a 3.90-fold increased risk (OR=3.90; CI: 1.26–12.05; p = 0.0183).For *CYP3A5*3, the heterozygous (*1/*3) genotype had a 5.11-fold increased risk (OR= 5.11; CI: 2.54–10.31; p= 0.0001), and the homozygous (*3/*3) genotype showed a 5.49-fold increase (OR = 5.49; CI: 2.45–12.28; p=0.0001) (Bellah et al., 2023).

Polymorphisms in DNA repair genes *XRCC1* and *XRCC3* were also examined. The *XRCC1* Arg399Gln Gln/Gln genotype was associated with a 5.7-fold increased risk of prostate cancer (OR = 5.67; CI: 1.51–21.27; p = 0.0102), while the *XRCC3* Thr241Met heterozygous Thr/Met genotype showed a 2-fold increase (OR = 2.03; CI: 1.05–3.95; p = 0.036) (Khan et al., 2019).

A separate study found that the *TP53* Arg72Pro SNP was significantly associated with prostate cancer risk. The Arg/Pro genotype conferred a 1.99-fold increased risk (OR = 1.99; CI: 1.31–3.05; p = 0.001), and the Pro/Pro genotype showed a 4.82-fold increase (OR =

4.82; CI: 2.39–9.73; $p< 0.001$) (Akter et al., 2021). The same study also confirmed a strong association with the *CDH1* rs16260 C/A genotype (OR = 2.20; CI: 1.44–3.36; $p< 0.001$) and A/A genotype (OR = 2.77; CI: 1.25–6.17; $p = 0.012$). Another study by Ahmed et al. (2024) reported that the *XRCC1* rs1799782 polymorphism was significantly associated with an increased risk of prostate cancer (OR = 5.51; 95% CI: 0.88–24.36; $p = 0.03$). In the same study, the *XPD* rs13181 variant was also investigated but did not show a significant association with prostate cancer risk.

In another study, researchers investigated the NAT2, GSTT1, and GSTM1 genes. GSTT1 and GSTM1, both members of the glutathione S-transferase family, were found to have null genotypes (homozygous deletions) that were significantly associated with an increased risk of prostate cancer. Additionally, the slow acetylator genotype of NAT2 was also linked to a higher prostate cancer risk (Nesa et al., 2023).

Table 4 shows information on all the Bangladeshi studies for prostate cancer SNPs including gene, chromosome location and reported association.

Cervical Cancer SNPs

Multiple polymorphisms have been studied in relation to cervical cancer risk in the Bangladeshi population. Table 5 shows information on all the Bangladeshi studies for cervical cancer SNPs including gene, chromosome location and reported association. A study investigated three SNPs: *INSIG2* rs6726538, *HLA-DRB1* rs9272143, and *GCNT1P5* rs7780883 (Hasan et al., 2021). The heterozygous A/T genotype of *INSIG2* was associated with a 3.30-fold increased risk (OR = 3.30; CI: 2.19–4.97; $p< 0.0001$), while the homozygous T/T genotype further elevated the risk (OR = 8.72; CI: 3.87–19.7; $p< 0.0001$). For *HLA-DRB1*, the heterozygous T/C genotype was protective (OR = 0.46; CI: 0.31–0.70; $p = 0.0004$). The *CNT1P5*

Table 4. SNPs of genes associated with risk of prostate cancer in Bangladesh.

Gene	Location in Chromosome	SNP	Sample Size (Patients/ Controls)	Reported Association	Ref.
CDH1	16q22.1	rs16260 C>A	100/100	Significant association found in heterozygous mutant	Imtiaz et al., 2019
EXO1	1q43	rs1047840 G>A	100/100	Significant association found	Imtiaz et al., 2019
CYP3A4	7q22.1	rs2740574 C>T	210/207	Significant association found	Bellah et al., 2023
CYP3A5	7q22.1	rs776746 T>C	210/207	Significant association found	Bellah et al., 2023
XRCC1	19q13.31	rs25487 T>C	100/100	Significant association found	Khan et al., 2019
XRCC3	14q32	rs861539 G>A	100/100	Significant association found	Khan et al., 2019
XRCC1	19q13.31	rs1799782 C>T	132/135	Significant association found	Ahmed et al., 2024
XPD	19q13.32	rs13181 A>C	132/135	No significant association found	Ahmed et al., 2024
TP53	17p13.1	rs1042522 G>C	210/210	Significant association found	Akter et al., 2021
CDH1	16q22.1	rs16260 C>A	210/210	Significant association found	Akter et al., 2021
NAT2	8p22	-	207/200	Significant association found	Nesa et al., 2023
GSTT1	22q11.23	-	207/200	Significant association found	Nesa et al., 2023
GSTM1	1p13.3	-	207/200	Significant association found	Nesa et al., 2023

