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Bombay and para-Bombay blood group phenotypes: Molecular basis, epidemiology, diagnosis, and transfusion management: An extended review

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Abstract

Bombay (Oh) and Para-Bombay are rare variants of the ABO blood group system that carry significant clinical importance. They are characterized by the absence or a marked reduction in the expression of the H antigen on red blood cells (RBCs). This deficiency leads to a failure in the synthesis of A and B antigens, predisposing patients—particularly those with the Bombay phenotype—to developing potent anti-H antibodies, which can cause severe hemolytic transfusion reactions.

Objective: The primary goal is to provide clinicians and laboratory specialists with a practical and comprehensive framework to prevent avoidable blood mismatch and improve clinical outcomes for patients suffering from H-deficient phenotypes.

Methods: This review summarizes the underlying biology behind FUT1/FUT2 deficiency and highlights its effects on population groups and founder effects. It integrates serological and molecular tools into a practical diagnostic workflow. Furthermore, it outlines management strategies for pre-surgery, obstetrics, and emergencies, and reviews emerging translational methods, such as the enzymatic removal of H from group O red blood cells.

Findings: The review emphasizes the critical nature of early engagement with rare donor networks. It identifies the biological mechanisms of H deficiency and provides a structured approach to managing the risks associated with these rare phenotypes, ensuring that high-potency antibodies do not lead to life-threatening transfusion complications.

Conclusion: Implementing a practical and comprehensive framework in laboratories is essential to prevent transfusion incompatibility. By utilizing advanced diagnostic tools and early coordination with donor networks, the safety and outcomes of patients with H antigen deficiency can be significantly enhanced.

Keywords: Bombay (Oh); Para-Bombay; FUT1; FUT2; H Antigen; Anti-H; Ulex Europaeus; Secretor; Rare Donor Programs.

1. Introduction

The ABO blood group system remains the fulcrum of transfusion medicine because naturally occurring anti-A and anti-B antibodies can precipitate immediate hemolytic reactions upon exposure to incompatible red cells. At the biochemical core of the ABO architecture lies the H antigen, an α(1,2)-fucosylated scaffold on type-1 and type-2 precursor chains upon which A and B transferases act; when H is absent or severely reduced, ABO expression collapses regardless of the underlying ABO genotype (1,2) In physiological terms, group O erythrocytes typically display the highest H density,

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whereas A, B, and AB cells bear proportionally less, an asymmetry that becomes decisive in H-deficient states and explains why the intuitive "safety" of group O is dangerously misleading for Bombay recipients (1). Beyond its centrality to ABO, H expression participates in a broader glycan landscape on both glycolipids and glycoproteins of the red-cell membrane, meaning that changes in H often coincide with subtle shifts in antigen presentation and lectin reactivity that confound routine tube and gel methods in busy transfusion services.

Clinically, H deficiency entered the literature with the Bombay discovery in 1952, when Bhende and colleagues described patients whose red cells failed to react not only with anti-A and anti-B but also with anti-H, and whose sera agglutinated all H-positive cells—thus defining the Bombay (Oh) phenotype and establishing H as the obligatory precursor for A and B antigens (3). The observation immediately reframed the concept of "universal donor" blood and set the stage for modern rare-donor logistics. Subsequent decades revealed that H-deficiency is not a single entity but a spectrum anchored by Bombay at the null end and para-Bombay states where residual H is weak, patchy, or adsorbed to the membrane. This spectrum has practical consequences: some para-Bombay individuals display inconspicuous serology until stressed by illness, pregnancy, or transfusion, at which point diagnostic ambiguity can delay life-saving therapy (4).

