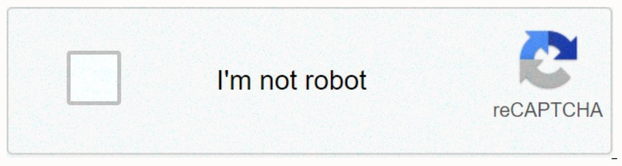

Continue

Types of inflammatory mediators

Inflammation of cardinal inflammatory symptoms include pain, heat, redness, swelling, and loss of function. Some of these indicators are seen here due to an allergic reaction. Immunology Rheumatology Sympathy Warm Pain Swelling PainCompliclicationsasthma pneumonia Autoimmune DiseasesDurationacute Few days chronic up to many months, Or for years because the bacterial virus has physical effects caused by the activation of inflammation of the immune system (from Latin: inflammatio) is part of the complex biological response of body tissues to harmful stimuli, such as pathogens, damaged cells, or stimulators, and a protective response includes immune cells, blood vessels. , and molecular mediators. The function of inflammation is to eliminate the primary cause of cell damage, turn on the necretic cells and damaged tissues from the main insult and inflammatory process, and begin tissue repair. The five classic signs of inflammation are heat, pain, redness, swelling, and loss of function (Latin calories, delore, bre, tumor, and laza phantom). [1] Inflammation is a general response, and therefore it is regarded as a dedication immune mechanism, as compared to adaptive immunity, which is specific to any pathogen. [2] Too little inflammation can lead to the destruction of progressive tissue by harmful stimulants (e.g. bacteria) and endanger the survival of the organism. In contrast, chronic inflammation is associated with various diseases such as hay fever, periodontal disease, atherosclerosis, and osteoarthritis. Inflammation can be classified as acute or chronic. Acute inflammation is the body's primary response to harmful stimuli and is achieved by increasing the movement of plasma and leukocytes (especially granulocytes) from the blood to damaged tissues. A series of biochemical events release and mature inflammatory responses, involving the local vascular system, immune system, and various cells inside damaged tissue. Prolonged inflammation, known as chronic inflammation, leads to a progressive change in the type of cells present at the site of inflammation such as mononu nucleus cells, and is determined by simultaneous degradation and tissue improvement from the inflammatory process. Inflammation is not synonymous with infection. The infection describes the interaction between microbial invasion action and the body's inflammatory response response — two components are considered together when discussing infections, and the word used means an invasive microbial cause for the observed inflammatory reaction. Inflammation, on the other hand, merely describes the body's immunovascular response, whatever the cause may be. But because every once in a while the two are in solidarity, words that end in suffixes - it is (which refers to inflammation) are sometimes informally described as a reference to infection. For example, the word uretretrite strictly means only uretral inflammation, but clinical health care providers typically discuss overtreat as an everthral infection because uretral microbial invasion is the most common cause of uretrite. It is useful for detecting inflammation and infection because there are typical situations in pathology and medical diagnosis where inflammation is not driven by microbial invasion - for example, atherosclerosis, trauma, ischemia, and self-help diseases including type III hypersensitivity. The causes of this section require additional citations to be verified. Please help improve this article by adding citations to reliable sources. Unse sourced materials may be challenged and removed. (December 2015) (Learn how and when to remove this mold message) Physical: Burns[3] Physical damage frostbite, blunt or penetrating[4] external bodies, including splant, soil and trauma debris[3] Biological radiation ionization: infection by pathogens[3] Immune reactions caused by excessive stress Chemical sensitivity:[3] Chemical stimulators of psychological alcohol toxins: excitement[5] See types: list of types of inflammation by the location of the appendicitis B.B.C. Colitis Cystitis Epididymitis 5Encephalitis Gingivitis Meningivitis Myelitis Nephritis Neuritis Pancreatitis Periodontitis Phlebngitis Phlebitis Prostatitis RSD/CRPS Rhinitis Sinus tenditis Tendonitis Tonsonitis Urethritis Vasculitis Vaginitis Comparison between acute and chronic inflammation: Acute Chronic Causative Agent Pathogens Damaged tissues have persistent acute inflammation due to non-biodegradable pathogens, viral infection, persistent external body, or immune reactions of major cells involving neutrophils (primarily), basophils (inflammatory responses), and echosinophiles (responses to helminth worms) And parasites), mononu core cells (monocytes, macrophages) mononumeric cells (monocytes, macrophages, lymphocytes, plasma cells), fibroasts are the primary mediators of vasoactive amines, E. IFN-γ and other cytokines, growth factors, reactive oxygen species, hydrolytic enzymes begin immediate delay periods of several days to