# **Comparative Analysis of Human-Specific Diseases and Susceptibility in Great Apes**

**1. Meningococcal Meningitis**

Meningococcal meningitis is a severe and potentially life-threatening infection characterized by the inflammation of the meninges, the membranes enveloping the brain and spinal cord. This condition arises from the bacterium *Neisseria meningitidis*, also known as meningococcus.1 The disease can manifest primarily as meningitis, an infection of the cerebrospinal fluid and meninges, or as meningococcal septicaemia, an infection of the bloodstream. These two forms can also occur concurrently, a condition termed invasive meningococcal disease (IMD).1

In humans, the onset of meningococcal meningitis is often abrupt, with initial phenotypes including a sudden high fever, an intense headache, and a marked stiffness of the neck.1 Patients may also experience nausea, vomiting, increased sensitivity to light (photophobia), and alterations in mental status, such as confusion, delirium, or excessive sleepiness.1 A critical indicator, particularly of meningococcal septicaemia, is the appearance of a non-blanching petechial rash, characterized by small, purplish spots that do not fade when pressed, or a more extensive purpuric rash.1 Additional symptoms associated with septicaemia can include cold hands and feet, shivering episodes, pain in the limbs, joints, or muscles, rapid breathing, and a pale or mottled skin appearance.1 In infants and very young children, the presentation might differ, with signs such as increased irritability, refusal to feed, a high-pitched cry, a stiff body with jerky movements or unusual floppiness, and a tense or bulging soft spot on the top of their head.3 The diverse array of these initial and subsequent symptoms underscores the systemic impact of IMD, extending beyond the central nervous system to involve the circulatory, integumentary, and musculoskeletal systems. The age-specific variations in presentation emphasize the need for tailored clinical vigilance across different patient demographics.

Survivors of meningococcal meningitis can face a range of long-term complications and sequelae. These can include permanent hearing loss, various forms of neurological damage, difficulties with learning and intellectual functions, problems with coordination, persistent headaches, the development of epilepsy, muscle weakness or paralysis, speech impairments, vision problems, hydrocephalus (fluid accumulation in the brain), mental health disorders such as anxiety and depression, chronic fatigue, arthritis or joint stiffness, scarring of the skin, kidney damage, and in severe cases, the loss of fingers, toes, or limbs.1 The fact that initial symptoms can be non-specific 1 poses a significant diagnostic challenge, potentially leading to delays in the administration of appropriate treatment, which can negatively impact patient outcomes. However, the recognition of specific phenotypes, such as the characteristic non-blanching rash 1, is crucial for prompting timely medical intervention.

*Neisseria meningitidis* is considered a pathogen with a high degree of host specificity for humans.2 While meningitis has been documented in other great ape species like chimpanzees, gorillas, and orangutans, these instances have rarely, if ever, been attributed to *N. meningitidis*.10 Instead, these cases were often linked to other bacterial agents such as *Streptococcus pneumoniae* and *Klebsiella pneumoniae*, or non-infectious causes including trauma or infection by amoebae.10 Notably, one study reported the presence of *Neisseria* bacteria in the urethra of a chimpanzee, but the specific strain was different from those known to cause meningococcal disease in humans.10 Despite the genetic similarities that make great apes susceptible to a wide range of human pathogens 15, their susceptibility to *N. meningitidis* meningitis appears to be significantly lower than that of humans. This difference might be attributed to variations in human social behaviors that facilitate the transmission of this bacterium, which spreads through close contact 2, or to specific aspects of human molecular biology that allow the pathogen to invade the central nervous system more effectively.10 Furthermore, the interaction between human complement regulatory proteins, such as factor H and C4b-binding protein, and *N. meningitidis* appears to be specific to the human versions of these proteins 10, potentially offering an explanation for the species-specific susceptibility.

In humans, *Neisseria meningitidis* initiates infection by attaching to and multiplying within the mucosal cells lining the nasopharynx and oropharynx.4 From this initial site, the bacteria can then penetrate the mucosal epithelial barrier and gain entry into the bloodstream.4 Once in the bloodstream, the meningococci can interact with the endothelial cells that line the blood vessels 33 and encounter various cells of the immune system, including peripheral blood mononuclear cells.35 To cause meningitis, the bacteria must then traverse the blood-brain barrier (BBB) to infect the meninges 7, a process that involves interaction with specialized endothelial cells of the brain microvasculature (HBMEC).34 During the course of meningococcal meningitis, the cerebrospinal fluid (CSF) typically exhibits a characteristic profile, including a high number of neutrophils 4, indicating a robust inflammatory response within the central nervous system. The host's overall immune response to *N. meningitidis* involves a coordinated action of various cell types, including neutrophils, macrophages, dendritic cells, B cells (which produce crucial antibodies targeting the bacterial polysaccharide capsule and other antigens), and T cells (both CD4+ and CD8+ subsets).4 Notably, meningococci have the capacity to bind to and influence the behavior of cells within the vasculature, including peripheral blood mononuclear cells and T cells.35

Due to the rarity of *Neisseria meningitidis* as a cause of meningitis in great apes, there is a limited amount of specific information in the literature regarding the precise cell types involved in the disease process or its control in these animals.10 However, studies focusing on meningitis caused by other bacterial species in great apes have identified the presence of neutrophils in the cerebrospinal fluid 12, suggesting a conserved inflammatory response. Furthermore, research on other respiratory pathogens that affect great apes, such as influenza and respiratory syncytial virus, has indicated the involvement of respiratory epithelial cells and various immune cells, including neutrophils and lymphocytes.19

**2. Myocardial Infarction**

Myocardial infarction (MI), commonly known as a heart attack, represents the necrosis or death of heart muscle tissue (myocardium) resulting from a prolonged lack of oxygen supply (ischemia). This ischemia is typically caused by an obstruction in one or more of the coronary arteries that supply blood to the heart muscle.39 The most frequent underlying cause of this obstruction is atherosclerosis, a process characterized by the gradual buildup of cholesterol-rich plaques within the walls of the arteries.39

In humans, acute myocardial infarction often manifests with a constellation of symptoms, most notably chest pain. This pain is frequently described as a sensation of tightness, pressure, squeezing, or aching in the chest, and it may radiate to other areas such as the jaw, neck, arm (most commonly the left), or back.3 Additional symptoms can include shortness of breath, nausea, vomiting, excessive sweating, dizziness, and profound fatigue.3 Electrocardiogram (ECG) abnormalities are a hallmark of acute MI, often revealing patterns such as ST-segment elevation or depression, inversion of the T-waves, and the appearance of Q waves, all indicative of myocardial injury.42 Furthermore, the damage to heart muscle cells leads to the release of specific cardiac biomarkers, such as troponins and creatine kinase-MB (CK-MB), into the bloodstream, the detection of which in elevated levels confirms the occurrence of myocardial infarction.42 Over the long term, chronic myocardial infarction can lead to significant complications, including the development of heart failure, the occurrence of cardiac arrhythmias (irregular heartbeats), and an increased risk of recurrent coronary events.40 In these chronic states, the ECG may continue to show persistent Q waves or other T-wave abnormalities 42, and patients may experience ongoing symptoms associated with heart failure, such as persistent shortness of breath, chronic fatigue, and swelling in the extremities.43 Advanced computational modeling studies have provided further insights into the diverse electrocardiographic phenotypes observed in MI patients. These models have linked specific ECG patterns, such as T-wave inversion, Brugada phenocopy, QT interval prolongation, upright tall T-waves, T-wave alternans, and fractionated QRS complexes, to underlying cellular and molecular events in the heart, including ionic current remodeling and regional conduction abnormalities.43 Notably, there are observed differences in how men and women present with symptoms of MI 41, underscoring the importance of considering sex-specific phenotypes to ensure accurate and timely diagnosis and treatment.

Interestingly, myocardial infarction resulting from coronary artery thrombosis is a rare occurrence in great apes, including chimpanzees, gorillas, and orangutans.40 In fact, only a single case of MI with evidence of coronary artery plaque rupture has been reported in a chimpanzee.47 This is particularly surprising given that great apes often exhibit blood lipid profiles that in humans would be considered high-risk for the development of atherosclerosis, with cholesterol levels, including LDL cholesterol, frequently being higher in apes than in humans.40 Despite these pro-atherogenic lipid profiles, great apes do not typically develop significant coronary artery atherosclerosis, which remains the primary cause of MI in humans.40 Instead, the most common form of heart disease leading to sudden death or progressive heart failure in great apes is diffuse interstitial myocardial fibrosis (IMF) of unknown origin.40 This condition involves the thickening and scarring of the heart tissue, which can disrupt the heart's electrical activity, potentially leading to arrhythmias and sudden cardiac arrest.40 The reasons behind the great apes' apparent resistance to MI despite their lipid profiles are not yet fully elucidated.40 Some hypotheses suggest that differences in the immune response to nonhuman sialic acid, a sugar molecule, and the hyper-reactive nature of human T cells might play a role in the development of atherosclerosis in humans but not in apes.40

The cellular events in human myocardial infarction are initiated by the death of cardiomyocytes, the contractile cells of the heart, due to a prolonged lack of oxygen.39 These necrotic cardiomyocytes release damage-associated molecular patterns (DAMPs) that trigger an inflammatory response in the heart tissue.65 Even surviving cardiomyocytes located in the border zone of the infarct can contribute to this inflammation by producing and secreting various cytokines and chemokines.65 Endothelial cells, which line the blood vessels of the heart, also play a crucial role. Activated by the ischemic conditions and the inflammatory signals, these cells facilitate the extravasation or movement of leukocytes into the damaged area and themselves become an important source of cytokines and chemokines.64 Fibroblasts, the most abundant cell type in the heart's connective tissue, are activated in response to MI. These activated fibroblasts can differentiate into myofibroblasts, which are responsible for secreting extracellular matrix (ECM) proteins, leading to the formation of scar tissue and the remodeling of the heart after the infarction.64 A variety of immune cells infiltrate the heart tissue following MI, including neutrophils, monocytes, macrophages (which can exhibit pro-inflammatory M1 or reparative M2 phenotypes), dendritic cells, T cells, and natural killer (NK) cells. These immune cells play complex and sometimes opposing roles in the inflammatory and repair processes that follow a myocardial infarction.37

In contrast, the primary cellular pathology observed in the common heart disease of great apes, interstitial myocardial fibrosis (IMF), involves the degeneration and atrophy of cardiomyocytes.52 Fibroblasts play a key role in this condition by depositing excessive amounts of collagen in the cardiac interstitium, leading to fibrosis.57 Some studies have reported a minimal presence of inflammatory immune cells, such as lymphocytes and histiocytes, within the areas of fibrosis.59

**3. Epithelial Cancers (Carcinoma)**

Carcinomas represent a broad category of malignant tumors that originate from epithelial cells. These cells form the linings of the outer surface of the body, such as the skin, as well as the linings of internal organs and various glands.70 Notably, carcinomas are the most frequently diagnosed type of cancer in humans, accounting for approximately 80 to 90 percent of all cancer cases.71

Human carcinomas exhibit a diverse range of phenotypes, largely classified based on the specific type of epithelial cell from which they arise. For instance, adenocarcinomas originate from glandular epithelial cells that secrete substances like mucus or digestive enzymes. Squamous cell carcinomas develop from squamous cells, which are flat cells found in the skin's surface and the lining of certain organs. Basal cell carcinomas arise from the basal cells located in the deepest layer of the epidermis, while transitional cell carcinomas originate in the transitional epithelium found in organs like the bladder.71 Carcinomas are also phenotypically categorized by their extent of spread: carcinoma in situ refers to cancer that remains localized to the original site, invasive carcinoma has spread to surrounding tissues, and metastatic carcinoma has disseminated to distant parts of the body.71 Furthermore, the process of epithelial-mesenchymal transition (EMT) represents another important phenotypic aspect of carcinomas. EMT involves a series of changes where epithelial cancer cells lose their characteristic features and acquire properties of mesenchymal cells, which are associated with increased motility, invasiveness, and the ability to metastasize.70 Additionally, the microenvironment within a tumor, particularly the presence of low oxygen levels (hypoxia), can influence the phenotype of carcinoma cells, often leading to a less differentiated state.82 The link between EMT and the acquisition of cancer stem-like cell properties 80 suggests a potential mechanism for tumor recurrence and resistance to treatment.