All genotyping was performed using the PCR-RFLP method.

Table 5: SNPs of genes associated with risk of cervical cancer in Bangladesh

Gene	Location in Chromosome	SNP	Sample Size (Patients/ Controls)	Method used for Genotyping	Reported Association	Ref.
INSIG2	2q14.1-q14.2	rs6726538 A>T	234/212	T-ARMS–PCR	Significant association found	Hasan et al., 2021
HLA-DRB1	6p21.32	rs9272143 T>C	234/212	T-ARMS–PCR	Reduced risk in heterozygous mutant	Hasan et al., 2021
GCNT1P5	7q11.23	rs7780883 G>A	234/212	T-ARMS–PCR	Significant association found in homozygous mutant	Hasan et al., 2021
RAD51	15q15.1	rs1801320 G>C	255/199	PCR-RFLP	Significant association found	Ivy et al., 2021
XRCC2	7q36.1	rs3218536 G>A	255/199	PCR-RFLP	Significant association found	Ivy et al., 2021
IL10	1q32.1	rs1800872 C>A	240/204	T-ARMS–PCR	Significant association found	Datta et al., 2020
IL10	1q32.1	rs1800896 A>G	240/204	T-ARMS–PCR	Significant association found in homozygous mutant	Datta et al., 2020
IL1β	2q14.1	rs16944 A>G	252/228	T-ARMS–PCR	Significant association found in homozygous mutant	Muhammad et al., 2021
IL4R	16p12.1	rs1801275 A>G	252/228	T-ARMS–PCR	Significant association found in homozygous mutant	Muhammad et al., 2021
IL6	7p21	rs1800797 G>A	252/228	T-ARMS–PCR	Significant association found	Muhammad et al., 2021
IL6	7p21	rs1800797 G>A	126/120	PCR-RFLP	Significant association found	Shaswati et al., 2023
IL6	7p21	rs1800795 G>C	126/120	PCR-RFLP	Significant association found	Shaswati et al., 2023
ERCC1	19q13.32	rs11615 C>T	210/200	PCR-RFLP	Reduced risk in heterozygous mutant	Das et al., 2021
ERCC4	16p13.12	rs2276466 C>G	210/200	PCR-RFLP	Significant association found in heterozygous mutant	Das et al., 2021
XPC	3p25.1	rs2228000 T>C	210/200	PCR-RFLP	Reduced risk in heterozygous mutant	Das et al., 2021
XPC	3p25.1	rs2228001 C>A	210/200	PCR-RFLP	Significant association foundin heterozygous mutant	Das et al., 2021
SMAD2	18q21.1	rs4940086 T>C	132/ 98	PCR-RFLP	Significant association found in heterozygous mutant	Haque et al., 2020
TP53	17p13.1	rs1800371 C>T	134/102	PCR-RFLP	Significant association found	Apu et al., 2020
TP53	17p13.1	rs1042522G> C	134/102	PCR-RFLP	Significant association found	Apu et al., 2020
TP53	17p13.1	rs1800371 C>T	129/122	PCR-RFLP	Significant association found	Mostaid et al., 2021
TP53	17p13.1	rs1042522 G>C	129/122	PCR-RFLP	Significant association found	Mostaid et al., 2021
MIR218-2	5q34	rs11134527 G>A	256/232	ARMS-PCR	Significant association found	Nazneen et al., 2021
CYP3A4	7q22.1	rs2740574 C>T	30/30	PCR-RFLP	No significant association found	Shamim et al., 2016
CYP1A1	15q24.1	4646903 T>C	185/220	PCR-RFLP	Significant association found	Barek et al., 2023
CYP1A1	15q24.1	1048943 A>G	185/220	PCR-RFLP	No significant association found	Barek et al., 2023
TGFβ1	19q13.2	rs1800469 C>T	134/102	PCR-RFLP	No significant association found	Apu et al., 2021
TGFβ1	19q13.2	rs1800470 T>C	134/102	PCR-RFLP	No significant association found	Apu et al., 2021
TNF-α	6p21.3	rs1799724 C>T	133/126	Taqman	Significant association found	Tishe et al., 2024
TNF-α	6p21.3	rs1800629 G>A	133/126	Taqman	Significant association found	Tishe et al., 2024
IL17A	6p12.2	rs3748067 C>T	156/156	T-ARMS-PCR	Significant association found in heterozygous mutant	Aziz et al., 2024

