

Advances In Neonatal Hematology

Neonatal fragment crystallizable receptor

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The neonatal fragment crystallizable (Fc) receptor (also FcRn, IgG receptor FcRn large subunit p51, or Brambell receptor) is a protein that in humans is encoded by the FCGRT gene. It is an IgG Fc receptor which is similar in structure to the MHC class I molecule and also associates with beta-2-microglobulin. In rodents, FcRn was originally identified as the receptor that transports maternal immunoglobulin G (IgG) from mother to neonatal offspring via mother's milk, leading to its name as the neonatal Fc receptor. In humans, FcRn is present in the placenta where it transports mother's IgG to the growing fetus. FcRn has also been shown to play a role in regulating IgG and serum albumin turnover. Neonatal Fc receptor expression is up-regulated by the proinflammatory cytokine, TNF, and down-regulated...

Salah Foundation Children's Hospital

renovation of the neonatal intensive care unit at the hospital, with \$100,000 of the donation going towards the pediatric hematology/oncology unit. In 2013 doctors

Salah Foundation Children's Hospital (SFCH), formerly Chris Evert Children's Hospital, is a pediatric acute care children's hospital located within Broward Health Medical Center in Fort Lauderdale, Florida. The hospital has 135 pediatric beds. It is affiliated with Nova Southeastern University College of Osteopathic Medicine, and is a member of Broward Health. The hospital provides comprehensive pediatric specialties and subspecialties to infants, children, teens, and young adults aged 0-21 throughout southeastern Florida. The hospital is also a Level I Pediatric Trauma Center.

The Broward Health Children's Hospital at Broward Health Medical Center was originally named after Broward native Chris Evert, before being renamed the "Salah Foundation Broward Health Children's Hospital." The hospital...

Neonatal red cell transfusion

Selamawit (January 2016). "Prevention of Iatrogenic Anemia in Critical and Neonatal Care". Advances in Clinical and Experimental Medicine. 25 (1): 191-197.

Neonates are defined as babies up to 28 days after birth. Most extremely preterm babies (less than 28 weeks) require at least one red cell transfusion; this is partly due to the amount of blood removed with blood samples compared to the baby's total blood volume (iatrogenic anemia) and partly due to anemia of prematurity. Most transfusions are given as small volume top-up transfusions to increase the baby's hemoglobin above a certain pre-defined level, or because the baby is unwell due to the anemia. Possible side-effects of anemia in babies can be poor growth, lethargy and episodes of apnea. Exchange blood transfusion is used to treat a rapidly rising bilirubin that does not respond to treatment with phototherapy or intravenous immunoglobulin. This is usually due to hemolytic disease of the...

Neutropenia

8% of all newborns in neonatal intensive care units (NICUs). Out of the approximately 600,000 neonates annually treated in NICUs in the United States,

Neutropenia is an abnormally low concentration of neutrophils (a type of white blood cell) in the blood. Neutrophils make up the majority of circulating white blood cells and serve as the primary defense against infections by destroying bacteria, bacterial fragments and immunoglobulin-bound viruses in the blood. People with neutropenia are more susceptible to bacterial infections and, without prompt medical attention, the condition may become life-threatening (neutropenic sepsis).

Neutropenia can be divided into congenital and acquired, with severe congenital neutropenia (SCN) and cyclic neutropenia (CyN) being autosomal dominant and mostly caused by heterozygous mutations in the ELANE gene (neutrophil elastase). Neutropenia can be acute (temporary) or chronic (long lasting). The term is sometimes...

Beta-2 microglobulin

but also with class I-like molecules such as CD1 (5 genes in humans), MR1, the neonatal Fc receptor (FcRn), and Qa-1 (a form of alloantigen). Nevertheless

β 2 microglobulin (B2M) is a component of MHC class I molecules. MHC class I molecules have α 1, α 2, and α 3 proteins which are present on all nucleated cells (excluding red blood cells). In humans, the β 2 microglobulin protein is encoded by the B2M gene.