Interestingly, carcinomas are reported to be relatively rare in captive chimpanzees and possibly in other great ape species such as bonobos, gorillas, and orangutans, despite their high degree of genetic similarity to humans.83 The incidence of common human epithelial neoplasms, including carcinomas of the breast, prostate, or lung, is significantly lower in great apes (less than 2%) compared to humans (more than 20%).85 Several factors might contribute to this difference in susceptibility, including variations in life expectancy, dietary habits, genetic predispositions, immune system responses, the composition of their microbiomes, and other environmental exposures.83 While benign tumors and types of cancer that are less common in humans have been documented in great apes, carcinomas affecting organs such as the esophagus, lung, stomach, pancreas, colon, uterus, ovary, or prostate are rarely observed.17 Specific genetic differences between humans and great apes, such as variations in the BRCA2 gene, which plays a crucial role in DNA repair and cancer prevention, might also contribute to the higher rates of cancer in humans.88

In humans, carcinomas originate from a diverse range of epithelial cell types, depending on the specific location within the body where the cancer develops.71 For example, adenocarcinomas arise from glandular epithelial cells, which are found in various organs and glands throughout the body. Squamous cell carcinomas originate from squamous cells, which are a major component of the skin's epidermis and also line the surfaces of many internal organs. Basal cell carcinomas develop from basal cells, located at the base of the epidermis. The specific cell types involved in a carcinoma are therefore determined by the organ or tissue where the cancer begins. For instance, lung carcinoma involves the malignant transformation of epithelial cells lining the lung's airways or alveoli, while breast ductal carcinoma arises from the epithelial cells lining the milk ducts of the breast.71 The fundamental process in all carcinomas is the transformation of normal epithelial cells into malignant cells that exhibit uncontrolled growth and the potential to invade surrounding tissues and metastasize to distant sites.

While carcinomas are rare in great apes, when they do occur, they likely originate from epithelial cells in various organs, similar to the process in humans.91 However, the specific types of carcinomas observed and their frequency of occurrence appear to differ significantly between great apes and humans.93 Due to the low incidence of carcinomas in great apes, detailed research on the specific cell types and the molecular changes involved in these rare cases is limited.

**4. Influenza-A Infections**

Influenza A virus (IAV) is a significant human pathogen that causes acute respiratory infections, ranging in severity from mild, seasonal flu to severe, pandemic outbreaks.94 These viruses are classified into subtypes based on the antigenic properties of two surface glycoproteins: hemagglutinin (H) and neuraminidase (N).94

In humans, common phenotypes of influenza A infection include a sudden onset of high fever, accompanied by a cough, sore throat, muscle aches (myalgia), headache, fatigue, and a runny nose (coryza).94 In more severe cases, IAV infection can lead to serious complications such as pneumonia, acute respiratory distress syndrome (ARDS), and even death.95 The duration of viral shedding, which is indicative of infectiousness, typically peaks around the second day of illness and lasts for approximately five days.99 A key characteristic of IAV is its ability to undergo rapid genetic evolution through mechanisms known as antigenic drift and antigenic shift. These processes result in changes to the H and N proteins, allowing the virus to evade the host's acquired immunity and leading to recurrent seasonal epidemics and occasional pandemics.95 The diverse clinical presentations and the virus's evolutionary agility underscore the ongoing threat that influenza A poses to human health, necessitating continuous surveillance and the development of annually updated vaccines.

Great apes exhibit a high degree of sensitivity to infection by human respiratory viruses, including influenza A.103 Serological studies have detected evidence of past H1N1 influenza A virus infections in captive chimpanzees.103 In contrast, gorillas and orangutans have shown lower or even absent levels of antibodies against both influenza A and B viruses in some serological surveys.103 Experimental infections of chimpanzees with human influenza virus isolates have been successful in inducing infection, but the resulting responses are often mild or even asymptomatic, typically requiring high concentrations of the virus delivered directly into the lower respiratory tract to establish infection.108 One potential explanation for the reduced susceptibility to natural infection with human influenza A viruses in chimpanzees is a possible difference in the type of sialic acid receptors present in their airway cells. Human influenza A viruses preferentially bind to alpha2-6-linked sialic acid, which may be less prevalent in chimpanzees compared to humans. Conversely, avian influenza viruses bind more readily to alpha2-3-linked sialic acid.108 This suggests that the specific type of sialic acid receptor expressed on airway epithelial cells plays a crucial role in determining the host's susceptibility to different influenza virus strains.

In humans, the primary target cells for influenza viruses are the epithelial cells that line the entire respiratory tract, from the nasal passages down to the alveoli of the lungs.95 Interestingly, human-adapted influenza viruses tend to preferentially infect nonciliated epithelial cells, whereas avian influenza viruses show a tropism for ciliated cells.113 Specifically, the H1N1 subtype of influenza A virus has been shown to bind to both ciliated epithelial cells and goblet cells, which are responsible for mucus production. In contrast, the highly pathogenic H5N1 subtype more readily infects alveolar macrophages, immune cells located in the alveoli, and alveolar epithelial cells.112 The host's initial response to influenza virus infection involves the innate immune system, where pattern recognition receptors (PRRs) located on both epithelial cells and various immune cells, such as Toll-like receptors (TLRs) and retinoic acid-inducible gene-I (RIG-I), detect the presence of the virus.112 This detection triggers the expression and secretion of pro-inflammatory cytokines and type I interferons, which play a crucial role in enhancing the antiviral innate immune responses. The adaptive immune response is also critical for clearing the infection and involves the activation of CD4+ and CD8+ T cells, B cells that produce hemagglutinin-specific neutralizing antibodies, and the generation of long-lasting memory T cells that can provide protection against future infections.110

Studies on respiratory disease outbreaks in wild bonobos have identified human respiratory syncytial virus and *Streptococcus pneumoniae* as the causative agents. Histological examination of lung tissue from these bonobos revealed massive aggregates of neutrophilic granulocytes and cell debris in the alveoli and bronchial areas, along with necrosis of alveolar and bronchial epithelial cells.38 This suggests a similar pattern of cellular involvement in response to respiratory pathogens in great apes as observed in humans. Additionally, experimental infection of macaques with the highly pathogenic avian influenza A (H5N1) virus resulted in a significant infiltration of neutrophils and macrophages into the lungs of the infected animals 115, further supporting the involvement of these immune cell types in the response to influenza viruses in nonhuman primates.

**5. Hepatitis B and C Late Complications**

**Hepatitis B Late Complications**

Hepatitis B virus (HBV) infection in humans can manifest as either an acute, short-term illness or a chronic, long-lasting infection.117 While many adults who contract HBV are able to clear the virus from their bodies, a significant proportion, particularly those infected at a young age, can develop chronic HBV infection. Over the course of many years or even decades, this chronic infection can lead to severe and potentially life-threatening late complications.117 The most serious of these include cirrhosis, a condition characterized by the extensive scarring of liver tissue that impairs its function; liver failure, where the liver loses its ability to perform its vital functions; and hepatocellular carcinoma (HCC), which is a primary cancer of the liver.117 Cirrhosis can manifest with a variety of symptoms such as persistent fatigue and weakness, loss of appetite, unexplained weight loss, jaundice (yellowing of the skin and eyes), itchy skin, the accumulation of fluid in the abdomen (ascites), swelling in the legs and ankles (peripheral edema), and bleeding from enlarged veins in the esophagus or stomach (variceal bleeding).117 Liver failure can present with symptoms like hair loss, significant fluid retention leading to swelling, dark urine, frequent nosebleeds and bleeding gums, easy bruising, vomiting blood, and neurological symptoms such as confusion and altered mental state (hepatic encephalopathy).117 Hepatocellular carcinoma may cause symptoms like loss of appetite, unintentional weight loss, persistent tiredness, feeling sick to the stomach, pain or swelling in the abdomen, and jaundice.117 In addition to these liver-related complications, chronic HBV infection can also lead to extrahepatic manifestations, affecting other organs and systems in the body, including glomerulonephritis (a kidney disorder), polyarteritis nodosa (inflammation of blood vessels), as well as various dermatologic, cardiopulmonary, joint, neurologic, hematologic, and gastrointestinal issues.119

Among the nonhuman primates, chimpanzees stand out as the only immunocompetent species that is fully susceptible to infection with human hepatitis B virus (HBV), making them an invaluable animal model for studying this disease.123 Similar to human patients, chimpanzees can develop both acute and chronic HBV infections, which can lead to the development of hepatitis.123 Furthermore, chimpanzees exhibit a cellular immune response to HBV that closely mirrors the response observed in humans.123 Interestingly, HBV has also been detected in wild populations of other great ape species, including gorillas, orangutans, and gibbons.126 The prevalence of asymptomatic HBV carriers can be quite high in some of these species, reaching 23-33% in gibbons and around 15% in orangutans.126 Phylogenetic analyses have revealed that the HBV strains found in nonhuman primates are closely related to those that infect humans 125, suggesting that cross-species transmission events have likely occurred in the evolutionary history of these viruses.

In humans, the hepatitis B virus primarily targets hepatocytes, the main functional cells of the liver, for infection and replication.121 The host's immune system mounts a complex response to HBV infection, involving various types of immune cells. B cells play a crucial role by producing antibodies, including anti-HBs antibodies that target the surface antigen of the virus and are important for protection, and anti-HBc antibodies that target the core antigen.130 T cells, both CD4+ helper T cells and CD8+ cytotoxic T cells, are also critical in controlling HBV infection, with CD8+ T cells playing a key role in clearing the virus during the acute phase.130 Other immune cells involved in the response to HBV include monocytes/macrophages and natural killer (NK) cells, which contribute to both the inflammatory response and the clearance of infected cells.130 In cases of chronic HBV infection, the persistent presence of the virus can lead to immune dysfunction and a state of T cell exhaustion, where the T cells become less effective at controlling the infection.133

Similar to humans, the hepatitis B virus infects hepatocytes in great ape species that are susceptible to the virus.125 Chimpanzees, in particular, have been shown to develop a cellular immune response to HBV infection that is very similar to the response observed in human patients.123

**Hepatitis C Late Complications**

Hepatitis C virus (HCV) infection in humans can also present as an acute, short-term illness, but in a majority of cases, it progresses to become a chronic, long-term infection.134 If left untreated, chronic HCV infection can lead to significant and often irreversible damage to the liver over many years or even decades.134 The most severe late complications of chronic HCV infection include cirrhosis, a condition marked by severe scarring of the liver; liver failure, where the liver's function becomes critically impaired; and hepatocellular carcinoma (HCC), a type of liver cancer.134 The symptoms associated with these late-stage complications are often similar to those seen in the late stages of HBV infection. Cirrhosis due to HCV can cause persistent fatigue, jaundice, ascites, and other signs of liver dysfunction.135 Liver failure resulting from chronic HCV can manifest with comparable symptoms to those seen in HBV-related liver failure.136 Similarly, HCC arising from chronic HCV infection presents with symptoms that are generally indistinguishable from HCC caused by other liver diseases.136 Beyond the liver, chronic HCV infection has also been linked to several extrahepatic manifestations, affecting other organs and systems in the body. These include conditions such as diabetes mellitus, glomerulonephritis (a kidney disorder), essential mixed cryoglobulinemia (a disorder involving abnormal proteins in the blood), porphyria cutanea tarda (a skin disorder), and non-Hodgkin's lymphoma (a type of cancer affecting the lymphatic system).134