The molecular basis was subsequently mapped to two homologous α(1,2)-fucosyltransferases on chromosome 19. FUT1 (the H gene) encodes the erythroid enzyme that generates H on the red-cell surface, while FUT2 (the Secretor gene) governs ABH synthesis in epithelial tissues and secretions (5,6). Biallelic loss-of-function in FUT1 abolishes RBC H and, in the setting of FUT2 non-secretor alleles, yields the canonical non-secretor Bombay phenotype; conversely, hypomorphic FUT1 activity on a Secretor-positive background produces para-Bombay with trace or adsorbed H on erythrocytes while ABH is detectable in saliva (7-9).Over the past three decades, comprehensive allele catalogs and functional studies have expanded the global FUT1 variant spectrum—spanning missense, nonsense, frameshift, and splice-site changes—and have clarified which variants are null (Bombay) versus weak (para-Bombay), thereby aligning genotype calls with clinical serology and reducing the number of "variants of uncertain significance" in reports (2,8-9). Importantly, this genotype–phenotype insight feeds back into practice by prioritizing confirmatory testing, family studies, and targeted donor recruitment in regions where founder mutations are enriched.

From a laboratory perspective, recognition proceeds stepwise. Initial flags are forward–reverse ABO discrepancies, followed by explicit interrogation of RBC H using Ulex europaeus (anti-H) lectin, assessment of Secretor status from saliva to separate Bombay from para-Bombay, and molecular confirmation by sequencing FUT1/FUT2 where serology is equivocal or where clinical planning depends on precise classification. While Ulex remains a mainstay, its agglutination profile varies with H density and, at times, by reagent lot; results therefore require internal controls and correlation with reverse typing to avoid misinterpretation (10,11). The clinical stakes are high: misclassification as group O has repeatedly triggered acute hemolytic transfusion reactions in unsuspected Bombay individuals (12), reinforcing the principle that unresolved ABO discrepancies must never default to routine O transfusion (1,12). In parallel, system-level measures—alerts in electronic health records, rare-donor pathways, and patient education cards—translate the biology into safer bedside outcomes.

2. Historical Background

The recognition of the Bombay phenotype reshaped ABO doctrine by proving that H is the biochemical precursor for A and B. Early post-war case descriptions documented severe hemolytic transfusion reactions following administration of H-positive blood to unrecognized Bombay recipients, establishing a cautionary narrative that reverberates in transfusion policies to this day (13). As case reporting expanded beyond India, investigators on Réunion Island described cohesive clusters with non-secretor status and consistent serology, indicating founder effects in geographically contained communities (14). Parallel work in large national services, such as Iran's multi-year donor screening, demonstrated that systematic approaches can quantify rarity, identify families, and seed rare-donor registries that materially alter patient trajectories during emergencies (15). The historical arc thus runs from a single index observation in Mumbai to a mature framework that integrates population genetics, serologic nuance, and international logistics.

3. Molecular and Biochemical Basis

FUT1 and FUT2 are homologous α(1,2)-fucosyltransferases that share catalytic logic but differ in tissue program. FUT1 is expressed in erythroid cells and vascular endothelium; FUT2 is active in epithelial tissues where it governs secretor status, shaping ABH expression in saliva, gastrointestinal mucosa, and other secretions (16). In biochemical terms, FUT1 transfers L-fucose to the terminal galactose of type-2 chains on glycoproteins and glycolipids, yielding the H

determinant that A and B transferases subsequently elaborate. Null FUT1 variants abolish this step, producing cells that cannot display A or B even if the ABO genotype encodes functional transferases. Hypomorphic missense variants may retain partial activity, manifesting as para-Bombay where H is weak or uneven; in secretor-positive individuals, soluble ABH can adsorb to RBC membranes and complicate interpretations of adsorption–elution studies (9, 17,18).

The genotype landscape is populated by regionally enriched alleles. Recurrent Indian missense changes such as c.725T>G have been reported alongside small deletions and frameshifts; Réunion Island studies documented a different founder allele (e.g., c.349C>T) in non-secretor backgrounds. Recent functional assays measured enzyme activity for previously uncharacterized FUT1 single-nucleotide variants and confirmed predicted loss- or weak-function categories, enhancing clinical confidence in assigning "Bombay versus para-Bombay" at the bench. This clarity also guides family counseling: once a causative FUT1 allele is defined, cascade testing can identify potential rare donors and inform reproductive genetics where consanguinity is common (8,14, 19).