many months, or outcomes resolution, abscess formation, chronic inflammation tissue destruction, fibrosis, cardinal necrosis signs the classic signs and symptoms of acute inflammation: English Latin Redness Rubor Swelling Tumor Heat Calor Pain Dolor Loss of function Functio laesa All the above signs may be observed in specific instances, But no single sign must, as a matter of course, be present. [6] These are the main, or cardinal signs of inflammation. [6] Functio laesa is an old imagination, because it is not unique to inflammation and is a feature of many states of the disease. [7] Infected swollen toenails indicate characteristic redness and swelling associated with acute inflammation of acute inflammation is a short-term trend, Appear within minutes or hours and start stopping after removing the damage stimulus. [8] This includes a coordinated and systemic mobilization response locally from various immune, endocrine and neurological mediators of acute inflammation. It is activated in a normal healthy response, cleanses the pathogen and begins a repair process and then stops. [9] It is marked by five cardinal symptoms:[10] An acronym that may be used to remember key symptoms is PRISH, for pain, redness, inactivity (loss of function), swelling and heat. Traditional names for signs of inflammation come from Latin: Delore (pain) calories (heat) berry (redness) tumor (swelling) Functio laesa (loss of function)[11] the first four (classic signs) were described by Celsus (circa 30 BC-38 AD), while performance loss was probably later added by Galen. However, the addition of this fifth mark is also attributed to Thomas Sydenham and Wirshu. [8] Redness and heat caused by increased blood flow at the body's core temperature are inflamed into place; Swelling is caused by fluid accumulation; Pain is caused by the release of chemicals such as bradykinin and hesamine, which stimulates the nerve endings. Loss of function has several causes. [10] Acute lung inflammation (usually caused by chest-to-chest response) does not cause pain unless inflammation includes a ahi-ahi pleural that has a sore nerve ending. [10] Acute inflammation micrograph process showing granulation tissue. Spotting H&E. The process of acute inflammation by resident immune cells already involved in tissues already exists, mainly resident macrophages, dendrite cells, tissueocytes, coupler cells and mast cells started. These cells have surface receptors known as pattern recognition receptors (PRRs) that detect two subclasses of molecules: pathogenic molecular patterns (PAMPs) and damage-related molecular patterns (DAMPs). PAMPs are compounds that are associated with various pathogens, but can be detected from host molecules. DAMPs are compounds that are associated with host-related damage and cell damage. At the beginning of an infection, burn, or other damage, these cells undergo activation (one of the PRRs detects a PAMP or DAMP) and the inflammatory mediators responsible for clinical symptoms release inflammation. Vasodilation and increased blood flow resulting from it cause redness (roabre) and increased heat (calories). Increasing the ability to transmit blood vessels leads to the withdrawal (leakage) of plasma and liquid proteins into tissue (edema), which itself shows as swelling (tumor). Some freed mediators such as bradykinin increase sensitivity to pain (Hyperalgesia, Delore). Mediator molecules also change blood vessels to allow the migration of leukocytes, mainly neutrophils and Outside the blood vessels (extravagance) to the tissue. Neutrophils migrate along a chemothetic gradient created by local cells to damage the site. [8] Loss of function (functio laesa) is probably the result of a neural reflex in response to pain. In addition to cell-derived mediators, several cellular biochemical cascading systems composed of pre-formed plasma proteins act in parallel with the initiation and propagation of inflammatory responses. Among them are the supplement system activated by bacteria and coagulation systems and fibrinolysis activated by necrosis, as a burn or impact sample. [8] Acute inflammation may be considered as the first line of defense against injury. The acute inflammatory response requires constant stimulation to be stable. Inflammatory mediators are short-lived and quickly degrade in tissue. Therefore, acute inflammation begins to stop after removing the stimulant. [8] Vasodilation component vessels and increase tram capability are defined as acute inflammation of the immunovascular response to inflammatory stimulants. This means that acute inflammation can be widely divided into a vascular phase that occurs first, followed by a cellular phase involving immune cells (specifically myeloid granulocytes in an acute environment). The vascular component of acute inflammation includes the movement of plasma fluid, containing important proteins such as fibrin and immunoglobulins (antibodies), to inflamed tissue. After contacting PAMPs, tissue macrophages and mastocytes release vasoactive amines such as hesamine and serotonin, as