Among the great apes, chimpanzees are the only species that have been found to be experimentally susceptible to infection with the hepatitis C virus (HCV).139 However, the course of HCV infection in chimpanzees tends to differ from that in humans. Chronic infection and the development of liver fibrosis, a significant feature of chronic HCV in humans, are less common in chimpanzees.124 It remains unknown whether other great ape species, such as gorillas and orangutans, can become infected with HCV or serve as natural reservoirs for the virus.124 Interestingly, research has shown that human interferon lambda 4 (IFNL4), a protein involved in the immune response to viral infections, has evolved to have reduced antiviral activity compared to the IFNL4 protein found in chimpanzees. This difference might contribute to the higher rates of chronic HCV infection observed in humans compared to chimpanzees.142

In humans, the hepatitis C virus primarily replicates within hepatocytes, the main cells of the liver.143 However, there is also evidence suggesting that HCV can replicate in peripheral blood mononuclear cells (PBMCs), which are a type of immune cell found in the blood.143 The host's immune response to HCV involves the activation of various pattern recognition receptors, including retinoic acid-inducible gene-I (RIG-I)-like receptors and Toll-like receptors.112 These receptors recognize specific components of the virus and trigger downstream signaling pathways that lead to the production of antiviral cytokines, such as interferons. The clearance of HCV-infected hepatocytes is mediated by cytotoxic T lymphocytes (CTLs) and natural killer (NK) cells, which can directly kill the infected cells by releasing cytotoxic molecules like perforin and granzyme B.147

Similar to humans, the hepatitis C virus infects hepatocytes in chimpanzees, the only great ape species known to be susceptible to HCV infection.125

**6. Muscular Dystrophy Severity**

Muscular dystrophies (MDs) represent a diverse group of genetic disorders that are characterized by the progressive weakening and wasting (atrophy) of skeletal muscles, which are responsible for voluntary movement.148

Among the various forms of muscular dystrophy, Duchenne muscular dystrophy (DMD) stands out as a particularly severe and rapidly progressive condition. DMD typically has an onset in early childhood, usually before the age of five, and is marked by a swift decline in muscle strength. Children with DMD often become dependent on a wheelchair by their early teens, and the average life expectancy for individuals with DMD is into their twenties.148 The phenotypes of DMD include delays in achieving motor milestones such as sitting, standing, and walking, a characteristic waddling gait, walking on the toes, frequent falls, and difficulty rising from a lying or sitting position, often requiring the use of the Gowers' sign (using hands to "walk up" the legs to stand).148 Another hallmark of DMD is the pseudohypertrophy, or enlargement, of the calf muscles, which is due to the replacement of muscle tissue with fat and connective tissue.148 As the disease progresses, individuals with DMD develop muscle contractures, scoliosis (curvature of the spine), and a significant weakening of the heart muscle, leading to cardiomyopathy.148 Becker muscular dystrophy (BMD) is another form of muscular dystrophy that is closely related to DMD but is characterized by a milder course. The onset of BMD typically occurs later in childhood or adolescence, and the progression of muscle weakness is much slower. Individuals with BMD often have a longer life expectancy, sometimes living into their forties or beyond.148 The symptoms of BMD are generally similar to those of DMD but are less severe and progress at a more gradual rate.148 Besides DMD and BMD, there are numerous other types of muscular dystrophies, each with its own characteristic pattern of muscle involvement, age of onset, rate of progression, and severity. These include conditions such as myotonic dystrophy, facioscapulohumeral MD (FSHD), limb-girdle MD (LGMD), congenital MD (CMD), distal MD (DD), oculopharyngeal MD (OPMD), and Emery-Dreifuss MD (EDMD), all of which exhibit a wide range of severities.150 The severity of DMD and BMD is directly linked to the type of mutation in the gene that codes for the dystrophin protein, with mutations that disrupt the reading frame of the gene leading to the complete absence of functional dystrophin and the more severe DMD phenotype.151

Reports on muscle-related issues in great apes indicate that captive animals have been observed to exhibit muscle degeneration and atrophy.52 However, there is no direct evidence in the provided research snippets to suggest that great apes naturally suffer from muscular dystrophies with the same severity and progressive nature as seen in humans. While one source mentions that great apes do not seem to get bronchial asthma, a common human disease, and briefly notes eosinophilic airway inflammation in a monkey 17, this does not provide information about muscular dystrophy. Similarly, the finding of a chimpanzee with a disorder resembling Smith-Magenis syndrome, which includes physical, mental, and behavioral symptoms 165, is not directly relevant to muscular dystrophy. Further research may be necessary to determine if muscular dystrophy, as defined and characterized in humans, occurs in great ape species.

In humans, the primary cell type affected in muscular dystrophy is the skeletal muscle cell, also known as a myocyte or myofiber.148 In the case of DMD, the absence or severe deficiency of the dystrophin protein within these muscle fibers leads to their damage and eventual death.149 Cardiac muscle cells, or cardiomyocytes, are also affected in many forms of muscular dystrophy, including DMD and BMD, resulting in the development of cardiomyopathy, a weakening of the heart muscle.148 The body attempts to repair the damaged muscle through the action of satellite cells, which are muscle-specific stem cells that can regenerate muscle fibers. However, in muscular dystrophy, this regenerative process becomes progressively less efficient over time.149 Additionally, connective tissue cells called fibroblasts proliferate and replace the lost muscle tissue with collagen, leading to fibrosis or scarring of the muscle.149

In the context of the general muscle degeneration observed in great apes, the primary cell type affected is the muscle cell (myocyte), which shows signs of degeneration and atrophy.52 In cases of cardiac fibrosis reported in apes, both cardiomyocytes and fibroblasts are involved.52

**7. Bronchial Asthma**

Bronchial asthma is a chronic respiratory disease characterized by inflammation and narrowing of the airways in the lungs. This condition leads to recurrent episodes of wheezing, shortness of breath, chest tightness, and coughing.167 These symptoms can vary in severity and may occur intermittently.168 Asthma is now understood to be a heterogeneous condition, encompassing different phenotypes and underlying mechanisms or endotypes.167

One way to categorize asthma is by its underlying immunological mechanisms, leading to the distinction between Type 2 (T2) high asthma and T2 low asthma.167 T2 high asthma is often associated with allergies and is characterized by eosinophilic inflammation, driven by T helper 2 (Th2) cells and Type 2 innate lymphoid cells (ILC2s). Key cytokines involved in this endotype include interleukin (IL)-4, IL-5, IL-13, and the production of immunoglobulin E (IgE) antibodies.167 In contrast, T2 low asthma is characterized by non-eosinophilic inflammation, which can be neutrophilic or paucigranulocytic, and may involve different immune responses mediated by Th1 and Th17 cells.167 Asthma can also be classified based on clinical characteristics and triggers, resulting in phenotypes such as early-onset allergic asthma, late-onset asthma (developing in adulthood), aspirin-exacerbated respiratory disease (AERD), exercise-induced asthma, and obesity-related asthma.169 The diverse range of phenotypes and endotypes highlights the complexity of asthma and the need for personalized approaches to diagnosis and treatment.

The evidence regarding bronchial asthma susceptibility in great apes is not entirely clear. While one source suggests that great apes do not seem to develop this condition, which is common in humans 17, it also mentions the occurrence of eosinophilic airway inflammation in a monkey. Furthermore, great apes are known to be susceptible to respiratory infections caused by human viruses 103, which can sometimes exacerbate or mimic asthma symptoms. Researchers have also developed experimental models of asthma in nonhuman primates, such as rhesus monkeys, which share many of the key features of human allergic asthma, including allergen-specific IgE production and airway eosinophilia.182 This suggests that while the full spectrum of human bronchial asthma might not be present in great apes, they may possess some susceptibility to asthma-like conditions or certain aspects of the disease. More research is needed to fully understand the prevalence and characteristics of asthma in these species.

In humans, the pathogenesis of bronchial asthma involves a complex interaction of various cell types within the airways. Epithelial cells, which line the airways, play a crucial role in initiating inflammation, producing mucus, and releasing cytokines such as TSLP, IL-25, and IL-33.178 Goblet cells, a type of epithelial cell, increase the production of mucus, contributing to airway obstruction.177 In contrast, the function of ciliated epithelial cells, responsible for clearing mucus, may be reduced in asthmatic airways.177 Various immune cells are also central to asthma. Th2 cells produce key cytokines like IL-4, IL-5, and IL-13, driving allergic inflammation.167 ILC2s also contribute to this process by producing IL-5 and IL-13.167 Mast cells release inflammatory mediators such as histamine, leukotrienes, and prostaglandin D2.170 Eosinophils are recruited to the airways and release a variety of inflammatory mediators.167 In certain phenotypes of asthma, such as neutrophilic asthma, neutrophils also play a significant role.168 Other immune cells involved in asthma include T regulatory cells, which help to control inflammation 190, and CD8+ T cells, whose role is still being investigated.191 Structural cells within the airways also contribute to asthma. Airway smooth muscle cells contract excessively, leading to bronchoconstriction 178, while fibroblasts are involved in the remodeling of the airway walls, leading to thickening and stiffening.185

Studies on experimentally induced asthma in monkeys have shown an increased presence of various immune cells within the airways following allergen challenge. These include CD1a+ dendritic cells, CD4+ T helper lymphocytes, CD25+ activated cells, IgE+ cells, eosinophils, and proliferating cells.184 This suggests that the major cell types involved in allergic asthma are largely conserved between humans and other primates.

**8. Preeclampsia**

Preeclampsia is a pregnancy-specific disorder characterized by the new onset of high blood pressure, typically occurring after 20 weeks of gestation, and often accompanied by the presence of protein in the urine or signs of other organ damage.78

Key phenotypes of preeclampsia include elevated blood pressure, defined as a systolic reading of 140 mmHg or higher or a diastolic reading of 90 mmHg or higher, proteinuria, which is an abnormal amount of protein in the urine (≥0.3 grams in a 24-hour period), and edema, or swelling, particularly in the face and hands.78 Other common symptoms can include persistent headaches, visual disturbances such as blurred vision or seeing spots, and pain in the upper abdomen.78 In severe cases of preeclampsia, additional phenotypes may emerge, including a significant increase in blood pressure (systolic ≥160 mmHg or diastolic ≥110 mmHg), signs of impaired liver function, a low platelet count (thrombocytopenia), evidence of kidney dysfunction, fluid accumulation in the lungs (pulmonary edema), and neurological symptoms like seizures.193 Preeclampsia can be broadly categorized based on the gestational age at which it occurs. Early-onset preeclampsia, developing before 34 weeks of pregnancy, is often associated with more severe placental dysfunction and may lead to fetal growth restriction. Late-onset preeclampsia, occurring at or after 34 weeks, is more commonly linked to underlying maternal health factors.194 While hypertension and proteinuria have historically been considered essential for a preeclampsia diagnosis, current understanding acknowledges that end-organ damage in the presence of hypertension can also be indicative of the disorder, even in the absence of significant proteinuria.78

Recent evidence has challenged the long-held belief that preeclampsia is a uniquely human condition, with observations in chimpanzees and gorillas suggesting that these great apes might also be susceptible.201 The placenta in humans and other great apes exhibits a deep level of extravillous trophoblast (EVT) invasion and remodeling of the spiral arteries in the uterus, a characteristic that is abnormal in human preeclampsia. This feature distinguishes the Homininae subfamily (humans, chimpanzees, gorillas) from other primates like gibbons and Old World monkeys, which have shallower trophoblast invasion.201 It has been hypothesized that the evolution of this more invasive placenta in the ancestors of great apes involved positive selection on genes crucial for EVT invasion and spiral artery remodeling, potentially increasing the risk of developing preeclampsia.201

The pathogenesis of preeclampsia in humans involves a complex interplay of various cell types, with a central role played by trophoblast cells in the placenta. Abnormal invasion and differentiation of cytotrophoblasts, a type of trophoblast cell, into the maternal uterine vasculature are considered a key initiating event.194 This inadequate placental development is thought to lead to placental ischemia and the release of factors that cause systemic endothelial dysfunction, a hallmark of preeclampsia.193 Various immune cells, including regulatory T cells, macrophages (with an imbalance between pro-inflammatory M1 and anti-inflammatory M2 types), natural killer cells, neutrophils, and B cells, are also implicated in the pathology of preeclampsia.203 In the kidneys, a characteristic lesion known as glomerular endotheliosis, involving swelling of the endothelial cells in the glomeruli, is often observed in preeclampsia.195

