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Review

J Clin Med Res. 2025;17(7):357-364

Association of ABO and Rhesus Blood Groups With Oral Cancers

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Abstract

The ABO and Rhesus blood systems have been studied extensively in relation to various diseases, including cancers. Recent evidence suggests that ABO blood group may influence susceptibility to oral potentially malignant disorders (OPMDs) and oral cancers, particularly oral squamous cell carcinoma (OSCC). The current review explores the association of ABO and Rhesus blood groups with oral cancers, summarizing epidemiological, molecular, and immunological studies to elucidate potential mechanisms underlying this relationship.

Keywords: ABO blood group; Oral cancers; Oral potentially malignant disorders; Rhesus blood groups

Introduction

Oral cancer is a significant global health concern, accounting for a considerable number of cancer-related morbidity and mortality. While early detection improves survival outcomes, addressing risk factors such as tobacco use, alcohol consumption, and viral infections is essential for effective prevention and control. However, genetic and hereditary predispositions, including ABO and Rhesus (Rh) blood groups, have emerged as potential factors influencing susceptibility and prognosis of oral potentially malignant disorders (OPMDs) and oral cancers. This review provides an up-to-date synthesis of the correlation between ABO/Rh blood group phenotypes and oral malignancies, integrating epidemiological, molecular, and immunological studies to elucidate the potential biological mechanisms underlying this association.

Manuscript submitted May 12, 2025, accepted July 9, 2025
Published online July 31, 2025

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doi: https://doi.org/10.14740/jocmr6276

The ABO Blood Group System and Its Link to Diseases

The ABO blood group antigens, in addition to their expression on erythrocytes, are also prominently expressed on various human cell types, including sensory neurons, platelets, and vascular endothelial cells [1]. Erythrocyte cell surface antigens play a crucial role in maintaining membrane structural integrity, facilitating cell motility, mediating tissue differentiation, participating in inflammatory responses, transporting molecules across the membranes, and enabling cell adhesion [2-4]. The ABO blood group system was first identified by Karl Landsteinner in 1900 [5]. This system comprises three antigens (A, B, and H) and categorizes individuals into four major groups based on the presence or absence of these antigens on the surface of erythrocytes: A (presence of agglutinogen A), B (presence of agglutinogen B), AB (presence of both A and B agglutinogens), and O (absence of both A and B agglutinogens) [5, 6]. The *ABO* gene is located on chromosome 9, specifically at locus q34.1 to q34.2, and encodes glycosyltransferase responsible for antigen synthesis [7, 8]. These antigens are oligosaccharides expressed on the extracellular surface of the erythrocyte membrane. The A antigen is characterized by the presence of N-acetylgalactosamine (GalNAc), while the B antigen is defined by galactose (Gal) [7, 8]. These sugars are enzymatically transferred to a precursor structure, the H antigen, under the regulation of the *ABO* gene. In epithelial tissues, ABH antigens are carbohydrate-based and exhibit highly regulated expression patterns associated with epithelial differentiation and cell maturation [9]. Following the ABO system, the Rh blood group system is the second most clinically significant. The Rh system is defined by the presence or absence of highly immunogenic D antigen on the erythrocyte membrane. Individuals expressing the D antigen are classified as Rh-positive, whereas those lacking this antigen are categorized as Rh-negative [5].

While people possess the same ABO system, the distribution of specific types varies across different geographic regions and ethnic populations due to genetic diversity and evolutionary factors. Scientific research focuses on the relationship between human genetics and disease, particularly the role of blood group antigens in malignancies [10]. Blood group antigens, expressed on erythrocytes and epithelial cells, have been implicated in cancer development, as most human cancers originate from epithelial cells [11, 12]. The first association between ABO blood groups and malignancies was reported in