4. Immunohematology and Serology

From an immunohematologic standpoint, H is not evenly distributed across ABO phenotypes: O cells carry the most H, followed by A2 and B, then A2B, with A1 and A1B showing the least—differences that modulate agglutination patterns and explain why Ulex reactions can appear deceptively strong with O controls and faint on certain subgroups. In Bombay sera, anti-H is typically a naturally occurring cold-reactive IgM that may fix complement at higher thermal amplitudes; IgG anti-H is less common but has been implicated in clinically significant reactions. Para-Bombay patients may produce anti-H or anti-HI with variable titers, and their serology can drift over time, especially after pregnancy or transfusion exposures. Practical pitfalls include cold autoagglutinins that mask specificity, prozone effects in high-titer sera, and adsorption of soluble ABH from secretions that transiently diminishes apparent RBC reactivity (10,11). Robust workflows therefore pair Ulex testing with temperature-controlled readings, defined positive/negative controls, and, where appropriate, pre-warm techniques to manage cold-reactive interference.

5. Diagnostic Approaches

Diagnosis begins with reconciling forward and reverse ABO results rather than privileging one stream over the other. Negative or very weak reactions with Ulex europaeus suggest H-deficiency and should trigger saliva testing for secretor status and adsorption–elution to unmask trace A/B/H on the membrane. Extended antibody screens, thermal amplitude studies, and, when indicated, direct antiglobulin testing (DAT) contextualize the serology in patients with recent transfusion or autoimmune background. Molecular confirmation by Sanger sequencing or targeted next-generation panels for FUT1/FUT2 anchors classification, resolves cryptic serology, and enables family screening and participation in rare-donor programs (10–11). Laboratories should document reagent lot numbers, control reactivity, and decision points in the algorithm to support reproducibility and external review, particularly when cases are escalated to reference centers.

6. Epidemiology

Prevalence varies by geography and marriage structure. Indian series cite figures near ~1 in 10,000 in certain locales; East Asian and European estimates range from ~1 in 300,000 to 1,000,000. Réunion Island and Iran provide well-characterized clusters shaped by founder effects and consanguinity, while sporadic reports from Korea and Taiwan show that para-Bombay may be under-detected in systems relying solely on routine ABO typing (20–22). These patterns argue for targeted education in high-prevalence districts, early contact with national rare-donor registries when elective surgery is planned, and integration of select molecular testing into tertiary-care transfusion services.

Table 1 Selected prevalence snapshots and epidemiological notes

Region/Population	Reported estimate / highlights	Reference
Mumbai / Maharashtra (India)	Clusters up to ~1:7,600; donor demand high	(23)
Southern Bengal (India)	Rare overall; ~1:10,000 cited for India; donor strategies	(20)
Réunion Island	42 H-deficient individuals characterized; non-secretors	(14)
Iran (national donors)	56 Bombay among >7M; ~0.0008%; consanguinity signal	(15)
Korea (tertiary center)	First para-Bombay case; Southeast Asian patient	(22)

7. Clinical Presentation and Risks

Most individuals are clinically silent until transfusion, surgery, or pregnancy exposes the phenotype. Acute hemolytic transfusion reactions have followed inadvertent infusion of H-positive blood—often group O—to Bombay recipients, with presentations ranging from fever and hemoglobinuria to fulminant intravascular hemolysis requiring critical care. In surgical settings, unrecognized para-Bombay individuals may experience unexpected incompatibility at the crossmatch bench, delaying procedures and necessitating rapid escalation to reference laboratories. Obstetric care introduces additional complexity: while classic anti-H is usually IgM and poorly transplacental, warmer-reactive or complement-activating specificities have been described; consequently, antenatal planning should include titers, partner typing, and neonatal surveillance for hemolysis or hyperbilirubinemia when risk factors coexist (24-26).