Research on preeclampsia in great apes is still in its early stages. One study using a baboon model of experimentally induced preeclampsia has shown involvement of endothelial cells and placental tissue.212

**9. Bipolar Disorders**

Bipolar disorder (BD), previously known as manic depression, is a chronic mental illness characterized by significant shifts in mood, energy, activity levels, concentration, and the ability to carry out day-to-day tasks. Individuals with BD experience recurrent episodes of intensely elevated mood (mania or a less severe form called hypomania) and periods of significantly depressed mood.214

During a manic episode, individuals may exhibit an abnormally elevated, expansive, or irritable mood that lasts for at least one week. This period is often accompanied by a marked increase in energy and activity levels, racing thoughts, rapid and pressured speech, a decreased need for sleep, an inflated sense of self-esteem or grandiosity, impulsive behavior, and sometimes psychotic symptoms such as delusions or hallucinations.214 Hypomania involves similar but less severe manic symptoms that do not include psychosis or cause significant impairment in social or occupational functioning.214 In contrast, depressive episodes in BD are characterized by a persistent sad, empty, or hopeless mood, a loss of interest or pleasure in activities, significant changes in appetite and sleep patterns, fatigue or loss of energy, feelings of worthlessness or excessive guilt, difficulty thinking or concentrating, and recurrent thoughts of death or suicide.214 There are several recognized types of bipolar disorder, including Bipolar I disorder, which involves at least one manic episode (and often depressive episodes); Bipolar II disorder, characterized by hypomanic and depressive episodes but no full manic episodes; and Cyclothymic disorder, which involves less severe but chronic fluctuations between hypomanic and depressive symptoms.214 The severity and specific presentation of bipolar disorder can vary considerably from person to person.

While bipolar disorder is considered a condition that primarily affects humans, research in primate genetics has identified specific genetic variations associated with an increased risk of bipolar disorder in humans.222 Notably, a specific protein isoform called PDE10A19, which is found only in primates and not in other commonly studied animal models like mice or rats, might play a role in the susceptibility to bipolar disorder.222 Some evolutionary hypotheses suggest that the origins of bipolar disorder in humans might be linked to adaptations related to seasonal changes and the regulation of the biological clock.224 The fact that bipolar disorder has not been diagnosed in great apes so far 225 might be due to fundamental differences in brain structure and function between humans and apes, or it could reflect the challenges in recognizing and diagnosing complex psychiatric conditions in non-human species.

Neuroimaging studies in humans with bipolar disorder have consistently implicated abnormalities in several brain regions that are involved in mood regulation, cognition, and emotion processing. These regions include the prefrontal cortex (particularly the ventral, medial, and dorsolateral areas), the amygdala, the hippocampus, the striatum, and the thalamus.227 At a cellular level, specific types of neurons within these regions appear to be particularly affected. These include GABAergic interneurons and medium spiny neurons, especially in the prefrontal cortex and hippocampus.227 Additionally, some studies have suggested the involvement of excitatory neurons and caudal ganglionic eminence interneurons.236 Interestingly, research has also pointed to potential involvement of cells in the intestine and pancreas in bipolar disorder, although the precise role of these cells is not yet well understood.227

In the brains of great apes, specialized types of neurons have been identified that are also found in humans and are thought to be involved in social cognition and emotional processing. One such type is the Von Economo neuron (VEN), which is a large, bipolar neuron located in the anterior cingulate and frontoinsular cortices of great apes and humans.237 Additionally, a primate-specific type of interneuron has been discovered in the striatum, a brain structure involved in cognition, reward, and movement.240 The presence of these specialized neuron types in great apes, particularly in brain regions that are implicated in human bipolar disorder, suggests that while apes may not exhibit the full spectrum of the human condition, they do possess some of the neural substrates that are potentially involved in mood regulation and social behavior.

**10. Schizophrenia**

Schizophrenia is a chronic and severe mental disorder that affects a person's ability to think, feel, and behave clearly. It is characterized by a range of symptoms that can include hallucinations (often auditory, such as hearing voices), delusions (false beliefs), disorganized thinking and speech, and negative symptoms such as blunted affect, reduced speech, lack of motivation, and social withdrawal.226 The onset of schizophrenia typically occurs during late adolescence or early adulthood.242

The symptoms of schizophrenia are often categorized into positive, negative, and cognitive symptoms. Positive symptoms include experiences that are not typically present in healthy individuals, such as hallucinations, delusions, and disorganized thoughts, which can manifest as incoherent speech or bizarre behavior.243 Negative symptoms refer to a reduction or absence of normal behaviors and feelings, such as blunted affect (reduced emotional expression), alogia (poverty of speech), anhedonia (inability to experience pleasure), asociality (lack of interest in social interactions), and avolition (lack of motivation).243 Cognitive deficits are also a core feature of schizophrenia and can include impairments in working memory, attention, executive functions (such as planning and problem-solving), and processing speed.245 The combination and severity of these symptoms can vary widely among individuals with schizophrenia, reflecting the heterogeneous nature of this complex disorder.

Schizophrenia appears to be a uniquely human condition, as it has not been diagnosed in great apes or other animal species to date.225 While one study reported the accidental finding of a genetic abnormality in a chimpanzee that resembled Smith-Magenis syndrome, a disorder with some overlapping symptoms in the behavioral domain 165, this is distinct from schizophrenia. Some evolutionary hypotheses propose that the development of schizophrenia in humans might be linked to the evolution of advanced human cognitive abilities, particularly the capacity for complex language.253 The absence of schizophrenia in our closest evolutionary relatives, despite their complex social behaviors and cognitive capacities, suggests that specific evolutionary changes in the human lineage may have conferred a susceptibility to this disorder.

Neuroimaging studies in humans with schizophrenia have identified structural and functional abnormalities in several brain regions, including the prefrontal cortex, temporal lobe, hippocampus, thalamus, amygdala, and basal ganglia.229 Genetic studies, particularly genome-wide association studies (GWAS), have implicated numerous genes and genomic loci in the risk for schizophrenia. By integrating these genetic findings with detailed maps of gene expression in different brain cell types, researchers have identified specific types of neurons that appear to be particularly relevant to the disorder. These include pyramidal cells, medium spiny neurons (MSNs), and certain types of interneurons.234 Additionally, some studies have suggested the involvement of excitatory neurons and caudal ganglionic eminence interneurons.236 Oligodendrocytes, which are responsible for producing myelin that insulates nerve fibers, have also been shown to exhibit altered patterns of gene expression and function in humans with schizophrenia compared to other primates, suggesting a potential role for these non-neuronal cells in the disorder.269

Great apes, including chimpanzees, gorillas, orangutans, and bonobos, possess specialized types of neurons in their brains that are also found in humans and are thought to be involved in higher-level cognitive and emotional functions. One such type is the Von Economo neuron (VEN), which is located in the anterior cingulate and frontoinsular cortices and is implicated in social cognition, empathy, and intuition.237 Additionally, a novel type of interneuron that is specific to primates has been discovered in the striatum, a brain region involved in motor control, cognition, and reward.240 The presence of these specialized neuronal types in great apes, particularly in brain regions that are also implicated in human schizophrenia, suggests that while the full disorder may be uniquely human, some of the underlying neural components and evolutionary changes in brain circuitry might be shared with other primates.

**Conclusions**

The analysis of these ten human-specific diseases reveals a complex landscape of conditions with varying degrees of host specificity and cellular involvement. Meningococcal meningitis, while caused by a bacterium that colonizes humans, shows a significantly lower susceptibility in great apes, potentially due to human-specific immune interactions and social behaviors. Myocardial infarction, primarily driven by atherosclerosis in humans, is rare in great apes, where heart disease more commonly manifests as myocardial fibrosis, suggesting fundamental differences in cardiovascular pathophysiology. Epithelial cancers (carcinomas), a major cause of mortality in humans, are notably less frequent in great apes, hinting at protective mechanisms or different evolutionary pressures. Influenza-A infections, while capable of infecting great apes, often present with milder symptoms compared to humans, possibly due to variations in viral receptor distribution. Hepatitis B and C, both capable of causing chronic liver disease and late complications in humans, show different patterns of susceptibility and disease progression in great apes, with chimpanzees serving as important research models. Muscular dystrophy, a group of genetic disorders causing progressive muscle weakness in humans, has not been definitively documented with similar severity in great apes. Bronchial asthma, a common inflammatory airway disease in humans, appears to be rare or absent in great apes. Preeclampsia, a pregnancy-specific hypertensive disorder, may not be entirely human-specific, with some evidence suggesting susceptibility in other great apes. Bipolar disorders and schizophrenia, complex psychiatric conditions with significant human prevalence, have not been diagnosed in great apes, suggesting a potential link to uniquely human aspects of brain development and function.

The cellular mechanisms underlying these diseases in humans often involve a complex interplay between pathogen invasion (where applicable), host immune responses, and the dysfunction of specific cell types within the affected organs or systems. In contrast, the cellular involvement in great apes, particularly for diseases that show differential susceptibility, is less well-defined, often due to the rarity of the human-specific pathogen in these animals or the absence of the full spectrum of the human disease. Further research, especially using comparative genomics, proteomics, and detailed physiological studies in great apes, may provide valuable insights into the evolutionary origins of these human-specific diseases and the factors that contribute to the observed differences in susceptibility and cellular involvement.

**Table 1: Comparison of Heart Disease in Humans and Great Apes**

| **Feature** | **Humans** | **Great Apes (Chimpanzees, Gorillas, Orangutans, Bonobos)** |
| --- | --- | --- |
| Primary Heart Disease Type | Myocardial Infarction (Heart Attack) | Interstitial Myocardial Fibrosis (IMF) |
| Common Underlying Pathology | Coronary Artery Atherosclerosis | Idiopathic Myocardial Fibrosis |
| Key Risk Factors | Hypercholesterolemia, Hypertension, Smoking, Diabetes | Unknown, potentially dietary, inactivity, age, genetics |
| Typical Clinical Presentation | Chest pain, Shortness of breath, Sudden Death | Sudden Cardiac Death, Progressive Heart Failure |
| Prevalence | Common | Common (IMF), MI Rare |

**Table 2: Summary of Major Asthma Endotypes (Human)**

| **Endotype** | **Key Inflammatory Cells** | **Major Cytokines Involved** | **Typical Triggers/Associations** |
| --- | --- | --- | --- |
| T2-high (Eosinophilic, Allergic) | Th2 cells, ILC2s, Eosinophils, Mast cells | IL-4, IL-5, IL-13, IgE | Allergens |
| T2-high (Eosinophilic, Non-Allergic) | Th2 cells, ILC2s, Eosinophils, Mast cells | IL-4, IL-5, IL-13 | Unknown |
| T2-low (Neutrophilic) | Neutrophils, Th17 cells | IL-17, IL-8, TNF-α | Smoking, Obesity, Air Pollution, Infections |
| T2-low (Paucigranulocytic/Minimal Inflammation) | Few inflammatory cells | Low levels of various cytokines | Often milder, may be triggered by irritants |

**Table 3: Brain Regions and Neuron Types Implicated in Bipolar Disorder and Schizophrenia (Human)**

| **Disorder** | **Brain Region(s)** | **Neuron Type(s)** |
| --- | --- | --- |
| Bipolar Disorder | Prefrontal Cortex, Amygdala, Hippocampus, Striatum, Thalamus | GABAergic Interneurons, Medium Spiny Neurons |
| Schizophrenia | Prefrontal Cortex, Temporal Lobe, Hippocampus, Thalamus, Amygdala, Basal Ganglia | Pyramidal Cells, Medium Spiny Neurons, Interneurons, Oligodendrocytes |

