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Brief biography

# Extension of *Homo Sapiens* Adapting to Every Environment with Divergent Phenotypes: Blood Type Incompatible in Pregnancy as an Abaxial Phenomenon

Since the genesis of living organisms, the selection pressure or survival competition of any species has emerged through continuous, uncountable mutations. Prior to the emergence of *Homo sapiens* from subhuman primates, many pathogens, such as malaria species, have long and severely affected them [1]. Beginning 100,000 to 40,000 years ago, modern humans migrated from Africa and adapted their cultures and phenotypes to extremes of altitudes, temperature, and humidity throughout the planet.

Various erythrocyte mutations are thought to have emerged with a survival neutrality, disadvantages, or advantages in avoiding death before reproductive ages, such as hemoglobinopathies and the distribution of blood groups. The most common allele of group O (O01) is derived primarily from the group A allele (A01) with a single base deletion resulting in failure to produce a functional A transferase. Subsequently, O individuals have manifested an immune response to produce strong anti-A and anti-B isoantibodies, which may provoke intrauterine fetal demise or severe postpartum morbidity. In modern humans, however, a reduction of A and B antigen sites on neonatal red cells to about one-third, combined with diminished branching of A and B chains, improve fetal and neonatal outcomes in the context of maternally-derived cognate antibodies [2].

This Theme Section offers guidance to readers who have been and those who will be making efforts for mothers and infants associated with blood group incompatible pregnancy and related issues.

Gonzalez CA and Gonzalez S outline the allo-immune system of the fetus and neonate. The fetus is immunocompetent and acquires the ability to generate an immune response and also specific tolerance as it is exposed to genetically foreign and non-inherited maternal antigens [3]. At birth, the immune system does not attack nor harm maternal tissues.

Takeshita A and East Asian collaborators demonstrate red cell alloimmunity: 1) anti-E is consistently one of the most frequently found antibodies; 2) anti-Mi$^a$ is frequently detected in patients from Hong Kong and Thailand but rarely in Japan and South Korea; 3) anti-Di$^a$ is observed in Japan, South Korea, and Shanghai; and 4) anti-Jr$^a$ is frequently detected in females in Japan and South Korea [4].

The Gupta GK et al. review has an emphasis on alloimmunization in pregnancy; 1) the impact of alloantibodies in the peripartum period; 2) pathophysiologic mechanisms to influence alloimmunization; 3) laboratory tools; and 4) future directions for transfusion related to alloimmunization affecting pregnancy [5].

Hyland and others state that non-invasive prenatal testing (NIPT) fetal genotyping for atypical (other than RhD) blood group antigens presents more challenges as most arise from a single nucleotide variant. Recent studies show potential for genomic and digital technologies to provide a personalised medicinal approach with NIPT to assess fetal blood group status for women with other (non-D) red cell antibodies to manage the risk for HDFN [6].

Tyndall C et al. review early postnatal and long-term outcomes of rhesus hemolytic disease of the fetus and newborn. Even when favorable outcomes for the fetus and neonate can be expected, ongoing investigation of long-term consequences will help optimize the health of adult survivors of affected infancies [7].

Ohto H and others postulate three non-classical mechanisms, including an apoptotic pathway, erythropoietic suppression, and clonal escape from erythropoietic suppression for anemic (less hemolytic) disease of the fetus and newborns for elucidating anti-Kell, anti-Ge3, anti-M, and anti-Jr$^a$ cases [8].

TJ Legler summarizes anti-D- immunoglobulin (RhIg), produced from specially donated plasma, that has been utilized to prevent an anti-D alloimmunization, and has decreased the frequency of hemolytic disease of the fetus and newborn. As fetal RhD status can be determined accurately using mother's peripheral blood, targeted antenatal anti-D prophylaxis is becoming a new standard. The efficacy of intravenous immunoglobulin (IVIG) in the management of these neonates will remain inconclusive until a large randomized, double blind study is performed [9].

Bianchi M et al. review transfusion-associated neonatal infections. Blood donor tests have made transfusions progressively safer; however, more evidence is needed to firmly establish the degrees of cytomegalovirus protection conferred by leukoreduction of blood products and selection of seronegative donors [10].

Orlando N et al. update us on clinical application of umbilical cord blood platelet derivatives for regenerative medicine [11].

This guest editor is deeply grateful for all contributors to this Theme Section. The genetic inheritance of *Homo sapiens* is a matter of natural selection, but our cultural and intellectual inheritance is a matter of volition. Among those readers, we dare to imagine those who will further advance fetomaternal care for generations to come.

## Declaration of Competing Interest

The authors report no declarations of interest.

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Biography of Professor Emeritus OHTO Hitoshi, the Guest Editor for this theme section, "*Maternofetal interaction, blood group incompatibility and its interventions*". In a career *including* 22 years on the editorial board of *Transfusion and Apheresis Science*, Prof. OHTO Hitoshi has guest edited four prior theme sections: 1) Efficient and safe stem cell apheresis (2004) [1], 2) Platelet preservation: past, present, and future (2011) [2], 3) Disaster and blood transfusion (2016) [3], and 4) Progenitor/stem cell apheresis and its related issues (2018) [4].

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