T-ARMS-PCR stands for Tetra-Primer Amplification Refractory Mutation System PCR, Taqman stands for Taqman-based SNP Genotyping Assay.

A/A genotype was strongly associated with increased risk (OR = 5.08; CI: 2.45–10.5; $p< 0.0001$).

RAD51 rs1801320 was also found to significantly increase cervical cancer risk in both G/C (OR = 2.21; CI: 1.43–3.42; $p = 0.0004$) and C/C (OR = 4.48; CI: 1.76–11.42; $p = 0.002$) genotypes (Ivy et al., 2021). *XRCC2* rs3218536 showed similar associations: G/A genotype (OR = 2.77; CI: 1.85–4.17; $p< 0.0001$) and A/A genotype (OR = 5.86; CI: 2.08–16.50; $p = 0.001$).

IL10 gene polymorphisms rs1800872 and rs1800896 were also implicated. The C/A and A/A genotypes of rs1800872 were associated with increased risk (OR = 1.59; CI: 1.01–2.49; $p = 0.043$ and OR = 2.75; CI: 1.53–4.93; $p = 0.0007$), while for rs1800896, only the G/G genotype showed a significant risk (OR = 3.48; CI: 1.46–8.31; $p = 0.005$) (Datta et al., 2020).

A separate study explored the polymorphisms of *IL1β* (rs16944), *IL4R* (rs1801275), and *IL6* (rs1800797) genes (Muhammad et al., 2021). The G/G genotype of rs16944 (A>G) showed a significantly increased risk of developing cervical cancer (OR= 2.10; CI= 95%, 1.24–3.5; p< 0.017). The *IL4R* polymorphism in the homozygous mutant (G/G) genotype also showed an increased risk of cervical cancer (OR= 2.66; CI= 95%, 1.49–4.75; p= 0.001) compared to the A/A genotype.

Lastly, the rs1800797 G>A polymorphism of *IL6* elevated the risk of developing cervical cancer in both G/A and A/A genotypes (OR= 8.13; CI= 95%, 5.27–12.55; p < 0.0001 and OR= 9.86; CI= 95%, 2.76–35.21; p = 0.0004 respectively) (Muhammad et al., 2021). A separate study also reported that two genotypes were significantly associated with an increased risk of cervical cancer: the G/A genotype showed a 6.94-fold increased risk (OR = 6.94; 95% CI: 3.76–12.81; $p < 0.0001$), while the A/A genotype was associated with a 3.88-fold increased risk (OR=3.88; 95% CI: 1.12–13.51; $p = 0.0332$). In addition, the study examined the IL6 rs1800795 (G>C) polymorphism and found it to be significantly associated with cervical cancer risk. Specifically, the heterozygous G/C genotype was linked to an almost 3-fold increased risk, and the homozygous mutant C/C genotype was associated with a 3.5-fold increased risk (Shaswati et al., 2023).

DNA repair genes were also examined. *ERCC1* rs11615 and *XPC* rs2228000 were associated with decreased risk (OR = 0.58; $p = 0.019$ and OR = 0.61; $p = 0.025$), while *ERCC4* rs2276466 and *XPC* rs2228001 increased cervical cancer risk (OR = 4.33; $p< 0.0001$ and OR = 1.67; $p = 0.012$, respectively) (Das et al., 2021).