#### עבודות שצוטטו

1. Meningitis: Causes, Symptoms, and Treatment | Doctor - Patient.info, נרשמה גישה בתאריך אפריל 20, 2025, <https://patient.info/doctor/meningitis-pro>
2. Neisseria meningitidis - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Neisseria_meningitidis>
3. Meningococcal meningitis, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.meningitis.org/meningitis/bacterial-meningitis/meningococcal-meningitis>
4. Invasive Meningococcal Disease| Neisseria meningitidis Laboratory ..., נרשמה גישה בתאריך אפריל 20, 2025, <https://www.health.gov.au/sites/default/files/2025-02/meningococcal-disease-laboratory-case-definition.pdf>
5. About Meningococcal Disease | Meningococcal | CDC, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.cdc.gov/meningococcal/about/index.html>
6. Meningococcal Disease Fact Sheet - New York State Department of Health, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.health.ny.gov/publications/2168/>
7. Human genetics of meningococcal infections - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC7272491/>
8. Clinical Overview of Meningococcal Disease - CDC, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.cdc.gov/meningococcal/hcp/clinical/index.html>
9. Factsheet about meningococcal disease - ECDC - European Union, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ecdc.europa.eu/en/meningococcal-disease/factsheet>
10. Meningococcal Meningitis - Center for Academic Research and Training in Anthropogeny (CARTA), נרשמה גישה בתאריך אפריל 20, 2025, <https://carta.anthropogeny.org/moca/topics/meningococcal-meningitis>
11. Meningitis, Encephalitis, and Encephalomyelitis in Animals - Nervous System - Merck Veterinary Manual, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.merckvetmanual.com/nervous-system/meningitis-encephalitis-and-encephalomyelitis/meningitis-encephalitis-and-encephalomyelitis-in-animals>
12. Clinicopathologic study of six cases of meningitis and meningoencephalitis in chimpanzees (Pan troglodytes) - PubMed, נרשמה גישה בתאריך אפריל 20, 2025, <https://pubmed.ncbi.nlm.nih.gov/6371376/>
13. CASE REPORT Eosinophilic meningoencephalomyelitis in an orangutan (Pongo pygmaeus) caused by Angiostrongylus cantonensis - Taylor and Francis, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.tandfonline.com/doi/pdf/10.1080/01652176.2013.880005>
14. Animal models for pathogenic Neisseria species - ASM Journals, נרשמה גישה בתאריך אפריל 20, 2025, <https://journals.asm.org/doi/pdf/10.1128/CMR.2.Suppl.S56>
15. Antimicrobial Resistance in African Great Apes - MDPI, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/2079-6382/13/12/1140>
16. Best Practice Guidelines for Health Monitoring and Disease Control in Great Ape Populations - IUCN Portals, נרשמה גישה בתאריך אפריל 20, 2025, <https://portals.iucn.org/library/sites/library/files/documents/ssc-op-056.pdf>
17. How Are Humans Different from Other Great Apes? - American Academy of Arts and Sciences, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.amacad.org/news/how-are-humans-different-other-great-apes>
18. Genetic Differences between Humans and Great Apes - Department of Cellular & Molecular Medicine, נרשמה גישה בתאריך אפריל 20, 2025, <https://cmm.ucsd.edu/research/labs/varki/_files/publications/b067.pdf>
19. Preventative Vaccination of Nonhuman Primates - MDPI, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/2673-5636/6/1/8>
20. Comparative physiological anthropogeny: exploring molecular underpinnings of distinctly human phenotypes, נרשמה גישה בתאריך אפריל 20, 2025, <https://journals.physiology.org/doi/full/10.1152/physrev.00040.2021>
21. Commissioned Paper: Comparison of Immunity to Pathogens in Humans, Chimpanzees, and Macaques - NCBI, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ncbi.nlm.nih.gov/books/NBK91442/>
22. Functional Comparison of Innate Immune Signaling Pathways in Primates | PLOS Genetics, נרשמה גישה בתאריך אפריל 20, 2025, <https://journals.plos.org/plosgenetics/article?id=10.1371/journal.pgen.1001249>
23. Innate immune recognition and inflammation in Neisseria meningitidis infection | Pathogens and Disease | Oxford Academic, נרשמה גישה בתאריך אפריל 20, 2025, <https://academic.oup.com/femspd/article/75/2/ftx022/3059204>
24. Marmosets as models of infectious diseases - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/cellular-and-infection-microbiology/articles/10.3389/fcimb.2024.1340017/full>
25. Opportunistic Infections in Immunologically Compromised Nonhuman Primates | ILAR Journal | Oxford Academic, נרשמה גישה בתאריך אפריל 20, 2025, <https://academic.oup.com/ilarjournal/article/49/2/191/674955>
26. New Streptococcus pneumoniae Clones in Deceased Wild Chimpanzees - ASM Journals, נרשמה גישה בתאריך אפריל 20, 2025, <https://journals.asm.org/doi/10.1128/jb.00468-07>
27. Comparative models for human nasal infections and immunity - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC7102639/>
28. Identifying Infectious Hazards Associated with the Use of Nonhuman Primates in Research, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ncbi.nlm.nih.gov/books/NBK43452/>
29. Neisseria meningitidis: Infectious substances pathogen safety data sheet - Canada.ca, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.canada.ca/en/public-health/services/laboratory-biosafety-biosecurity/pathogen-safety-data-sheets-risk-assessment/neisseria-meningitidis.html>
30. Travel Health Alerts | Travelvax, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.travelvax.com.au/travel-health-alerts?page=180>
31. Meningococcal Meningitis - WHO | Regional Office for Africa, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.afro.who.int/health-topics/meningococcal-meningitis>
32. Chapter 14: Meningococcal Disease | Pink Book - CDC, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.cdc.gov/pinkbook/hcp/table-of-contents/chapter-14-meningococcal-disease.html>
33. Comprehensive Identification of Meningococcal Genes and Small Noncoding RNAs Required for Host Cell Colonization | mBio - ASM Journals, נרשמה גישה בתאריך אפריל 20, 2025, <https://journals.asm.org/doi/10.1128/mbio.01173-16>
34. Molecular mechanisms involved in the interaction of Neisseria meningitidis with cells of the human blood–cerebrospinal fluid barrier - Oxford Academic, נרשמה גישה בתאריך אפריל 20, 2025, <https://academic.oup.com/femspd/article/75/2/ftx023/3061359>
35. Cellular and molecular biology of Neisseria meningitidis colonization and invasive disease, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC2830671/>
36. Meningococcal Disease (Neisseria meningitidis Infection) - StatPearls - NCBI Bookshelf, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ncbi.nlm.nih.gov/books/NBK549849/>
37. Single-Cell Dissection of the Immune Response After Acute Myocardial Infarction | Circulation: Genomic and Precision Medicine - American Heart Association Journals, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ahajournals.org/doi/10.1161/CIRCGEN.123.004374>
38. Human Respiratory Syncytial Virus and Streptococcus pneumoniae Infection in Wild Bonobos - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC7087961/>
39. Myocardial infarction - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Myocardial_infarction>
40. Heart disease is common in humans and chimpanzees, but is ..., נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC3352420/>
41. Sex Differences in Symptom Phenotypes Among Patients With Acute Myocardial Infarction | Circulation: Cardiovascular Quality and Outcomes - American Heart Association Journals, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ahajournals.org/doi/10.1161/CIRCOUTCOMES.119.005948>
42. Myocardial Infarction - StatPearls - NCBI Bookshelf, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ncbi.nlm.nih.gov/books/NBK537076/>
43. Clinical phenotypes in acute and chronic infarction explained ... - eLife, נרשמה גישה בתאריך אפריל 20, 2025, <https://elifesciences.org/reviewed-preprints/93002>
44. Clinical phenotypes in acute and chronic infarction explained ..., נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC11668532/>
45. Clinical phenotypes in acute and chronic infarction explained through human ventricular electromechanical modelling and simulations | bioRxiv, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.biorxiv.org/content/10.1101/2022.02.15.480392v2.full-text>
46. Clinical phenotypes in acute and chronic infarction explained through human ventricular electromechanical modelling and simulations | bioRxiv, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.biorxiv.org/content/10.1101/2022.02.15.480392v3>
47. Examples of histological comparisons of human and chimpanzee hearts and coronary blood vessels. Top panels - ResearchGate, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/figure/Examples-of-histological-comparisons-of-human-and-chimpanzee-hearts-and-coronary-blood_fig4_227533110>
48. Examples of prior publications describing unexplained myocardial fibrosis in chimpanzees and other great apes. - ResearchGate, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/figure/Examples-of-prior-publications-describing-unexplained-myocardial-fibrosis-in-chimpanzees_tbl1_227533110>
49. Heart disease is common in humans and chimpanzees, but is caused by different pathological processes, נרשמה גישה בתאריך אפריל 20, 2025, <https://cmm.ucsd.edu/research/labs/varki/_files/publications/a173.pdf>
50. Getting to the heart of cardiovascular evolution in humans - PMC, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC6538371/>
51. Cardiovascular Disease in Great Apes | Veterian Key, נרשמה גישה בתאריך אפריל 20, 2025, <https://veteriankey.com/cardiovascular-disease-in-great-apes/>
52. Pathology and Diseases of Great Apes at the National Zoological Park - Smithsonian Institution, נרשמה גישה בתאריך אפריל 20, 2025, <https://repository.si.edu/bitstream/handle/10088/11628/Munson1990.pdf?sequence=1&isAllowed=y>
53. An overview of nutritional factors in the etiopathogenesis of myocardial fibrosis in great apes: A new role of vitamin D? - Ghent University Library, נרשמה גישה בתאריך אפריל 20, 2025, <https://libstore.ugent.be/fulltxt/RUG01/002/838/065/RUG01-002838065_2020_0001_AC.pdf>
54. A retrospective review of great ape cardiovascular disease epidemiology and pathology, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/publication/325056529_A_retrospective_review_of_great_ape_cardiovascular_disease_epidemiology_and_pathology>
55. Great ape cardiovascular disease: aetiopathogenesis, risk factors and diagnostic tools - - Nottingham ePrints, נרשמה גישה בתאריך אפריל 20, 2025, <https://eprints.nottingham.ac.uk/65647/1/Thesis%20SM%20final2.pdf>
56. A retrospective review of great ape cardiovascular disease epidemiology and pathology V. J. STRONG1, M. MARTIN2, S. REDROBE3, K - CORE, נרשמה גישה בתאריך אפריל 20, 2025, <https://core.ac.uk/download/pdf/157770258.pdf>
57. Interstitial Myocardial Fibrosis in a Captive Chimpanzee (Pan troglodytes) Population - PMC, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC2706041/>
58. An overview of nutritional factors in the aetiopathogenesis of myocardial fibrosis in great apes - Cambridge University Press, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.cambridge.org/core/journals/nutrition-research-reviews/article/an-overview-of-nutritional-factors-in-the-aetiopathogenesis-of-myocardial-fibrosis-in-great-apes/53F814AD5283423B8DF231BF49A41369>
59. Fatal myocardial fibrosis in an aged chimpanzee (Pan troglodytes) - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC3679521/>
60. Full article: Fatal myocardial fibrosis in an aged chimpanzee (Pan troglodytes), נרשמה גישה בתאריך אפריל 20, 2025, <https://www.tandfonline.com/doi/full/10.3402/pba.v3i0.21073>
61. Conference 18 - 2013 Case: 03 20140312, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.askjpc.org/wsco/wsc_showcase2.php?id=UHZoS0tDUXI1NndpemRpTHpaYlpYQT09>
62. Brain-type natriuretic peptide is a useful biomarker of cardiovascular disease and predictor of cardiac-related mortality in chimpanzees (Pan troglodytes) in - AVMA Journals, נרשמה גישה בתאריך אפריל 20, 2025, <https://avmajournals.avma.org/view/journals/ajvr/86/3/ajvr.24.09.0287.xml>
63. Cardiomyocytes Cellular Phenotypes After Myocardial Infarction - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/cardiovascular-medicine/articles/10.3389/fcvm.2021.750510/full>
64. Pathophysiological Effects of Various Interleukins on Primary Cell ..., נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/1422-0067/24/7/6497>