well as icocanoids such as prostaglandin E2 and leukothrin B4, to regenelis local vasculators. Macrophages and andothelial cells release nitric oxide. These mediators vasodilate vessels and destabilize blood vessels, leading to a net distribution of blood plasma from the vessel into the tissue space. Increased fluid collection into the tissue causes it to swell (edema). This removed tissue fluid contains various antimicrobial mediators from plasma such as supplements, lysozyms, antibodies that can immediately counter-damage to microbes, and opsonise germs in preparation for the cellular phase. If the inflammatory stimulant is a grating wound, ostracized platelets, coagulants, plasmines and quinines can clot the wounded area and provide hemususus in the first sample. This flocculation mediator also provides a structural stagering framework on the site of inflammatory tissue in the form of fibrin nets - as construction scaffolds at a construction site - in order to help phagocytic residues and wound healing later. Some tissue fluid exuded is also funneled by lymphatic into the regional lymph nodes, a kindle of bacteria along to begin the detection and attack phase of the adaptive immune system. Inflammation is acute With marked vasodilation changes, including vasodilation, increased transmission capability and increased blood flow, which is induced by the actions of various inflammatory mediators. Vasodilation first occurs on the arterial surface, progressing up to the piping level and bringing a pure increase in the amount of blood available, which causes redness and heat of inflammation. Increasing the teramad capability of the veins leads to the movement of plasma into the tissues, with the stazies caused by an increase in the concentration of cells inside the blood - a condition marked by enlarged vessels packed with cells. Stasis allows leukocytes to marginalize along the endothelium, a vital process for absorbing them into tissues. Natural flowing blood prevents this, as the force of the screzes along the margins of the veins moves the cells inside the blood into the middle of the vessel. When activated, plasma cascading systems create a cascade of chemical reactions that promote opsonization, chemotaxis, and agglutination, and produce MAC. The quinine system produces proteins capable of maintaining vasodilation and other physical inflammatory effects. Coagulation system or clotting cascade that form a protective protein mesh on damage sites. The fibrinellis system, which acts in opposition to the coagulation system, is used to counter-clotting the balance and produce several other inflammatory mediators. Plasma-derived mediators * The name of the non-comprehensive list produced by the Bradykinin Quinine System Description is a vasoactive protein capable of inducing vasodilation, increasing vasodilation capability, causing smooth muscle contraction, and pain induction. C3 complements the Cleaves system for the production of C3a and C3b. C3a stimulates the release of tissue by mast cells, thereby producing vasodilation. C3b is able to bind to bacterial cell walls and act as an upsonin that marks the attacker as a target for phagocytos. The C5a supplement system stimulates the release of hesamine by mast cells, resulting in vasodilation. It is also able to act as a strychant chemistry to guide cells to the site of inflammation through chemistry. The twelfth factor (hodgeman factor) of the liver is an inactive protein A that circulates until it is activated by collagen, platelets, or basement membranes exposed through a general change. When activated, in turn it is able to activate three plasma systems involved in inflammation: the quinine system, the fibrinolysis system, and the coagulation system. Membrane Attack Complex Supplement System is a collection of supplement proteins C5b, C6, C7, C8, and multiple C9 units. The composition and activation of this range of complementary proteins constitutes a membrane attack complex that is able to enter the bacterial cell walls and causes cell lysis with the next bacterial death. Plasmin fibrinols system capable of breaking fibrin clots, cleave C3 protein supplements, and activating XII. Thrombin Coagulation system Cleaves the soluble plasma protein fibrinogen to produce insoluble fibrin, which aggregates to form a blood clot. Thrombin can also bind to cells via PAR1 receptor to initiate several other inflammatory responses, such as the production of shimikins and nitric oxide. The cellular component of the cellular component contains leukocytes, which normally live in the blood and must move to inflamed tissue through extravagance to help with inflammation. Some act as phagocytes, bacteria, viruses, and cellular debris. Others release enzymatic granules that harm pathogenic invaders. Leukocytes also release inflammatory mediators that develop and maintain an inflammatory response. In general, acute inflammation is mediated by granulocytes, while chronic inflammation is mediated by mononu core cells such as monocytes and lymphocytes. Leukocyte waste