SMAD2 rs4940086 (T/C) was associated with a higher risk of cervical cancer (OR = 3.89; CI: 1.78–8.51; $p = 0.001$), particularly among urban residents (OR = 2.59; CI: 1.02–6.59; $p = 0.045$) (Haque et al., 2020).

TP53 codon 72 variants were consistently linked to increased risk in multiple studies. Arg/Pro and Pro/Pro genotypes showed elevated risk in both studies cited (Apu et al., 2020; Mostaid et al., 2021).

The *miR-218-2* gene SNP rs11134527 was also found to be significantly associated with cervical cancer risk. The A/G and A/A genotypes increased the risk by 2.26-fold and 3.64-fold, respectively ($p = 0.0008$ and $p< 0.0001$) (Nazneen et al., 2021).

*CYP3A4**1B polymorphism was studied to explore the association with cervical cancer in the Bangladeshi population. Still, no significant association was observed between the said polymorphism and the development of cervical cancer (Shamim et al., 2016), with the heterozygous mutant and homozygous mutant samples showing low odds ratio (OR=1.33; CI= 95%, 0.47-3.82; p= 0.5925 and OR=1.11; CI= 95%, 0.02-58.72, p=0.9596, respectively). No association with cervical cancer was found in another study either, where *TGFβ1* rs1800469 (OR= 1.083; CI= 95%, 0.612–1.915; p> 0.05 and OR= 1.216; CI= 95%, 0.829–1.785; p> 0.05 for heterozygous and homozygous mutant respectively) and rs1800470 (OR= 1.309; CI= 95%, 0.742–2.310; p> 0.05 and OR= 1.316; CI= 95%, 0.888–1.949; p> 0.05 for heterozygous and homozygous mutant respectively) polymorphisms were studied (Apu et al., 2021). A separate study suggested a possible correlation between *CYP1A1*

polymorphisms rs4646903 (T>C) and rs1048943 (A>G) and the risk of developing cervical cancer. The researchers found that the rs4646903 variant was significantly associated with cervical cancer across all genetic models, with at least a 2-fold increase in risk. In contrast, the rs1048943 polymorphism showed no significant association (Barek et al., 2023).

A research group investigated Tumor Necrosis Factor-alpha (*TNF-α*) gene polymorphisms and found significant associations with increased susceptibility to cervical cancer. The rs1799724 variant was associated with a 3.26-fold increased risk (OR = 3.26; 95% CI: 1.15–9.28; *p*=0.027). Similarly, the rs1800629 polymorphism showed a 2.85-fold increased risk in individuals with the heterozygous genotype (OR=2.85; 95% CI: 1.20–6.74; *p* = 0.017) and a 4.55-fold increased risk in those with the homozygous mutant genotype (OR = 4.55; 95% CI: 1.24–16.60; *p* = 0.022) (Tishe et al., 2024).

The *IL17A* rs3748067 polymorphism, similar to its association with colorectal cancer, was found to be significantly associated with the development of cervical cancer in the heterozygous model (C/T; OR = 1.79; *p* = 0.021) in the same study. However, no such association was observed for the breast cancer development (Aziz et al., 2024).

Lung Cancer SNPs

Several gene polymorphisms have been studied in relation to lung cancer risk in the Bangladeshi population (Table 6).

A study investigated the *SULT1A1* (rs9282861) and *XRCC1* (rs25487) gene polymorphisms. *SULT1A1* encodes an enzyme involved in the metabolism of various compounds, while *XRCC1* contributes to base excision and single-strand DNA repair. Both the heterozygous Arg/His and homozygous His/His genotypes of *SULT1A1* were significantly associated with increased lung cancer risk (OR = 5.06; CI: 3.05–8.41; *p*< 0.05 and OR = 3.88; CI: 2.20–6.82; *p*< 0.05). Similarly, the *XRCC1* Arg/Gln and Gln/Gln genotypes were also linked to increased risk (OR = 4.57; CI: 2.79–7.46; *p*< 0.05 and OR = 4.99; CI: 2.66–9.36; *p*< 0.05) (Tasnim et al., 2017).