65. The Biological Basis for Cardiac Repair After Myocardial Infarction ..., נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ahajournals.org/doi/10.1161/circresaha.116.303577>
66. Macrophages in myocardial infarction - American Journal of Physiology, נרשמה גישה בתאריך אפריל 20, 2025, <https://journals.physiology.org/doi/full/10.1152/ajpcell.00230.2022>
67. The role of major immune cells in myocardial infarction - PMC, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC9892933/>
68. The inflammatory response in myocardial infarction - Oxford Academic, נרשמה גישה בתאריך אפריל 20, 2025, <https://academic.oup.com/cardiovascres/article/53/1/31/433387>
69. Cells of the Immune System in Cardiac Remodeling: Main Players in Resolution of Inflammation and Repair After Myocardial Infarction - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2021.664457/full>
70. Epithelial-Mesenchymal Transition: A Cancer Researcher's Conceptual Friend and Foe - PMC, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC2671246/>
71. Carcinoma: Types, Treatment & What it Is - Cleveland Clinic, נרשמה גישה בתאריך אפריל 20, 2025, <https://my.clevelandclinic.org/health/diseases/23180-carcinoma>
72. Types of cancer - Cancer Research UK, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.cancerresearchuk.org/about-cancer/what-is-cancer/how-cancer-starts/types-of-cancer>
73. Cancer Classification - News-Medical.net, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.news-medical.net/health/Cancer-Classification.aspx>
74. Types of Carcinoma - Cancer - Verywell Health, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.verywellhealth.com/carcinoma-5092211>
75. Cancer Classification - SEER Training Modules, נרשמה גישה בתאריך אפריל 20, 2025, <https://training.seer.cancer.gov/disease/categories/classification.html>
76. Carcinoma - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Carcinoma>
77. Revisiting Epithelial Carcinogenesis - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC9267463/>
78. Polycystic ovary syndrome - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Polycystic_ovary_syndrome>
79. Epithelial-Mesenchymal Transitions in Human Cancer - Madame Curie Bioscience Database - NCBI Bookshelf, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ncbi.nlm.nih.gov/books/NBK6362/>
80. Cancer Stem Cells and Epithelial-to-Mesenchymal Transition (EMT)-Phenotypic Cells: Are They Cousins or Twins? - MDPI, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/2072-6694/3/1/716>
81. DCTD Scientists Develop a Clinical Monitoring Tool for Epithelial-Mesenchymal Phenotype, נרשמה גישה בתאריך אפריל 20, 2025, <https://dctd.cancer.gov/NewsEvents/20200116_Epithelial-Mesenchymal_Phenotype.htm>
82. Hypoxic Conditions Induce a Cancer-Like Phenotype in Human Breast Epithelial Cells, נרשמה גישה בתאריך אפריל 20, 2025, <https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0046543>
83. On the apparent rarity of epithelial cancers in captive chimpanzees - PMC, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC4581030/>
84. On the apparent rarity of epithelial cancers in captive chimpanzees - PubMed, נרשמה גישה בתאריך אפריל 20, 2025, <https://pubmed.ncbi.nlm.nih.gov/26056369/>
85. On the apparent rarity of epithelial cancers in captive chimpanzees | Philosophical Transactions of the Royal Society B: Biological Sciences - Journals, נרשמה גישה בתאריך אפריל 20, 2025, <https://royalsocietypublishing.org/doi/abs/10.1098/rstb.2014.0225>
86. (PDF) Comparative analysis of cancer genes in the human and chimpanzee genomes, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/publication/7334379_Comparative_analysis_of_cancer_genes_in_the_human_and_chimpanzee_genomes>
87. Low Cancer Rates in Nonhuman primates - Macroevolution.net, נרשמה גישה בתאריך אפריל 20, 2025, <http://www.macroevolution.net/cancer-in-nonhuman-primates.html>
88. Cancer Genes in Humans vs. Chimps: Why Are We More Susceptible?, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mskcc.org/news/cancer-genes-humans-vs-chimps-why-are-we-more-susceptible>
89. Primary Human Cancer Associated Epithelial Cells - BioIVT, נרשמה גישה בתאריך אפריל 20, 2025, <https://bioivt.com/primary-human-cancer-associated-epithelial-cells>
90. What Is Cancer? - NCI, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.cancer.gov/about-cancer/understanding/what-is-cancer>
91. Proportion of different types of cancer in male and female non-human... - ResearchGate, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/figure/Proportion-of-different-types-of-cancer-in-male-and-female-non-human-primates-Created_fig2_324565833>
92. An Examination of Chimpanzee Use in Human Cancer Research - WBI Studies Repository, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.wellbeingintlstudiesrepository.org/cgi/viewcontent.cgi?article=1028&context=acwp_lab>
93. An Examination of Chimpanzee Use in Human Cancer Research - Cruelty Free International, נרשמה גישה בתאריך אפריל 20, 2025, <https://crueltyfreeinternational.org/sites/default/files/2021-10/Bailey_%20chimpanzees_cancer_ATLA_2009.pdf>
94. Influenza A virus - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Influenza_A_virus>
95. Human Influenza Virus Infections - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC7174870/>
96. Types of Influenza Viruses - CDC, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.cdc.gov/flu/about/viruses-types.html>
97. Influenza - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Influenza>
98. The Pathology of Influenza Virus Infections - PMC, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC2504709/>
99. Time Lines of Infection and Disease in Human Influenza: A Review of Volunteer Challenge Studies - Oxford Academic, נרשמה גישה בתאריך אפריל 20, 2025, <https://academic.oup.com/aje/article/167/7/775/83777>
100. Influenza A genomic diversity during human infections underscores the strength of genetic drift and the existence of tight transmission bottlenecks | Virus Evolution | Oxford Academic, נרשמה גישה בתאריך אפריל 20, 2025, <https://academic.oup.com/ve/article/10/1/veae042/7686346>
101. Full article: Influenza virus genotype to phenotype predictions through machine learning: a systematic review - Taylor & Francis Online, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.tandfonline.com/doi/full/10.1080/22221751.2021.1978824>
102. Deep Sequencing of Influenza A Virus from a Human Challenge Study Reveals a Selective Bottleneck and Only Limited Intrahost Genetic Diversification, נרשמה גישה בתאריך אפריל 20, 2025, <https://journals.asm.org/doi/10.1128/jvi.01657-16>
103. (PDF) Retrospective Serology Study of Respiratory Virus Infections in Captive Great Apes, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/publication/261067820_Retrospective_Serology_Study_of_Respiratory_Virus_Infections_in_Captive_Great_Apes>
104. Retrospective Serology Study of Respiratory Virus Infections in Captive Great Apes - MDPI, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/1999-4915/6/3/1442>
105. (PDF) Pediatric Respiratory Pathogens Circulate in Children and Adults in Communities Near Susceptible Wild Great Ape Populations in Uganda - ResearchGate, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/publication/385203085_Pediatric_Respiratory_Pathogens_Circulate_in_Children_and_Adults_in_Communities_Near_Susceptible_Wild_Great_Ape_Populations_in_Uganda>
106. Great apes and COVID-19: Experts raise the alarm for endangered species, נרשמה גישה בתאריך אפריל 20, 2025, <https://news.emory.edu/stories/2020/04/esc_covid_19_great_apes/campus.html>
107. Could COVID-19 impact great apes? - Medical News Today, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.medicalnewstoday.com/articles/great-apes-covid>
108. Influenza A Infection | Center for Academic Research and Training in Anthropogeny (CARTA), נרשמה גישה בתאריך אפריל 20, 2025, <https://carta.anthropogeny.org/moca/topics/influenza-infection>
109. Influenza Virus Receptor Specificity and Cell Tropism in Mouse and Human Airway Epithelial Cells | Journal of Virology - ASM Journals, נרשמה גישה בתאריך אפריל 20, 2025, <https://journals.asm.org/doi/10.1128/jvi.02677-05>
110. Evasion of Influenza A Viruses from Innate and Adaptive Immune Responses - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC3499814/>
111. Comprehensive single cell analysis of pandemic influenza A virus infection in the human airways uncovers cell-type specific host transcriptional signatures relevant for disease progression and pathogenesis - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2022.978824/full>
112. Immune Response to Influenza A Virus - Bio-Rad Antibodies, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.bio-rad-antibodies.com/influenza-immune-response.html>
113. Human and avian influenza viruses target different cell types in cultures of human airway epithelium | PNAS, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.pnas.org/doi/10.1073/pnas.0308001101>
114. Host Immune Response to Influenza A Virus Infection - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2018.00320/full>
115. Massive Mobilization of Dendritic Cells During Influenza A Virus Subtype H5N1 Infection of Nonhuman Primates - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC4038140/>
116. Pathogenesis of Influenza A (H5N1) Virus Infection in a Primate Model - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC114392/>
117. Hepatitis B - Symptoms and causes - Mayo Clinic, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mayoclinic.org/diseases-conditions/hepatitis-b/symptoms-causes/syc-20366802>
118. Hepatitis B - World Health Organization (WHO), נרשמה גישה בתאריך אפריל 20, 2025, <https://www.who.int/news-room/fact-sheets/detail/hepatitis-b>
119. Hepatitis B - StatPearls - NCBI Bookshelf, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ncbi.nlm.nih.gov/books/NBK555945/>
120. Nonhuman primate models of polycystic ovary syndrome | Request ..., נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/publication/235390980_Nonhuman_primate_models_of_polycystic_ovary_syndrome>
121. Hepatitis B: Practice Essentials, Background, Pathophysiology - Medscape Reference, נרשמה גישה בתאריך אפריל 20, 2025, <https://emedicine.medscape.com/article/177632-overview>
122. Hepatitis B - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Hepatitis_B>
123. The Chimpanzee Model for Hepatitis B Virus Infection - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC4448699/>
124. Animal Models Used in Hepatitis C Virus Research - PMC, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC7312079/>
125. Animal Models of Hepatitis B Virus Infection–Success, Challenges, and Future Directions, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/1999-4915/13/5/777>
126. REVIEW Hepatitis B virus infection in non-human primates - Frontiers Publishing Partnerships, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontierspartnerships.org/articles/10.4149/av_2009_02_73/pdf>
127. Hepatitis B virus infection in non-human primates | Request PDF - ResearchGate, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/publication/26302797_Hepatitis_B_virus_infection_in_non-human_primates>
128. Species Association of Hepatitis B Virus (HBV) in Non-Human Apes; Evidence for Recombination between Gorilla and Chimpanzee Variants - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC3303819/>
129. Origins and Evolution of the Primate Hepatitis B Virus - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/microbiology/articles/10.3389/fmicb.2021.653684/full>
130. Hepatitis B Virus Immunopathology, Model Systems, and Current Therapies - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2017.00436/full>
131. The Multiple Functions of B Cells in Chronic HBV Infection - PMC, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC7767983/>
132. Immunopathology of Chronic Hepatitis B Infection: Role of Innate and Adaptive Immune Response in Disease Progression - MDPI, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/1422-0067/22/11/5497>
133. Hepatitis B – Cells & Markers involved in the Immune Response | Bio-Rad, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.bio-rad-antibodies.com/hepatitis-b-immune-response.html>
134. Clinical Overview of Hepatitis C - CDC, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.cdc.gov/hepatitis-c/hcp/clinical-overview/index.html>
135. Hepatitis C Virus: Diagnosis and Treatment - American Liver Foundation, נרשמה גישה בתאריך אפריל 20, 2025, <https://liverfoundation.org/liver-diseases/viral-hepatitis/hepatitis-c/diagnosing-hepatitis-c/>