neutrophils migrate from blood vessels to infected tissue through chemistry, where they remove pathogens through phagocytosis, and inflammation is a process by which the cells and white blood materials of the body that produce protect us from infection with foreign organisms such as bacteria and viruses. White blood cells are an unsymable immune response, meaning they attack any external body. However, in some diseases, such as osteoarthritis, the immune system's immune system causes an inflammatory response when there are no foreign invaders to fight it. In these diseases, called autoimmune diseases, the normally protective immune system causes damage to their tissues. The body responds as if natural tissues are contaminated or somehow abnormal. Main article: Different leukocytes, especially neutrophils, are critically active in the onset and maintenance of inflammation. These cells should be able to move from their usual place in the blood to the site of damage, so there are mechanisms for absorbing and guiding leukocytes to the right place. The process of moving leukocytes from blood to tissues through blood vessels is known as extravagance, and can be widely divided into a number of stages: leukocyte marginalization and endothelial glue: white blood cells within the veins, which are generally centrally located, move toward the walls of the veins. [15] Macrophages active in tissue-free cytokines such as IL-1 and TNFα, which in turn lead to the production of shimikins that bind to proteoglycans that form the gradient in inflamed tissue and along the endothelial wall. Inflammatory cytokines induce immediate expression of P-selectin on the surfaces of endothelial cells, and weak P-selectin binds to carbohydrate symunds on leukocyte levels, causing them to roll along the endothelial surface by making and breaking links. Cytokines Of the damaged cells inducing the expression of E-selectin on the andothelial cells, which function similar to P-selectin. Cytokines also induce expression of integrins such as ICAM-1 and VCAM-1 on endothelial cells, which lower the adhesive mediator and slower leukocytes. These weakly restricted leukocytes are free to be separated if not activated by shimikins produced in damaged tissue after signal transmission via the corresponding G receptors of a paired protein that activates integrin on the leukocyte surface for company glue. Such activation increases the afenthus of bound integrin receptors for ICAM-1 and VCAM-1 on the surface of endothelial cells and binds leukocytes to firm endothelium. Migration across the endothelium, known as migration, through the dipadesis process: The ectokine gradients stimulate the sticking leukocytes to move between the cells within the adjacent helial. The andothelial cells retreat, and leukocytes pass through the basement membrane to the surrounding tissue using adhesive molecules such as ICAM-1. [15] The movement of leukocytes inside the tissue through chemotaxis: Leukocytes that reach the tissue interstiteium are connected to extracellular matrix proteins through expressed integrins and CD44 to prevent them from leaving the site. A variety of molecules behave as chamvatractantes, for example C3a or C5, causing leukocytes to move along a chemiotactic gradient toward the source of inflammation. Phagocytos Original article: Phagocyte-powered ferratophils come into contact with microbes in inflamed tissue in cell phase. Phagocytes express cellular endocyte pattern detection receptors (PRRs) that are closely related to molecular patterns associated with non-specific microbes (PAMPs). Most PAMPs that bind to endocytic PRRs and initiate phagocytosis are cell wall components, including complex carbohydrates such as mannans and β-glucans, lipopolysaccharides (LPS), peptidoglicans, and surface proteins. Endocyte PRRs on phagocytes reflect these molecular patterns, with Lectin-type C receptors binding to mannans and β glucans, and veneer receptors binding to LPS. After binding the endocytic PRR, the rearrangation of actin-myocin citoscle in the vicinity of plasma membrane occurs in a way that the plasma membrane containing the PRR-PAMP complex campbell, and the microbes are endocytosis. Phosphatidylositol and Vps34-Vps15-Beclin1 signaling pathways have been implicated into endocytosis phagosome traffic to intracellular lysosomes, where fusion of fagosome and lysosome produces fagolysum. Reactive oxygen species, superoxides and hypochlorite bleach inside the fagolystomas then kill microbes inside phagocytes. Phagocytic effectiveness can be enhanced by opsonization. Plasma is a C3b supplement and antibody that binds out to inflamed tissue during the vascular phase and coats the microbial-derived antigens. As As endocyte PRRs, phagocytes also express The Receptors of Upsonian Receptors Fc and Supplemental Receptor 1 (CR1), which bind to antibodies and C3b, respectively. Joint stimulation of endosytic PRR and upsonine receptor increases the effectiveness of the phagocytic process and increases the removal of infectious agent lysosomeal. Cell-derived