Allied Academies

Reproductive Endocrinology Hematology and Blood Disorders Immune System and Disorders Journal Immunology Case Reports Insights in Nutrition and Metabolism

Allied Academies (also known as Allied Business Academies) is a reportedly fraudulent corporation chartered under the laws of North Carolina. Its postal address is in London, United Kingdom. It presents itself as an association of scholars, with supporting and encouraging research and the sharing and exchange of knowledge as its stated aims. The organization consists of 30 affiliate academies, which provide awards to academics and publish academic journals both online and in hard copy for members. Since 2015 the organization has been listed on Jeffrey Beall's list of "potential, possible, or probable predatory scholarly open-access publishers". It is in a partnership with OMICS Publishing Group which uses its website and logo. In 2018, OMICS owner Srinubabu Gedela declared that he had informed...

Hemolytic jaundice

of Hematology. 92 (3): 432-8. doi:10.1007/s12185-010-0667-9. PMID 20820969. S2CID 71018193. Billing BH (June 1978). "Twenty-five years of progress in bilirubin

Hemolytic jaundice, also known as prehepatic jaundice, is a type of jaundice arising from hemolysis or excessive destruction of red blood cells, when the byproduct bilirubin is not excreted by the hepatic cells quickly enough. Unless the patient is concurrently affected by hepatic dysfunctions or is experiencing hepatocellular damage, the liver does not contribute to this type of jaundice.

As one of the three categories of jaundice, the most obvious sign of hemolytic jaundice is the discolouration or yellowing of the sclera and the skin of the patient, but additional symptoms may be observed depending on the underlying causes of hemolysis. Hemolytic causes associated with bilirubin overproduction are diverse and include disorders such as sickle cell anemia, hereditary spherocytosis, thrombotic...

Reticular dysgenesis

(1998). "Recent advances in the pathogenesis and treatment of nonimmune neutropenias in the neonate". Current Opinion in Hematology. 5 (1): 37-41. doi:10

Reticular dysgenesis (RD) is a rare, inherited autosomal recessive disease that results in immunodeficiency. Individuals with RD have mutations in both copies of the AK2 gene. Mutations in this gene lead to absence of AK2 protein. AK2 protein allows hematopoietic stem cells to differentiate and proliferate. Hematopoietic stem cells give rise to blood cells.

Differentiation and proliferation of hematopoietic stem cells require a lot of energy and this energy is supplied by the mitochondria. The energy metabolism of mitochondria is regulated by the AK2 protein. If there is a mutation in the protein, that means that the mitochondria metabolism most likely will be altered and will not be able to provide enough energy to the hematopoietic stem cells. As a result, hematopoietic stem cells will not...

Thalassemia

(December 2017). "Impact of bone disease and pain in thalassemia". Hematology. American Society of Hematology. Education Program. 2017 (1): 272-277. doi:10

Thalassemias are a group of inherited blood disorders that manifest as the production of reduced hemoglobin. Symptoms depend on the type of thalassemia and can vary from none to severe, including death. Often there is mild to severe anemia (low red blood cells or hemoglobin), as thalassemia can affect the production of red blood cells and also affect how long the red blood cells live. Symptoms include tiredness, pallor, bone problems, an enlarged spleen, jaundice, pulmonary hypertension, and dark urine. A child's growth and development may be slower than normal.

Thalassemias are genetic disorders. Alpha thalassemia is caused by deficient production of the alpha globin component of hemoglobin, while beta thalassemia is a deficiency in the beta globin component. The severity of alpha and beta...

Beta thalassemia

Neufeld EJ (2010). "Update on iron chelators in thalassemia". *Hematology. American Society of Hematology. Education Program*. 2010: 451–455. doi:10

Beta-thalassemia (β -thalassemia) is an inherited blood disorder, a form of thalassemia resulting in variable outcomes ranging from clinically asymptomatic to severe anemia individuals. It is caused by reduced or absent synthesis of the beta chains of hemoglobin, the molecule that carries oxygen in the blood. Symptoms depend on the extent to which hemoglobin is deficient, and include anemia, pallor, tiredness, enlargement of the spleen, jaundice, and gallstones. In severe cases death ensues.

Beta thalassemia occurs due to a mutation of the HBB gene leading to deficient production of the hemoglobin subunit beta-globin; the severity of the disease depends on the nature of the mutation, and whether or not the mutation is homozygous. The body's inability to construct beta-globin leads to reduced...

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