Cell cycle regulatory genes *P27KIP1* and *P16INK4A* were also associated with lung cancer risk. The *P27KIP1* Val109Gly polymorphism conferred a 3.38-fold and 7.09-fold increased risk in the heterozygous Val/Gly and homozygous Gly/Gly genotypes, respectively (OR = 3.38; CI: 1.64–6.96; *p* = 0.0010 and OR = 7.09; CI: 2.27–22.14; *p* = 0.0007). The *P16INK4A* Ala148Thr polymorphism was also linked to higher risk in both heterozygous (OR = 3.86; CI: 1.45–10.23; *p* = 0.0067) and homozygous mutant genotypes (OR = 3.86; CI: 1.19–12.48; *p* = 0.0242) (Zilani et al., 2018).

Polymorphisms in *GST* genes were also studied. While *GSTM1* and *GSTT1* variants were not significantly associated with lung cancer, *GSTP1* rs1695 was. The A/G and G/G genotypes showed a 3.56-fold (CI: 1.70–7.46; *p* = 0.001) and 6.57-fold (CI: 1.28–33.81; *p* = 0.024) increased risk, respectively (Nasir Uddin et al., 2014). Both non-significant *GST* variants from the previous study were further investigated by another research group (Nairuz and Kabir, 2024). The study revealed that the *GSTM1* null genotype (homozygous deletion) was significantly associated with a 1.6-fold increased risk of lung cancer (95% CI: 1.01–2.52; *p* = 0.0491). In contrast, the *GSTT1* null genotype showed no significant association.

The tumor suppressor gene *TP53* also showed strong associations. Codon 72 Arg/Pro and Pro/Pro genotypes were linked to significantly increased risk (OR = 2.51; CI: 1.38–4.82; *p* = 0.00232 and OR = 4.62; CI: 2.31–9.52; *p* = 0.00001) (Mostaid et al., 2014). Another study confirmed increased risk for the Pro/Pro genotype (OR = 3.00; CI: 1.1–8.4; *p* = 0.03) (Chowdhury et al., 2015). However, a third study did not find a significant association with *TP53* but reported a significant link between the *XPD* rs13181 polymorphism and lung cancer. The Gln/Gln genotype increased risk 3.58-fold compared to the Lys/Lys genotype (OR = 3.58; CI: 1.58–8.09; *p* = 0.002) (Nairuz et al., 2020).

Table 6. SNPs of genes associated with risk of lung cancer in Bangladesh.

Gene	Location in Chromosome	SNP	Sample Size (Patients/ Controls)	Reported Association	Ref.
SULT1A1	16p11.2	rs9282861G>A	202/242	Significant association found	Tasnim et al., 2017
XRCC1	19q13.31	rs25487 G>A	202 /242	Significant association found	Tasnim et al., 2017
P27KIP1	12p13.1	V109G T>G	100/100	Significant association found	Zilani et al., 2018
P16INK4A	9p21.3	A148T G>A	100/100	Significant association found	Zilani et al., 2018
GSTM1	1p13.3	-	106 /116	No significant association found	Nasir Uddin et al., 2014
GSTP1	11q13.2	rs1695 A>G	106 /116	Significant association found	Nasir Uddin et al., 2014
GSTT1	22q11.23	-	106/ 116	No significant association found	Nasir Uddin et al., 2014
*GSTM1	1p13.3	-	180/200	Significant association found	Nairuz and Kabir, 2024
*GSTT1	22q11.23	-	180/200	No significant association found	Nairuz and Kabir, 2024
TP53	17p13.1	rs1800371 C>T	106/116	No significant association found	Mostaid et al., 2014
TP53	17p13.1	rs1042522 G>C	106/116	Significant association found	Mostaid et al., 2014
TP53	17p13.1	rs1042522 G>C	50/50	Significant association found in homozygous mutant	Chowdhury et al., 2015
TP53	17p13.1	rs1042522 G>C	180/200	No significant association found	Nairuz et al., 2020
XPD	19q13.32	rs13181 T>G	180/200	Significant association found in homozygous mutant	Nairuz et al., 2020
CYP3A4	7q22.1	rs2740574 C>T	106/116	No significant association found	Islam et al., 2014
CYP3A5	7q22.1	rs776746 A>G	106/116	No significant association found	Islam et al., 2014
CYP3A5	7q22.1	rs776746 A>G	106/116	No significant association found	Islam et al., 2014
CYP1A1	15q24.1	rs4646903 T>C	106/116	Significant association found	Islam et al., 2013
CYP1A1	15q24.1	rs1048943 A>G	106/116	Significant association found in heterozygous mutant	Islam et al., 2013
CYP2A6	19q13.2	CYP2A6*4	106/110	Reduced risk	Islam et al., 2013
CHRNA5	15q25.1	rs16969968 G>A	106/110	No significant association found	Islam et al., 2013
MDM2	12q15	rs2279744 T>G	126/133	Significant association found	Al Reza et al., 2020a
MDM2	12q15	rs117039649 G>C	126/133	Reduced risk	Al Reza et al., 2020b