mediators * Non-comprehensive list name source type describes Lysosome granules granules of the granulocyte enzyme these cells contain a large variety of enzymes that perform a number of functions. Granules can be classified as specific or azophilic depending on the contents, and are able to break down a number of substances, some of which may be plasma-derived proteins that allow these enzymes to act as an inflammatory mediator. Monoamine yogurt cells and basophils stored in pre-formed granules are released in response to a number of stimuli. Causes arterial dice, increased venous terminality, and a wide range of organ-specific effects. IFN-γ Cytokine T-cells, NK cells Antiviral, immunoregulatory, and anti-tumour properties. This interferon was originally called the macrophage activator, and is particularly important in maintaining chronic inflammation. IL-8 Chemokine primarily activates macrophages and neutrophil chemoattraction, with poor effect on monocytes and exinophiles. Leukotriene B4 Eicosanoid Leukocytes, cancer cells are able to mediate leukocyte glue and activation, allowing them to bind to endothelium and migrate throughout it. In neutrophils, it is also a potent chemwatrakant, capable of inducing the formation of reactive oxygen species and the release of lesosome enzymes by these cells. LTC4, LTD4 Eicosanoid eosinophils, Mast cells, Macrophages These three cysteines contain leukothins of the lung airways, increase the capability of the micro vascular tramway, stimulate mucosal secretion, and promote eosinophil-based inflammation in the lungs, skin, nose, eyes, and other tissues. 5-oxo-eicosatetraenoic acid Eicosanoid leukocytes, strong stimulant cancer cells from neutrophil chemotaxis, release lesosome enzymes, and react the formation of oxygen species; monocyte chemotaxis; And even more powerful is eosinophil chemotaxis, releasing the lesosome enzyme, and forming reactive oxygen species. 5-HETE Eicosanoid Leukocytes is a metabolic probator to 5-Oxo-eicosatetraenoic acid, it is a less potent stimulant of neutrophil chemistry, releases lesosome enzymes, and reacts to the formation of oxygen species; monocyte chemotaxis; and cymotaxie eosinophils, release of lezosome enzymes, and the formation of reactive oxygen species. Prostaglandins Eicosanoid Mast cells A group of lipids that can cause vasodilation, fever, and pain. Nitric oxide soluble gas macrophages, endothelial cells, some strong vasodilator neurons, smooth muscle relaxation, reduce platelet aggregation, assist in leukocyte absorption, direct Activity at high concentrations. TNF-α and IL-1 cytokines primarily affect macrophages both a wide range of cells to induce many of the same inflammatory reactions: fever, cytokines production, endothelial gene regulation, chemotaxis, leukocyte adherence, fibroblast activation. Responsible for the systemic effects of inflammation, such as loss of appetite and increased heart rate. TNF-α prevents osteoblasting. The trumpetase enzymes of yogurt cells this serine protease is believed to be stored exclusively in mast cells and secreted, along with hesamine, during the activation of the mast cell. [16] [17] [18] Morphological patterns of certain patterns of acute and chronic inflammation are seen in certain conditions that arise in the body, such as when inflammation occurs on an epithelial surface, or pyogenic bacteria are involved. Granulomatosis inflammation: Marked by the formation of granulomas, they result in a limited but varied number of diseases, which include among others tuberculosis, leprosy, sarcoidosis, and syphilis. Fibrin inflammation: Inflamation as a result of a large increase in vascular teramity allows fibrin to pass through blood vessels. If there is a suitable procoagulative stimulant, such as cancer cells, an exudate fibrinus precipitates. This is commonly seen in the cerus cavities, where converting fibrin exudate into a wound can occur between the cerus membranes and limit their function. The deposit sometimes constitutes a pseudo-tailed sheet. During intestinal inflammation (pseudodombranous colitis), membrane-like tubes can form. Purulent inflammation: Inflammation is the result of a large amount of pus, which is composed of neutrophils, dead cells, and fluid. Infection by pyogenic bacteria such as Staphylococcus is a feature of this type of inflammation. Large, locally enclosed collections of pus enclosed by surrounding tissues are called abscesses. Cerus inflammation: Marked by abundant effusion of non-vascular cerus fluid, commonly produced by cerus membrane mesosis cells, but may be derived from blood plasma. Skin blisters are typical of this pattern of inflammation. Ulcerative inflammation: Inflammation occurring near an epithelium can lead to necotic tissue loss from the surface, exposing the lower layers. The next excavation in the epithelium is known as scarring. Inflammatory asthma disorders are a mediated inflammatory disorder. On the right is an inflamed airway due to asthma. Colitis (colon inflammation) caused by Crohn's disease. Inflammatory