1953, linking blood group A with an increased risk of stomach cancer [13, 14]. Subsequently, multiple studies have examined the relationship between ABO antigens and various cancers, including gastric, breast, oral, skin, renal, esophagus, cardiac, lung, laryngeal, salivary gland, gynecologic, colorectal, pancreatic, bone, urinary bladder, testicular, melanoma, and prostate cancer [15]. Proposed mechanisms underlying this association include inflammation, immune response to malignant cells, intercellular adhesion, surface membrane signaling, and downregulation of glycosyltransferase involved in the biosynthesis of A and B antigens [2, 16, 17]. Thus, loss or altered expression of ABO blood group antigens in tumor cells can enhance cellular motility or facilitate the interaction with endothelial cells, promoting metastasis [18, 19]. Additionally, the accumulation of precursor antigens due to deficiency of A or B epitopes is associated with an increased risk of malignancies [20]. Despite substantial evidence linking ABO antigens to cancer susceptibility and progression, inconsistencies exist due to sample size limitations and regional variations in ABO frequencies [21, 22].

Oral Cancer

Oral cancer is a malignant condition characterized by abnormal cell proliferation capable of local tissue invasion and distant metastasis. It is a significant global health burden, accounting for about 5% of all human cancers, with a notable higher prevalence in developing countries, particularly South Asia [23, 24]. Globally, it ranks sixth among all tumors, being third most prevalent in developing countries and eighth in developed ones [25, 26]. Around 270,000 new cases are diagnosed annually, with a worldwide mortality rate of 2.9 per 100,000 [24, 27]. The disease primarily affects individuals over 50 years of age, with the highest prevalence in the 50 - 59 age group [28-31]. However, some studies indicate a rise in younger populations, likely due to evolving risk factor exposures such as tobacco consumption which leads to structural changes in the mucosa [27]. Males reveal a higher incidence than females, with a global male-to-female ratio ranging from 2:1 to 8:1 [7, 29, 32-34]. This gender disparity is attributed to increased exposure to tobacco and alcohol among men.

Oral cancer typically refers to carcinoma originating in the oral mucosal, arising from either epithelial or connective tissue. The malignancy typically originates in the buccal mucosa, tongue, and palate, often linked to chronic irritants like tobacco or betel quid [35]. It predominantly manifests as oral squamous cell carcinomas (OSCCs), accounting for 92-95% of cases [36]. OSCCs arise from the squamous epithelium, in about 90% of cases, often due to multifactorial etiologies that disrupt cellular growth regulation [37, 38]. Clinically, it is associated with the presence of leukoplakia, erythroplakia, ulcerative lesions, or exophytic masses [37, 38]. A two-step model of carcinogenesis in the oral mucosa is identified, including premalignant lesions and conditions that precede invasive carcinoma, collectively referred to as OPMDs [36]. OPMDs encompass a spectrum of conditions marked by increased cancer risk, such as leukoplakia, erythroplakia, dysplasia, oral hypoplasia, and oral submucous fibrosis (OSMF) [36, 39, 40]. Notably, OPMDs show varying rates of malignant transformations, ranging from 0.6% to 36% [41, 42], emphasizing the critical need for regular screening and intervention strategies.

Oral cancer arises from a complex interplay of genetic, environmental, and lifestyle factors [43, 44]. Tobacco and alcohol consumption are primary contributors, accounting for over 90% of cases [43]. The risk of developing oral cancer rises proportionally with duration, quantity, and frequency of tobacco and alcohol consumption [43, 44]. Tobacco-specific carcinogens, including polycyclic aromatic hydrocarbons, nitrosoproline, and polonium, and alcohol-induced mucosal damage, act synergistically, significantly elevating cancer risk [29, 44]. The use of smokeless tobacco products, such as betel quid and areca nut chewing, common in South Asia [45], further increases susceptibility by generating reactive oxygen species and carcinogenic nitrosamines [44, 46]. Other risk factors include microbe infections (e.g., human papillomavirus (HPV) and *Candida albicans*), chronic irritation, dietary deficiencies, poor oral hygiene, and socioeconomic status [29, 44, 47]. On the other hand, around 5% to 10% of cases are totally attributed to genetic factors [43, 48]. The ABO blood system has been proposed as a genetic determinant potentially influencing the risk of various types of cancers [16, 39, 43]. While ABO blood group is determined at birth, oral cancer develops much later and arises from a complex interplay of genetic predispositions and environmental factors. Several studies have suggested that ABO blood groups are not a direct cause for oral cancers, but they might be associated with cancer susceptibility or progression [16, 39, 43]. Additionally, genetic research continues to uncover insights into susceptibility and disease progression, highlighting the role of genetic factors in oral cancer development.