8. Transfusion Management

Bombay recipients must receive Bombay (Oh) units only and should be flagged permanently in the medical record to prevent emergency issuance of uncrossmatched O blood. Para-Bombay transfusion is individualized according to antibody profile, thermal amplitude, and crossmatch performance; in select cases, carefully vetted O units may be acceptable, but practice should default to caution and specialist input. Elective surgery benefits from autologous pre-deposit when feasible, titration studies to understand antibody behavior, and early activation of rare-donor pathways such as the International Rare Donor Panel (IRDP) and the American Rare Donor Program (ARDP/REGGI) to locate compatible units and, where available, request cryopreserved stocks (27–28). Institutions should maintain checklists that forbid issuing "universal" O blood in unresolved ABO discrepancies and require senior authorization, thereby converting lessons from case reports into durable safety culture.

Table 2 Practical transfusion guidance

Clinical situation	Preferred action
Confirmed Bombay (Oh)	Bombay RBC units only; consider autologous and cryopreserved stocks; alert in EHR
Suspected para-Bombay	Full serology + genotyping; individualized crossmatch; O units only if strictly compatible
Emergency surgery	Activate IRDP/ARDP search early; reference-lab consultation; avoid uncrossmatched O
Obstetrics	Plan delivery with blood bank; type partner; monitor maternal anti-H; secure compatible units

9. Genotype–Phenotype Correlation

Documented FUT1 variants include recurrent Indian alleles (e.g., c.725T>G), Réunion founder alleles (e.g., c.349C>T), frameshifts, and diverse missense changes. Hypomorphic alleles align with para-Bombay, whereas null alleles align with Bombay. The practical value of classification is twofold: first, it calibrates the expected serologic behavior, guiding whether adsorption–elution or pre-warm workflows are likely to help; second, it identifies at-risk relatives who may be both potential donors and future patients. ISBT allele tables and recent functional assays help resolve borderline interpretations and harmonize reports across laboratories, reducing uncertainty in counseling and clinical decision-making (29,30).

Bombay (Oh) and para-Bombay phenotypes also differ substantially in their antigen expression, secretor status, antibody profiles, and transfusion management requirements. In Bombay phenotype, H antigen is completely absent from the red cell surface due to biallelic loss-of-function mutations in FUT1, typically combined with a non-secretor FUT2 background, leading to the absence of ABH substances in saliva. These patients often produce strong anti-H antibodies, usually IgM with potential complement activation, and react negatively with Ulex europaeus lectin. Transfusion with any H-positive blood, including group O, can trigger severe acute hemolytic reactions, making Bombay-compatible units the only safe option (31,32). In contrast, para-Bombay individuals generally carry hypomorphic FUT1 variants that allow weak, patchy, or adsorbed H antigen expression; they are frequently secretor-positive, with ABH substances detectable in saliva, and may produce anti-H or anti-HI of variable titers. Their Ulex europaeus lectin reactivity is weak or inconsistent, and transfusion strategies must be individualized, with group O units

used only if strict compatibility is confirmed (33). The following table summarizes the main distinctions relevant to laboratory diagnosis and transfusion planning.

Table 3 Key Differences Between Bombay and para-Bombay Phenotypes

Feature	Bombay (Oh)	para-Bombay
H expression on RBC surface	Completely absent (null)	Weak, patchy, or adsorbed (partial)
FUT1 genotype	Biallelic loss-of-function variants	Hypomorphic variants with residual activity
FUT2 (Secretor) status	Usually non-secretor	Frequently secretor-positive
ABH expression in saliva	Absent	Present
Antibody profile	Strong anti-H, typically IgM; may fix complement	Anti-H or anti-HI with variable titers
Reaction with Ulex europaeus lectin	Negative	Weak or variable
Transfusion risk	Severe hemolytic reaction if given H-positive blood	Variable risk depending on antibody thermal amplitude and titer
Transfusion management	Bombay units only	Individually crossmatched units; O units only if strictly compatible