abnormalities are a large group of disorders that affect a wide variety of human diseases. The immune system is often involved with inflammatory disorders, shown in both allergic reactions and some myopathy, with many immune system disorders resulting in abnormal inflammation. Non-safety The origins of inflammatory processes include cancer, atherosclerosis, and ischemic heart disease. [8] Examples of inflammatory-related disorders include: vulgar acne asthma autoimmune diseases inflammatory disease celiac disease chronic prostatitis phyroticitis family Mediterranean fever glomerolonphoreitis hydrdnitis hypersensitivity inflammatory bowel disease Interstitial Cystitis Lichen Planus Mast Cell Activation Syndrome Mastocytosis Otitis Pelvis Inflammatory Disease Pneumonia Reperfusion Damage Rheumatic Fever Rheumatoid Arthritis Rhinitis Sarcoidosis Transplant Rejection Vasculitis Atherosclerosis Main Article : Atherosclerosis atherosclerosis, previously considered a blood fat storage disease, actually involves an on-site inflammatory response. Recent advances in basic sciences have played an essential role for inflammation in mediating all stages of the disease from the onset through progression and ultimately the thrombotic complications of atherosclerosis. These new findings provide important links between risk factors and atherogenes mechanisms. Clinical studies have shown that this emerging biology of inflammation in atherosclerosis applies directly to human patients. Climbing in markers of inflammation predicts the outcomes of patients with acute coronary syndrome independently of myocardial injury. In addition, low-grade chronic inflammation, as shown by inflammatory marker C reactive protein levels, prospectively defines the risk of atherosclerotic complications, thereby adding to prognostic information provided by traditional risk factors. Certain treatments that reduce the risk of coronary artery disease also limit inflammation. In the case of fat loss with statins, this anti-inflammatory effect does not appear to be associated with a decrease in low-density lipoprotein levels. These new insights into inflammation in atherosclerosis not only enhance our understanding of the disease but also practical clinical applications in the ordering of risk and targeting treatment for this pest of growing importance around the world. [19] Allergic reaction allergies, formally known as type 1 hypersensitivity, are thereby inappropriate immune responses causing inflammation, vasodilation, and nerve stimulation. A common example is hay fever, caused by an overly sensitive response by cell masts to allergens. Pre-allergic mast cells respond with degradation and release vasoactive chemicals such as hesamine. These chemicals release an over-the-limit inflammatory response marked by diffusion of blood vessels, produce pro-inflammatory molecules, release cytokines, and employ leukocytes. [8] Severe inflammatory responses may mature to a systemic response known as anaphylaxis. Inflammatory myopathies caused by immune system are inappropriate Muscle components, leading to signs of muscle inflammation. They may occur in conjunction with other immune disorders, such as systemic sclerosis, and include dermatomyositis, polymyosite, and body myositis inclusion. [8] Leukocyte defects due to the central role of leukocytes in the development and dissemination of inflammation, defects in leukocyte function often lead to reduced capacity for inflammatory defenses with subsequent vulnerability to infection. [8] Dysfunctional leukocytes may not be able to properly bind to blood vessels due to mutations in surface receptors, digestion of bacteria (Chédiak-Higashi syndrome), or the production of syd microbes (chronic granulomatous disease). In addition, diseases affecting bone marrow may lead to abnormal or quantitative leukocytes. Specific pharmacological drugs or exogenous chemical compounds are known to affect inflammation. Vitamin A deficiency increases inflammatory responses[20] and anti-inflammatory drugs specifically work by inhibiting enzymes that produce inflammatory eiclanoids. Some illicit drugs such as cocaine and ecstasy may exert some detrimental effects by activating transcription agents that are intimately involved with inflammation (such as NF-ʎB). [21] [22] Cancer inflammation regulates microenvironment around tumors, helping to replicate, survive and migrate. [23] Cancer cells use their selector, shimikins and receptors to invade, migrate and metastasis. [24] On the other hand, many immune system cells contribute to cancer's immunology, suppressing cancer. [25] The molecular intersection between receptors of steroid hormones, which have important effects on cellular development, and transcription factors that play key roles in inflammation, such as NF-ʎB, may mediate some of the most critical effects of inflammatory stimuli on cancer cells. [26] This capacity of mediator inflammation to influence the effects of steroid hormones on cells, is highly likely to affect carcinogenesis on the one hand; On the other