All genotyping was performed using the PCR-RFLP method except *Nairuz and Kabir, 2024* where Allele-specific PCR was performed.

A study on *CYP3A4* and *CYP3A5* polymorphisms found that individuals with at least one *CYP3A4**1B allele had a 3.35-fold increased risk of lung cancer, though the association was not statistically significant (p = 0.271). No *CYP3A5* mutations were observed in cases (Islam et al., 2014).

Another study examined polymorphisms in *CYP1A1*, *CYP2A6*, and *CHRNA5* genes. *CYP1A1* rs4646903 polymorphism, mentioned previously in cervical cancer, all cancer SNP studies included in this reviewwas presented in Fig. 2.

Polymorphisms in *MDM2* were also studied. The rs2279744 T/G and G/G genotypes were significantly associated with increased lung cancer risk (OR = 1.93; CI: 1.12–3.33; p = 0.017 and OR = 2.84; CI: 1.27–6.34; p = 0.011) (Al Reza et al., 2020a). Conversely, the rs117039649 G/C and C/C genotypes were associated with reduced risk (OR = 0.31; CI: 0.15–0.63; p = 0.001 and OR = 0.18; CI: 0.04–0.90; p =

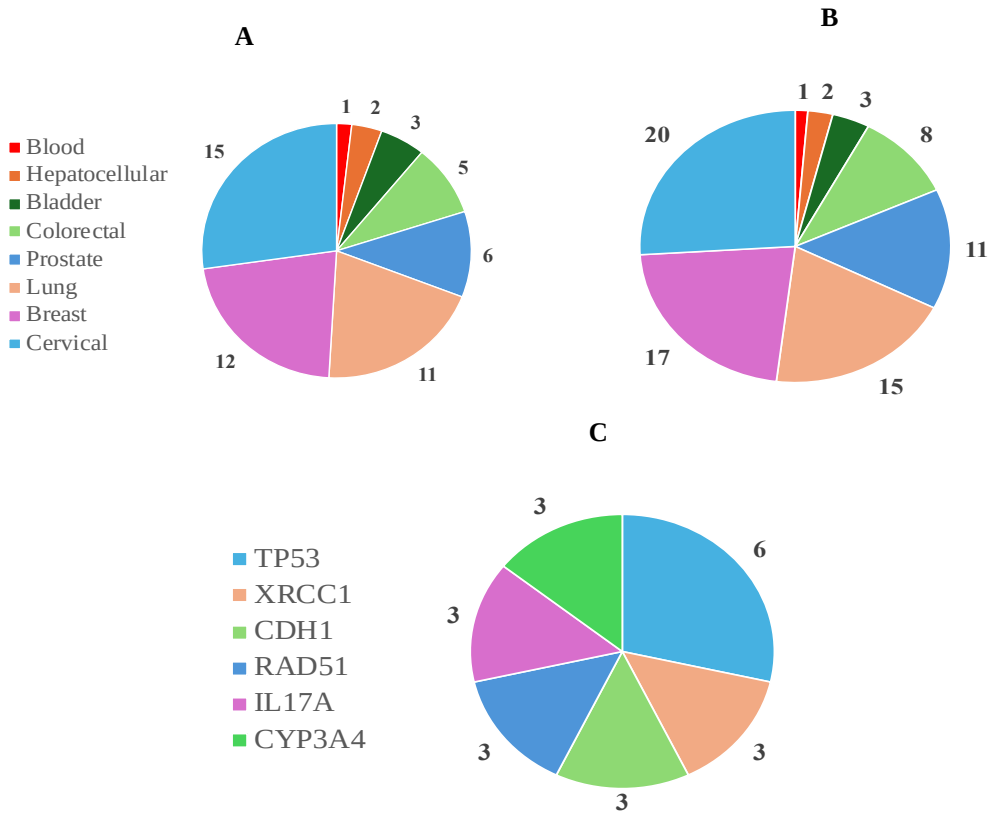

Fig. 2. Overview of all cancer SNP studies included in this review. (A) Number of studies conducted for each cancer type, (B) Number of genes investigated across different cancer types. (C) Top six frequently studied genes across all included studies.

significantly increased lung cancer risk in both T/C (OR = 1.79; CI: 1.01–3.19; p = 0.046) and C/C (OR = 2.85; CI: 1.12–7.30; p = 0.029) genotypes. The rs1048943 SNP in a dominant model (A/G + G/G) also increased risk (OR = 2.21; CI: 1.26–3.89; p = 0.006). In contrast, *CYP2A6**4 was associated with reduced risk (OR = 0.40; CI: 0.17–0.92; p = 0.030). No association was found with *CHRNA5* rs16969968 (Islam et al., 2013). Overview of

0.037) (Al Reza et al., 2020b). *MDM2* negatively regulates *TP53* and helps maintain its cellular balance.

SNPs Affecting Cancer Therapy