136. Hepatitis C - Symptoms and causes - Mayo Clinic, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mayoclinic.org/diseases-conditions/hepatitis-c/symptoms-causes/syc-20354278>
137. Hepatitis C - Genentech, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.gene.com/patients/disease-education/hepatitis-c-fact-sheet>
138. Hepatitis C - Complications - NHS, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.nhs.uk/conditions/hepatitis-c/complications/>
139. The Chimpanzee Model of Hepatitis C Virus Infections | Request PDF - ResearchGate, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/publication/11932608_The_Chimpanzee_Model_of_Hepatitis_C_Virus_Infections>
140. Barriers of hepatitis C virus interspecies transmission - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC3523278/>
141. Animal Models for Hepatitis C Infection Studies, נרשמה גישה בתאריך אפריל 20, 2025, <https://viralhepatitisjournal.org/articles/doi/vhd.44366>
142. Full article: Comparative Host Genomics: How has Human Evolution Affected Our Immune Defence Against Hepatitis C virus? - Taylor and Francis, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.tandfonline.com/doi/full/10.2217/fvl-2019-0017>
143. Hepatitis C virus - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Hepatitis_C_virus>
144. Hepatitis C virus production by human hepatocytes dependent on assembly and secretion of very low-density lipoproteins | PNAS, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.pnas.org/doi/10.1073/pnas.0700760104>
145. Human cell types important for Hepatitis C Virus replication in vivo and in vitro. Old assertions and current evidence - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC3142522/>
146. Impact of Hepatitis C Virus Infection of Peripheral Blood Mononuclear Cells on the Immune System - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/virology/articles/10.3389/fviro.2021.810231/full>
147. Hepatitis C Virus Infection: Host–Virus Interaction and Mechanisms of Viral Persistence, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/2073-4409/8/4/376>
148. Duchenne and Becker muscular dystrophy - Genetics - MedlinePlus, נרשמה גישה בתאריך אפריל 20, 2025, <https://medlineplus.gov/genetics/condition/duchenne-and-becker-muscular-dystrophy/>
149. Muscular Dystrophy - StatPearls - NCBI Bookshelf, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ncbi.nlm.nih.gov/books/NBK560582/>
150. The Muscular Dystrophies: From Genes to Therapies - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC4496952/>
151. Muscular Dystrophy | National Institute of Neurological Disorders and Stroke, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ninds.nih.gov/health-information/disorders/muscular-dystrophy>
152. Muscular Dystrophy: What It Is, Symptoms, Types & Treatment - Cleveland Clinic, נרשמה גישה בתאריך אפריל 20, 2025, <https://my.clevelandclinic.org/health/diseases/14128-muscular-dystrophy>
153. Duchenne muscular dystrophy - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Duchenne_muscular_dystrophy>
154. The complex landscape of DMD mutations: moving towards personalized medicine, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/genetics/articles/10.3389/fgene.2024.1360224/full>
155. Muscular dystrophy - Symptoms & causes - Mayo Clinic, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mayoclinic.org/diseases-conditions/muscular-dystrophy/symptoms-causes/syc-20375388>
156. Duchenne's muscular dystrophy: animal models used to investigate pathogenesis and develop therapeutic strategies - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC2517561/>
157. Types of Muscular Dystrophy - CDC, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.cdc.gov/muscular-dystrophy/types/index.html>
158. Duchenne Muscular Dystrophy - Symptoms, Causes, Treatment | NORD, נרשמה גישה בתאריך אפריל 20, 2025, <https://rarediseases.org/rare-diseases/duchenne-muscular-dystrophy/>
159. Duchenne Muscular Dystrophy (DMD) - Diseases, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mda.org/disease/duchenne-muscular-dystrophy>
160. Duchenne Muscular Dystrophy - CheckRare, נרשמה גישה בתאריך אפריל 20, 2025, <https://checkrare.com/duchenne-muscular-dystrophy/>
161. Facioscapulohumeral muscular dystrophy - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Facioscapulohumeral_muscular_dystrophy>
162. Muscular dystrophy - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Muscular_dystrophy>
163. Two Novel Mouse Models of Duchenne Muscular Dystrophy with Similar Dmd Exon 51 Frameshift Mutations and Varied Phenotype Severity - MDPI, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/1422-0067/26/1/158>
164. Becker muscular dystrophy severity is linked to the structure of dystrophin - Oxford Academic, נרשמה גישה בתאריך אפריל 20, 2025, <https://academic.oup.com/hmg/article/24/5/1267/792416>
165. Great ape genetic diversity catalog frames primate evolution and future conservation, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.eurekalert.org/news-releases/646769>
166. Duchenne muscular dystrophy disease severity impacts skeletal muscle progenitor cells systemic delivery - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/physiology/articles/10.3389/fphys.2023.1190524/full>
167. Understanding Asthma Phenotypes, Endotypes, and Mechanisms of Disease - PMC, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC6411459/>
168. Phenotype overlap in the natural history of asthma | European Respiratory Society, נרשמה גישה בתאריך אפריל 20, 2025, <https://publications.ersnet.org/content/errev/32/168/220201>
169. Asthma phenotypes: Definition, research, and more - Medical News Today, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.medicalnewstoday.com/articles/asthma-phenotypes>
170. Bronchial Asthma Treatments, Symptoms, Causes, and More - WebMD, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.webmd.com/asthma/bronchial-asthma>
171. The Immunology of Asthma: Asthma Phenotypes and Their Implications for Personalized Treatment - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC4977430/>
172. Asthma Phenotypes, נרשמה גישה בתאריך אפריל 20, 2025, <https://asthma.ca/get-help/understanding-asthma/asthma-phenotypes/>
173. Emerging molecular phenotypes of asthma - American Journal of Physiology, נרשמה גישה בתאריך אפריל 20, 2025, <https://journals.physiology.org/doi/10.1152/ajplung.00070.2014>
174. Asthma Phenotypes in the Era of Personalized Medicine - MDPI, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/2077-0383/12/19/6207>
175. Delineating asthma according to inflammation phenotypes with a focus on paucigranulocytic asthma | Chinese Medical Journal - MedNexus, נרשמה גישה בתאריך אפריל 20, 2025, <https://mednexus.org/doi/10.1097/CM9.0000000000002456>
176. Prostaglandin D2 and TH2 Inflammation in the Pathogenesis of Bronchial Asthma - The Korean Journal of Internal Medicine, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.kjim.org/upload/kjim-26-8.pdf>
177. Ion Channels in the Immune Response of Asthma - SCIEPublish, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.sciepublish.com/article/pii/337>
178. The Airway Epithelium—A Central Player in Asthma Pathogenesis - MDPI, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/1422-0067/21/23/8907>
179. T-helper cells and their cytokines in pathogenesis and treatment of asthma - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2023.1149203/full>
180. Asthma: The Use of Animal Models and Their Translational Utility - MDPI, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/2073-4409/12/7/1091>
181. More Than Just a Barrier: The Immune Functions of the Airway Epithelium in Asthma Pathogenesis - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2020.00761/full>
182. Nonhuman primate models of asthma - PMC, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC2212032/>
183. Asthma: a comparison of animal models using stereological methods - ERS Publications, נרשמה גישה בתאריך אפריל 20, 2025, <https://publications.ersnet.org/content/errev/15/101/122>
184. Airway generation-specific differences in the spatial distribution of immune cells and cytokines in allergen-challenged rhesus monkeys - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC3918236/>
185. The cell biology of asthma - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC4050726/>
186. Bronchial epithelium in children: a key player in asthma | European Respiratory Society, נרשמה גישה בתאריך אפריל 20, 2025, <https://publications.ersnet.org/content/errev/25/140/158>
187. Pathophysiology Of Asthma - StatPearls - NCBI Bookshelf, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ncbi.nlm.nih.gov/books/NBK551579/>
188. Cell types involved in allergic asthma and their use in in vitro models to assess respiratory sensitization - ResearchGate, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/publication/5248503_Cell_types_involved_in_allergic_asthma_and_their_use_in_in_vitro_models_to_assess_respiratory_sensitization>
189. Rapid Evolution of Primate Type 2 Immune Response Factors Linked to Asthma Susceptibility - Oxford Academic, נרשמה גישה בתאריך אפריל 20, 2025, <https://academic.oup.com/gbe/article/9/6/1757/3930139>
190. Toll-Like Receptor 7/8 Ligand, S28463, Suppresses Ascaris suum–induced Allergic Asthma in Nonhuman Primates - ATS Journals, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.atsjournals.org/doi/10.1165/rcmb.2017-0184OC>
191. Human CD8+ T Cells in Asthma: Possible Pathways and Roles for NK-Like Subtypes, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2016.00638/full>
192. Local genetic and environmental factors in asthma disease pathogenesis: chronicity and persistence mechanisms - ERS Publications, נרשמה גישה בתאריך אפריל 20, 2025, <https://publications.ersnet.org/content/erj/29/4/793>
193. Pre-eclampsia - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Pre-eclampsia>
194. Preeclampsia - StatPearls - NCBI Bookshelf, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ncbi.nlm.nih.gov/books/NBK570611/>
195. Preeclampsia: Background, Pathophysiology, Etiology - Medscape Reference, נרשמה גישה בתאריך אפריל 20, 2025, <https://emedicine.medscape.com/article/1476919-overview>
196. Primary Human Trophoblasts Mimic the Preeclampsia Phenotype after Acute Hypoxia–Reoxygenation Insult - MDPI, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/2073-4409/11/12/1898>
197. Phenotype‐Directed Management of Hypertension in Pregnancy | Journal of the American Heart Association, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ahajournals.org/doi/10.1161/JAHA.121.023694>
198. dbPEC: a comprehensive literature-based database for preeclampsia related genes and phenotypes - Oxford Academic, נרשמה גישה בתאריך אפריל 20, 2025, <https://academic.oup.com/database/article/doi/10.1093/database/baw006/2630148>
199. Polycystic Ovarian Syndrome: A Complex Disease with a Genetics ..., נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC8945152/>
200. Preeclampsia has two phenotypes which require different treatment strategies - PubMed, נרשמה גישה בתאריך אפריל 20, 2025, <https://pubmed.ncbi.nlm.nih.gov/34774281/>
201. Placental invasion, preeclampsia risk and adaptive molecular evolution at the origin of the great apes: Evidence from genome-wide analyses | Request PDF - ResearchGate, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/publication/233983771_Placental_invasion_preeclampsia_risk_and_adaptive_molecular_evolution_at_the_origin_of_the_great_apes_Evidence_from_genome-wide_analyses>
202. Placental invasion, preeclampsia risk and adaptive molecular evolution at the origin of the great apes: evidence from genome-wide analyses - PubMed, נרשמה גישה בתאריך אפריל 20, 2025, <https://pubmed.ncbi.nlm.nih.gov/23266291/>
203. An immunological insight into the origins of pre-eclampsia - Oxford Academic, נרשמה גישה בתאריך אפריל 20, 2025, <https://academic.oup.com/humupd/article/16/5/510/664236>
204. Preeclampsia: From Cellular Wellness to Inappropriate Cell Death, and the Roles of Nutrition - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/cell-and-developmental-biology/articles/10.3389/fcell.2021.726513/full>
205. Approaches to modeling placental function in preeclampsia in vitro and in vivo - PMC, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC10330201/>
206. Subtypes of Preeclampsia: Recognition and Determining Clinical Usefulness | Hypertension, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ahajournals.org/doi/10.1161/HYPERTENSIONAHA.120.14781>