Epidemiological Evidence Linking ABO and Rh Blood Groups to Oral Cancers

Several epidemiological studies have investigated the relationship between ABO and Rh blood groups and the risk of oral cancer and OPMDs. The current review included articles published over the last decade, including case-control, cross-sectional, cohort, and meta-analysis studies that investigated the objectives of the present study. Among 26 published articles (Table 1) [10, 28, 29, 39, 47, 49-69], a higher proportion of oral cancer patients and OPMDs were found to belong to blood type A (14 studies) [28, 29, 39, 49-59], followed by blood type B (seven studies) [47, 60-65], and blood type AB (two studies) [66, 67]. Blood group O showed the lowest association with both oral cancer and OPMDs [28, 66-68]. Conversely, some articles reported no correlation between ABO blood groups and oral cancers [10, 69]. Few studies about the correlation between Rh factors and oral malignancies were available. There was a general consensus among Rh factor studies, with most indicating no significant correlation between the Rh factor and OPMDs or oral cancers, although a positive trend was noted in most patients with oral malignancies (Table 1) [29, 50, 51, 62, 65]. Together, these findings suggest that blood type A may

Table 1. Epidemiological Evidence Linking ABO and Rhesus (Rh) Blood Groups to Oral Cancers

Authors, year, country	Study design	Disease	Patients/controls	Main results
Panchbhai et al, 2024 [49], India	Cross-sectional	Oral squamous carcinomas (OSCC)	35 patients	Blood type A was the most prevalent in patients diagnosed with OSCCs. The Rh factor was universally positive among all patients.
Pokala et al, 2024 [60], India	Case-control	Oral cancer, and oral potentially malignant disorder (OPMD)	120/120	Compared to individuals of other blood groups, individuals with blood type B showed a 1.24 times higher risk of acquiring oral precancerous and cancerous lesions.
Qudrath et al, 2023, Bangladesh [50]	Cohort study	Oral cancer	110 patients	Most patients belonged to blood type A (45%), then type B (23%), type O (20%), and type AB (12%). There was no observed correlation between the Rh factor and oral cancer.
Pal et al, 2023, India [39]	Cohort study	Oral cancer	600 patients	Individuals with blood type A positive (A+) demonstrated a significantly higher predisposition to developing oral cancer.
Mahalakshmi et al, 2022, India [61]	Cross-sectional	OPMD	55 patients	Individuals with blood type B positive (B+) were 1.46 times more likely to develop OPMD.
Ramamoorthy et al, 2022, India [47]	Cross-sectional	Oral cancer	Not reported	Although a higher proportion of oral cancer patients belonged to blood type B (46%), the association did not reach statistical significance.
Rezvaninejad et al, 2021, Iran [51]	Case-control	Oral cancer	64/60	Compared to the control group, the blood group A in patients with oral cancer was significantly higher in patients. There was no significant difference in Rh frequency between patients and the control group.
Ashwinirani et al, 2021, India [29]	Cross-sectional	Oral cancer	63 patients	Blood group A constituted 50% of cases. There was no significant relationship between the Rh factor and oral cancer.
Singh et al, 2021, India [28]	Meta-analysis case-control	Oral cancer OPMD	2,056/26,388	Blood group A demonstrated significant associations with both oral cancer and OPMD ($P = 0.04$). Blood group O showed a significant protective effect against oral cancer.
Verma et al, 2021, India [52]	cross-sectional	Oral cancer	73 patients	Individuals with blood type A positive (A+) showed a 3.22-fold higher risk of developing OSCC compared to those with other blood types.
Gaurav et al, 2021, India [53]	Case-control	Oral cancer OPMD	100/100	There was a significant correlation between the blood group A and both OPMD and oral cancer groups.
Shishodia et al, 2019, India [54]	Case-control	Oral cancer OPMD	105/7027	Blood group A was significantly associated with increased susceptibility to OSCC. However, there was no significant relationship between ABO blood group and OPMD.
Gupta et al, 2019, India [66]	Case-control	Oral cancer	76/90	Blood group AB showed a significantly higher frequency, while blood group O had a significantly lower frequency patients compared to controls.
Singh et al, 2019, India [55]	Case-control	Oral cancer	27/250	There was a significant correlation between blood group A and oral cancer compared to other blood groups.
Jalili et al, 2018, Iran [67]	Case-control	OSCC	133/2,000	Blood group AB showed a significantly higher frequency in patients with OSCC compared to healthy controls, while blood group O showed the lowest frequency in OSCC patients.
Poornima et al, 2018, India [62]	Case-control	OPMDs OSCC	70/30	Blood group B showed a significantly higher frequency in both OPMDs and OSCC than control group, with no significant difference observed in Rh factor.
Anjum et al, 2017, India [56]	Cohort study	OPMDs	50 patients	Cases with blood group A positive (36%) were more associated with the development of OPMDs, followed by those with blood group B positive (28%).
Mehrotra et al, 2017, India [63]	Cross-sectional	Oral submucous fibrosis (OSMF)	50/50	Individuals with blood group B had a 1.32-fold higher tendency to develop OSMF than other groups.