Table 7 shows information on all the Bangladeshi studies for cancer treatment toxicity based on SNPs including gene, chromosome location, cancer type and reported association.

Table 7. List of all gene SNPs studied for treatment toxicity in Bangladeshi cancer patients.

Gene	Location in Chromosome	SNP	Sample Size (Patients/ Controls)	Method used for Genotyping	Cancer type	Reported Association	Ref.
DPYD	1p21. 3	rs3918290 G>A	161	PCR-RFLP	Colorectal cancer	Grade 3 and 4 toxicities of 5-FU	Nahid et al., 2018
MTHFR	1p36.6	rs1801133 G>A	161	PCR-RFLP	Colorectal cancer	Increased 5-FU response	Nahid et al., 2018
XPD	19q13.32	rs13181 T>G	180	PCR-RFLP	Lung cancer	No association found	Nairuz et al., 2021
TP53	17p13.1	rs1042522 G>C	180	PCR-RFLP	Lung cancer	No association found	Nairuz et al., 2021
GSTP1	11q13.2	rs1695 A>G	285	PCR-RFLP	Non-small cell lung cancer	Reduced toxicity	Bushra et al., 2020
GSTP1	11q13.2	rs1695 A>G	285	PCR-RFLP	Non-small cell lung cancer	Increased response to platinum-based chemotherapy	Bushra et al., 2020
XRCC1	19q13.31	rs25487 T>C	285	PCR-RFLP	Non-small cell lung cancer	Grade 3 and 4 toxicities	Bushra et al., 2020
XPC	3p25.1	rs2228001 G>T	285	PCR-RFLP	Non-small cell lung cancer	Neutropenia of grade 3 and 4	Bushra et al., 2020
ERCC1	19q13.32	rs11615 A>G	285	PCR-RFLP	Non-small cell lung cancer	No association found	Bushra et al., 2020
TPMT	6p22. 3	rs1142345 A>G	75/75	TaqMan	Acute Lymphoblastic Leukemia	No association found	Zaman et al., 2019
TPMT	6p22. 3	rs1800462 G>C	75/75	TaqMan	Acute Lymphoblastic Leukemia	No association found	Zaman et al., 2019
TPMT	6p22. 3	rs1800460 G>A	75/75	TaqMan	Acute Lymphoblastic Leukemia	No association found	Zaman et al., 2019
ITPA	20p13	rs1127354 C>A	75/75	TaqMan	Acute Lymphoblastic Leukemia	6 times higher chance of absolute neutropenia	Zaman et al., 2019
MTHFR	1p36.3	rs1801133 C>T	160	PCR-RFLP	Acute Lymphoblastic Leukemia	Mucositis and diarrhea by MTX	Zahra et al., 2020
MTHFR	1p36.3	rs1801131 A>C	160	PCR-RFLP	Acute Lymphoblastic Leukemia	Mucositis and diarrhea by MTX	Zahra et al., 2020