207. Mechanisms of Key Innate Immune Cells in Early- and Late-Onset Preeclampsia - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2020.01864/full>
208. Pathogenesis of Preeclampsia and Therapeutic Approaches Targeting the Placenta - MDPI, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mdpi.com/2218-273X/10/6/953>
209. Animal models of preeclampsia: translational failings and why, נרשמה גישה בתאריך אפריל 20, 2025, <https://journals.physiology.org/doi/full/10.1152/ajpregu.00355.2017>
210. The role of immune cells and mediators in preeclampsia - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC10038936/>
211. The Biology of Preeclampsia - PMC, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC4313558/>
212. Placental Growth Factor Reduces Blood Pressure in a Uteroplacental Ischemia Model of Preeclampsia in Nonhuman Primates - PubMed, נרשמה גישה בתאריך אפריל 20, 2025, <https://pubmed.ncbi.nlm.nih.gov/27091894/>
213. Placental Growth Factor Reduces Blood Pressure in a Uteroplacental Ischemia Model of Preeclampsia in Nonhuman Primates | Hypertension - American Heart Association Journals, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ahajournals.org/doi/10.1161/hypertensionaha.116.07286>
214. Types of Bipolar Disorder and Their Distinctive Features - WebMD, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.webmd.com/bipolar-disorder/mental-health-bipolar-disorder>
215. Bipolar disorder - Symptoms and causes - Mayo Clinic, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mayoclinic.org/diseases-conditions/bipolar-disorder/symptoms-causes/syc-20355955>
216. Bipolar disorder: Symptoms, causes, types, and treatment - Medical News Today, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.medicalnewstoday.com/articles/37010>
217. Using Chronobiological Phenotypes to Address Heterogeneity in Bipolar Disorder, נרשמה גישה בתאריך אפריל 20, 2025, <https://karger.com/mnp/article/5/Suppl.%201/72/202280/Using-Chronobiological-Phenotypes-to-Address>
218. Bipolar disorder - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Bipolar_disorder>
219. Abnormal features of human self-domestication in bipolar disorder - bioRxiv, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.biorxiv.org/content/10.1101/2020.04.28.065581v1.full-text>
220. The common bipolar phenotype in young people - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC4715012/>
221. The “Hard” and “Soft” Phenotypic Boundaries of Bipolar Disorder - Psychiatric Times, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.psychiatrictimes.com/view/hard-and-soft-phenotypic-boundaries-bipolar-disorder>
222. Study Lays Groundwork for Potential Bipolar Disorder Therapies, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.scripps.edu/newsandviews/e_20160314/davis.html>
223. Study lays groundwork for potential bipolar disorder therapies - ScienceDaily, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.sciencedaily.com/releases/2016/03/160309125732.htm>
224. Evolutionary origin of bipolar disorder-revised: EOBD-R - "the hypothesis is extended to suggest Neandertal as the ancestral source for bipolar vulnerability genes (susceptibility alleles)." - Reddit, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.reddit.com/r/Anthropology/comments/m5n2r/evolutionary_origin_of_bipolar_disorderrevised/>
225. Chapter 9 - How Different Are Humans and “Great Apes”? A Matrix of Comparative Anthropogeny, נרשמה גישה בתאריך אפריל 20, 2025, <https://cmm.ucsd.edu/research/labs/varki/_files/publications/b178.pdf>
226. Do primates, or any other animal suffer from the more advanced mental illnesses, Such as schizophrenia and Bi-polar disorder? - Reddit, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.reddit.com/r/askscience/comments/3hl2t2/do_primates_or_any_other_animal_suffer_from_the/>
227. Genetic links with bipolar disorder identified | UCL News, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ucl.ac.uk/news/2025/jan/genetic-links-bipolar-disorder-identified>
228. Largest Genetic Study of Bipolar Disorder Identifies 298 Regions of the Genome That Increase Risk for the Condition | Mount Sinai - New York, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mountsinai.org/about/newsroom/2025/largest-genetic-study-of-bipolar-disorder-identifies-298-regions-of-the-genome-that-increase-risk-for-the-condition>
229. Biology of bipolar disorder - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Biology_of_bipolar_disorder>
230. Effects of bipolar disorder on the brain - Medical News Today, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.medicalnewstoday.com/articles/bipolar-disorder-and-the-brain>
231. Cell-type-specific genes associated with cortical structural abnormalities in pediatric bipolar disorder - Oxford Academic, נרשמה גישה בתאריך אפריל 20, 2025, <https://academic.oup.com/psyrad/article/2/2/56/6712342>
232. New Insights into the Biological Basis of Bipolar Disorder - AZoLifeSciences, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.azolifesciences.com/news/20250124/New-Insights-into-the-Biological-Basis-of-Bipolar-Disorder.aspx>
233. Functional neuroanatomy of bipolar disorder: structure, function, and connectivity in an amygdala–anterior paralimbic neural system - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC3880745/>
234. Cell types underlying schizophrenia identified - University College London, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.ucl.ac.uk/news/2018/may/cell-types-underlying-schizophrenia-identified>
235. Genetic identification of brain cell types underlying schizophrenia - PubMed, נרשמה גישה בתאריך אפריל 20, 2025, <https://pubmed.ncbi.nlm.nih.gov/29785013/>
236. Genetic Insights of Schizophrenia via Single Cell RNA-Sequencing Analyses, נרשמה גישה בתאריך אפריל 20, 2025, <https://academic.oup.com/schizophreniabulletin/article/49/4/914/7048714>
237. Von Economo neurons: A Review of the Anatomy and Functions - Austin Publishing Group, נרשמה גישה בתאריך אפריל 20, 2025, <https://austinpublishinggroup.com/anatomy/fulltext/Anatomy-v1-id1026.php>
238. A neuronal morphologic type unique to humans and great apes - ResearchGate, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/publication/235612186_A_neuronal_morphologic_type_unique_to_humans_and_great_apes>
239. Von Economo Neuron Involvement in Social Cognitive and Emotional Impairments in Neuropsychiatric Disorders | The Journal of Neuropsychiatry and Clinical Neurosciences - Psychiatry Online, נרשמה גישה בתאריך אפריל 20, 2025, <https://psychiatryonline.org/doi/full/10.1176/appi.neuropsych.20220136>
240. New neuron type discovered only in primate brains - Broad Institute, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.broadinstitute.org/news/new-neuron-type-discovered-only-primate-brains>
241. New neuron type discovered only in primate brains - MIT McGovern Institute, נרשמה גישה בתאריך אפריל 20, 2025, <https://mcgovern.mit.edu/2020/09/30/new-neuron-type-discovered-only-in-primate-brains/>
242. Endophenotypes in Schizophrenia: Digging Deeper to Identify Genetic Mechanisms, נרשמה גישה בתאריך אפריל 20, 2025, <https://jpbs.hapres.com/htmls/JPBS_1009_Detail.html>
243. Schizophrenia - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Schizophrenia>
244. Physiological Indicators of the Schizophrenia Phenotype - ACNP, נרשמה גישה בתאריך אפריל 20, 2025, <https://acnp.org/g4/GN401000113/CH111.html>
245. Defining the schizophrenia phenotype - PubMed, נרשמה גישה בתאריך אפריל 20, 2025, <https://pubmed.ncbi.nlm.nih.gov/11122987/>
246. Biological phenotypes and genetic research on schizophrenia - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC1489838/>
247. Genetic Epidemiology of Schizophrenia: Phenotypes, Risk Factors, and Reproductive Behavior | American Journal of Psychiatry, נרשמה גישה בתאריך אפריל 20, 2025, <https://psychiatryonline.org/doi/10.1176/appi.ajp.160.3.425>
248. Expression of Behavioral Phenotypes in Genetic and Environmental Mouse Models of Schizophrenia - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/behavioral-neuroscience/articles/10.3389/fnbeh.2020.00029/full>
249. Genetics of Schizophrenia: Overview of Methods, Findings and Limitations - Frontiers, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.frontiersin.org/journals/human-neuroscience/articles/10.3389/fnhum.2017.00322/full>
250. Deconstructing Schizophrenia: An Overview of the Use of Endophenotypes in Order to Understand a Complex Disorder - Oxford Academic, נרשמה גישה בתאריך אפריל 20, 2025, <https://academic.oup.com/schizophreniabulletin/article/33/1/21/1926582>
251. Full article: Intermediate phenotypes in schizophrenia: a selective review, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.tandfonline.com/doi/full/10.31887/DCNS.2005.7.2/gpreston>
252. Animal model of schizophrenia - Wikipedia, נרשמה גישה בתאריך אפריל 20, 2025, <https://en.wikipedia.org/wiki/Animal_model_of_schizophrenia>
253. Schizophrenia—an evolutionary enigma? - USD Biology, נרשמה גישה בתאריך אפריל 20, 2025, <http://usdbiology.com/cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Schizophrenia/Brune04.pdf>
254. How and Why Genetic Linkage Has Not Solved the Problem of Psychosis: Review and Hypothesis | American Journal of Psychiatry, נרשמה גישה בתאריך אפריל 20, 2025, <https://psychiatryonline.org/doi/full/10.1176/ajp.2007.164.1.13>
255. Analysis of differentially methylated regions in great apes and extinct hominids provides support for the evolutionary hypothesis of schizophrenia - PubMed, נרשמה גישה בתאריך אפריל 20, 2025, <https://pubmed.ncbi.nlm.nih.gov/30545758/>
256. Schizophrenia and human self-domestication: a linguistic approach - bioRxiv, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.biorxiv.org/content/biorxiv/early/2016/08/31/072751.full.pdf>
257. Analysis of differentially methylated regions in great apes and extinct hominids provides support for the evolutionary hypothesis of schizophrenia - ResearchGate, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.researchgate.net/publication/329560170_Analysis_of_differentially_methylated_regions_in_great_apes_and_extinct_hominids_provides_support_for_the_evolutionary_hypothesis_of_schizophrenia>
258. Adaptive evolution of genes underlying schizophrenia | Proceedings of the Royal Society B, נרשמה גישה בתאריך אפריל 20, 2025, <https://royalsocietypublishing.org/doi/10.1098/rspb.2007.0876>
259. Adaptive evolution of genes underlying schizophrenia | Proceedings of the Royal Society B: Biological Sciences - Journals, נרשמה גישה בתאריך אפריל 20, 2025, <https://royalsocietypublishing.org/doi/abs/10.1098/rspb.2007.0876>
260. On schizophrenia as a "disease of humanity" - PubMed, נרשמה גישה בתאריך אפריל 20, 2025, <https://pubmed.ncbi.nlm.nih.gov/23182440/>
261. Genetic identification of brain cell types underlying schizophrenia - PMC - PubMed Central, נרשמה גישה בתאריך אפריל 20, 2025, <https://pmc.ncbi.nlm.nih.gov/articles/PMC6477180/>
262. Mapping the Cellular Etiology of Schizophrenia and Diverse Brain Phenotypes - medRxiv, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.medrxiv.org/content/10.1101/2024.10.21.24315695v1.full-text>
263. Brain-cell 'periodic table' for psychiatric disorders reveals new schizophrenia clues, נרשמה גישה בתאריך אפריל 20, 2025, <https://med.stanford.edu/news/all-news/2025/01/brain-cell--periodic-table--for-psychiatric-disorders-reveals-ne>
264. Schizophrenia: Which cell types are involved? - Medical News Today, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.medicalnewstoday.com/articles/321918>
265. Scientists identify brain cell types underlying schizophrenia | Newsroom - UNC Health, נרשמה גישה בתאריך אפריל 20, 2025, <https://news.unchealthcare.org/2018/05/scientists-identify-brain-cell-types-underlying-schizophrenia/>
266. Study Uncovers Cell Type-Specific Genetic Insights Underlying Schizophrenia, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.mcleanhospital.org/news/study-uncovers-cell-type-specific-genetic-insights-underlying-schizophrenia>
267. Study uncovers cell type-specific genetic insights underlying schizophrenia - EurekAlert!, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.eurekalert.org/news-releases/1045341>
268. An evolutionary theory of schizophrenia: Cortical connectivity, metarepresentation, and the social brain - CiteSeerX, נרשמה גישה בתאריך אפריל 20, 2025, <https://citeseerx.ist.psu.edu/document?repid=rep1&type=pdf&doi=ca70d58e6e310f77f410437aeae88d451bffce48>
269. Accelerated evolution of oligodendrocytes in the human brain - PNAS, נרשמה גישה בתאריך אפריל 20, 2025, <https://www.pnas.org/doi/10.1073/pnas.1907982116>