Table 1. Epidemiological Evidence Linking ABO and Rhesus (Rh) Blood Groups to Oral Cancers - *(continued)*

Authors, year, country	Study design	Disease	Patients/controls	Main results
Ramesh et al, 2017, India [64]	Case-control	Oral cancer	100/50	Individuals with blood group B positive were found to be at an increased risk of developing OSCC compared to people with other blood groups.
Kumari et al, 2017, India [57]	Case-control	Oral cancer	300/800	Individuals with blood group A showed a 1.51-fold higher risk of developing oral cancers when compared to those with other blood groups.
Saxena et al, 2016, India [58]	Cross-sectional	Oral cancer	171 patients	Blood group A had a 6.54-fold higher association with oral cancer compared to blood group O, B and AB.
Reddy et al, 2016, India [10]	Cross-sectional	OSMF	164/180	There was no significant correlation between OSMF and ABO blood group.
Zhang et al, 2016, China [68]	Case-control	Oral cancer	3,832/24,912	Individuals with type O blood had a significantly lower proportion of oral cancer than that of controls.
Nikam et al, 2015, India [65]	Case-control	OSMF	50/50	Individuals with blood group B had higher risk of developing OSMF compared to other groups. There was no correlation between the Rh factor and OSMF.
Rai et al, 2015, India [69]	Cross-sectional	OPMDs	45/45	There was no statistically significant correlation between ABO blood groups and OPMDs.
Trupti et al, 2015, India [59]	Case-control	OSCC OSMF	60/30	Blood group A conferred an approximately 60-70% increased risk of developing OSCC compared to people with other blood groups. Individuals with blood group A were at 3.98 times greater risk of developing OSMF.

be associated with a higher prevalence of oral cancer and OPMDs, while blood group O appears to have a protective effect. Geographical and ethnic variations in study populations may influence the observed association, necessitating the need for further large-scale studies.

Mechanisms Linking ABO Blood Groups to Oral Cancers

The biological mechanisms underlying the association between ABO/Rh blood groups and the development of oral cancers are not entirely understood. However, some assumptions have been introduced to explain this association such as molecular mechanisms, immune responses, cell adhesion alterations, and inflammatory pathways.

ABO blood group antigens are complex carbohydrate structures formed from the precursor H antigen [70]. Since the protective role of H antigen, particularly in individuals with O blood groups, has been introduced, altered expression of A or B antigens has been implicated in oral carcinogenesis [70-72]. Immunohistochemical studies have shown significant loss of A or B antigens in oral cancers, correlating with reduced transferase levels, which is responsible for the biosynthesis of A and B antigens [17, 73]. Early in carcinoma development, A or B antigens may still be expressed, aiding the survival of genetically altered cells and potentially evading immune detection [74]. This may account for the slightly increased prevalence of various types of carcinomas among individuals with blood groups A and B. However, as tumors progress, downregulation of glycosyltransferase and linkage disequilibrium linking ABO genes to other genes lead to antigen loss or alteration, as genetic modifications within the ABO locus are commonly detected across multiple cancer types [17, 29]. This dysregulated expression may alter the epithelial cell surface and facilitate tumor progression and metastasis [7, 8, 17, 29, 75]. Contrasting results regarding blood groups A, B, and AB are noted across published papers, with some suggesting blood group A as more predisposed to oral cancers, and others highlighting blood group B or AB as more prevalent in certain populations [47, 60-62, 66, 67].