Taqman stands for Taqman-based SNP Genotyping Assay.

Gene polymorphisms, translocations, and other genomic aberrations have been shown to contribute to the development of resistance against anticancer drugs (Aschauer and Muller, 2016; Wang, 2018). Therefore, studying SNPs as potential biomarkers for predicting drug toxicity and informing treatment strategies is critical for enhancing the efficacy of cancer therapies. Several studies have investigated the effects of SNPs on cancer therapy in the Bangladeshi population. Polymorphisms in *DPYD2A* and *MTHFR* C677T have been linked to drug toxicity and increased response in colorectal cancer patients. The *DPYD2A* polymorphism was identified as a predictive factor for grade 3 and 4 toxicities from 5-fluorouracil (5-FU, $p = 0.0023$), while the *MTHFR* C677T polymorphism significantly enhanced the 5-FU response ($p = 0.006$) (Nahid et al., 2018).

A separate study explored the role of *TP53* and *XPD* polymorphisms in lung cancer patients and their potential influence on chemotherapy toxicity (Nairuz et al., 2021). No significant association was found between *TP53* codon 72 and *XPD* codon 751 polymorphisms and platinum-based chemotherapy toxicity ($p > 0.05$). However, non-small cell lung cancer patients with the *XRCC1* rs25487 mutation in the A/G genotype experienced grade 3 and 4 toxicities, such as anemia and leukopenia. In contrast, the *XPC* rs2228001 SNP in A/C and A/C+C/C genotypes were associated with neutropenia in grades 3 and 4 (Bushra et al., 2020). The *GSTP1* rs1695 polymorphism in the G/G genotype resulted in reduced toxicity, while the same SNP in A/G and A/G+G/G genotypes showed an increased response to platinum-based chemotherapy. Similar findings were observed for *XRCC1* rs25487 SNP in A/G and A/A+A/G genotypes, though the *ERCC1* rs11615 mutation had no significant effect (Bushra et al., 2020).

A study on children with Acute Lymphoblastic Leukemia (ALL) found that the *ITP* rs1127345 mutation significantly increased the likelihood of neutropenia (6-fold) and hyperbilirubinemia (3-fold) during chemotherapy. Meanwhile, *TPMT* polymorphisms were not significantly associated with cancer treatment toxicity (Zaman et al., 2019). Another study in ALL patients investigating Methotrexate (MTX) toxicity revealed that *MTHFR* C677T and A1298C polymorphisms were significantly linked to mucositis and diarrhea caused by MTX treatment ($p < 0.05$) (Zahra et al., 2020).

Conclusion

Single nucleotide polymorphisms (SNPs) are the most common type of mutation in the human genome. Various SNPs have been extensively studied for their association with different types of cancer. This review has summarized all SNP studies conducted on Bangladeshi cancer patients. Notably, the absence of studies on esophageal cancer patients was observed. Given the increasing incidence of esophageal cancer in Bangladesh, further research on this group is essential. Additionally, lack of SNP studies for blood, bladder, and hepatocellular cancer was noticed. This review will be beneficial for clinicians and research groups working on cancer SNP studies in this particular population. It will also support the development of biomarker panels and personalized medicines for cancer patients. However, it is important to set realistic expectations for the findings of such studies, and a pathway-based genotyping approach should be pursued. SNPs in genes within the same biological pathway are likely to have a more synergistic or additive effect, making those prime candidates for extensive population studies.

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Conflict of Interest

The authors declare no conflicts of interest.

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