10. Pregnancy and Neonates

Peripartum management benefits from early extended typing, anti-H titration, and Secretor testing, ideally in the second trimester or sooner when obstetric risk is anticipated. Coordination with rare-donor networks ensures that compatible units are available if hemorrhage occurs, while patient blood management—iron optimization, tranexamic acid where indicated, and cell salvage in appropriate theaters—minimizes allogeneic exposure. Neonatal risk is generally low for cold-reactive IgM anti-H, but vigilance is warranted for atypical specificities; cord blood testing (including DAT with eluate if positive), bilirubin trends, and clinical observation provide an adequate safety net in most scenarios (34-36).

11. Systems and Rare-Donor Infrastructure

National and international rare-donor programs shorten search times and provide cryopreserved inventories that can be mobilized for urgent cases. The IRDP and ARDP/REGGI function as clearinghouses that connect phenotype-defined patients to compatible donors across borders; enrollment requires accurate serology, ideally with molecular confirmation, and institutional contacts familiar with shipping and thawing protocols (37-39). Hospitals that care for diverse populations should maintain internal SOPs that spell out escalation triggers, 24/7 contact routes to reference laboratories, and documentation standards so that transient staff do not default to unsafe “universal” strategies in crises.

12. Challenges in Low-Resource Settings

In low-resource healthcare environments, the recognition and management of Bombay and para-Bombay phenotypes face several significant obstacles:

- Limited awareness and training – Many healthcare workers are unfamiliar with the clinical implications of H-deficiency, leading to misinterpretation of ABO discrepancies and inappropriate use of “universal” O units.
- Lack of advanced diagnostic tools – Absence of Ulex europaeus lectin, adsorption–elution methods, or access to FUT1/FUT2 genotyping hampers definitive classification, especially in equivocal cases.
- Weak rare-donor infrastructure – Many countries lack national rare donor registries or cryopreserved inventories, making emergency sourcing of compatible units challenging.
- Delays in cross-border unit procurement – Logistical barriers, customs regulations, and limited transport networks slow down international unit transfer from organizations such as the IRDP or ARDP.

- Risk of inappropriate "universal" donor use – In the absence of diagnosis, Bombay patients may receive O+ or O− blood, triggering life-threatening hemolytic reactions.

Practical recommendations for such settings include developing local rare-donor lists, ensuring the availability of basic serologic reagents in regional reference labs, integrating H-deficiency into transfusion training programs, and collaborating with global rare-donor networks to expedite unit sourcing.

12.1. Future Directions

Three fronts are likely to shape practice. Population genotyping can pre-identify rare donors in high-prevalence regions, reducing lead-times and enabling strategic cryopreservation. Harmonized variant classification anchored in ISBT tables and prospective functional studies will continue to shrink the gray zone of VUS calls and standardize counseling (40). Finally, enzymatic engineering of donor red cells has moved from concept to laboratory reality: bacterial α-1,2-fucosidase (FucOB) can remove terminal fucose from H on group O red cells ex vivo, generating cells serologically compatible with anti-H; if forthcoming safety and efficacy studies prove favorable, such tools could complement rather than replace rare-donor networks, offering contingency capacity during surges or in geographically isolated settings (41–42).

13. Conclusion

Bombay and para-Bombay phenotypes transform routine ABO typing into a high-stakes diagnostic exercise. The essential safeguards are consistent across settings: reconcile ABO discrepancies without compromise; interrogate H explicitly with validated reagents; confirm with FUT1/FUT2 genotyping where necessary; and plan transfusion pathways early via rare-donor infrastructure. When these elements are embedded into laboratory policy and clinical workflows, catastrophic incompatibility becomes preventable rather than inevitable, even in resource-limited environments.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflict of interest.

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