Blood group A individuals have shown an increased susceptibility to oral cancer and other carcinomas, which may be attributed to immunological interaction involving blood group antigens. Cancerous cells often express Forssman and Thomsen-Friedenreich (T) antigens, which share structural similarities with the A antigen, including the terminal sugar (N-acetylgalactosamine) [76]. Since individuals with blood groups A and AB do not possess anti-A antibodies, their immune system may fail to recognize and attack these precancerous and malignant cells effectively, facilitating tumor progression [16, 77, 78]. Conversely, blood group O individuals, possessing anti-A antibodies, may have a protective advantage against these cancers by targeting cells expressing A-like antigens.

Furthermore, interactions between blood group carbohydrates and related lectins play a crucial role in metastatic processes, enhancing cancer cell adhesion and extravasation [79]. One mechanism by which loss of A and B antigens contributes

to tumor progression is the reduction in cell adhesion properties. Blood group antigens are known to interact with adhesion molecules such as integrins, antigens on epidermal growth factor (EGF), cadherins, intercellular adhesion molecule-1 (ICAM-1), and CD44, which are associated with cellular adhesion, motility, and proliferation. Tumor cells that lose these antigens may gain enhanced migratory capabilities, facilitating metastasis [17, 69, 70, 80, 81]. Microbial interactions may further influence carcinogenesis, particularly with blood group A, which shows increased susceptibility to microbial adhesion. Studies have linked blood group A with elevated risks of oral cancer, possibly due to microbial affinity for A antigen structures [17, 58, 59, 70, 78, 82].

The secretor phenotype theory, controlled by the presence of the *Se* gene, particularly prevalent in individuals with blood group O, has also been introduced as a protective mechanism against oral malignancy [77]. Secretor people possess the ability to secret ABO (H) blood group antigens in body fluids such as saliva, whereas non-secretors lack this ability. Several studies have demonstrated a significant correlation between non-secretor status and the prevalence of OPMDs. For instance, it was found that 87% of individuals with OPMDs were non-secretors compared to 16% in the control group [69]. Additional studies indicated that non-secretors were more prone to developing conditions like OSMF [83], with all OSF patients in one study being non-secretors [84]. However, conflicting findings exist regarding the role of secretors status in oral cancer susceptibility. While some studies support the hypothesis that non-secretors have a higher risk of oral cancer, others, such as Cerovic et al and Lamey et al, found no significant association between secretor status and oral cancer risk [85, 86].

Conclusions and Future Directions

Despite the association between ABO blood groups and oral diseases like OPMDs and oral cancer, discrepancies in findings underscore the need for future research into genetic and environmental factors influencing these relationships. Patients with blood group A may require closer monitoring for early-stage lesions, while individuals with blood group O may have a better prognosis. Loss or alteration of ABO blood group antigens, downregulation of glycosyltransferases, and their interaction with adhesion molecules and microbial factors contribute to oral cancer risk. Public health efforts should focus on educating populations about risk factors and promoting early detection to enhance survival rates and improve overall health outcomes. Future studies should consider large, diverse populations and standardized methodologies to better elucidate the mechanisms underlying these associations and their implications for clinical practice and public health interventions.

Acknowledgments

Khaldun Jacoub and Ali Abdelfattah were supported by the Hashemite University, Zarqa, Jordan.

Financial Disclosure

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of Interest

The authors declare no conflict of interest.

Author Contributions

Conceptualization, KJ and AA; data collection and analysis, KJ and AA; writing - original draft preparation, KJ and AA; writing - review and editing, KJ and AA. All authors have read and agreed to the published version of the manuscript.

Data Availability

The authors declare that data supporting the findings of this study are available within the article.

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