hand, due to the modular nature of many steroid hormone receptors, this interaction may offer ways to interfere with cancer progression, by targeting a specific protein amplitude in a specific cell type. Such an approach may limit side effects that are out of touch with a favorite tumor, and may help maintain vital hemostatic functions and evolutionary processes in the organism. According to a 2009 study, recent data suggests that cancer-related inflammation (CRI) may lead to a accumulation of random genetic changes in cancer cells. [27] In 1863, Rudolph Wirshu hypothesly hypotheslyed that cancer originated in places of chronic inflammation. [28] [29] Currently, chronic inflammation is estimated to contribute to about 15% to 25% of human cancers. [29] Mediators and DNA Damage in Inflammatory Mediator Cancer Messenger that acts in blood vessels and/or cells to promote inflammatory responses. [31] Inflammatory mediators that contribute to neoplastics include prostaglandins, inflammatory cytokines such as IL-1β, TNF-α, IL-6 and IL-15, and shimikins such as IL-8 and GRO-alpha. [32] [29] These inflammatory mediators, and others, regulate an environment that breeds proliferation and survival. [28] Inflammation also causes DNA damage caused by induction of reactive oxygen species (ROS) by various intracellular inflammatory mediators. [28] [29] In addition, leukocytes and other phagocytic cells absorbed into the site of inflammation induce DNA damage in proliferating cells through their generation of ROS and reactive nitrogen species (RNS). ROS and RNS are typically produced by these cells to fight infection. [28] ROS, alone, cause more than 20 types of DNA damage. [33] Oxidative DNA damage causes both mutations[34] and epigenetic changes. [35] [29] [36] RNS also causes mutagenic DNA damage. [37] A normal cell may undergo carcinogens to become a cancer cell if it is often subjected to DNA damage over long periods of chronic inflammation. DNA damage may cause genetic mutations due to improper repair. In addition, mistakes in the DNA repair process may cause epigenetic changes. [29] [32] Mutations and epigenetic changes that multiply and provide a selective advantage during somatic cell proliferation may be carcinogenic. Extensive genome analysis of human cancer tissues suggests that a single regular cancer cell may have almost 100 mutations in coding areas, of which 10 to 20 are driver mutations that contribute to cancer development. [29] However, chronic inflammation also causes epigenetic changes such as DNA methylation, which is often more common than mutations. Typically, several hundred to thousands of genes are methylated in a cancer cell (see DNA methylation in cancer). Oxidative damage sites in chromatin can absorb complexes that contain DNA methyl transferases (DNMTs), a histone diacetylase (SIRT1), and a histone methyl transferase (EZH2), thereby inducing DNA methylation. [29] [38] [39] DNA methylation of a CpG island in a promoting area may cause its downstream gene silencing (see CpG site and transcription regulation in cancer). DNA repair genes, in particular, are often deactivated by methylation in different cancers (see hypermethylation of DNA repair genes in cancer). A 2018 report assessed the relative importance of mutations and epigenetic changes in progression to two different types of cancer. The report found that epigenetic changes are far more important than mutations in the production of stomach cancers (associated with inflammation). [41] However, mutations and epigenetic changes of nearly equal importance in the production of esophageal squamo cell cancer (in association with Chemicals and staldehyde, the product of alcohol metabolism). HIV and AIDS have long been recognized that HIV infection is marked not only by the development of profound immunodeficiency but also by sustained inflammation and immune activation. [42] [43] [44] Significant body evidence implies chronic inflammation as a critical driver of immune dysfunction, premature appearance of aging-related diseases, and immune deficiencies. [42] [45] Many currently consider HIV infection not only as an immunodeficiency caused by the evolving virus but also as a chronic inflammatory disease. [46] Even after the introduction of effective anti-retroviral therapy (ART) and effective suppression of virma in hiv-infected individuals, chronic inflammation persists. Animal studies also support the relationship between immune activation and progressive cellular immune deficiency: SIVsm infection of its natural inhumane primer hosts, Mangaby Sussi, causes high-level viral replication but limited evidence of the disease. [47] This lack of pathogenicity is associated with no inflammation, immunity activation and cell proliferation. In contrast to severe, the experimental SIVsm infection of Rasus Macak produces immune activation and AIDS-like disease in many parallels with human HIV infection. [49] Delineating how CD4 T cells are depleted and how chronic inflammation and immune activation are induced lies at the heart of understanding HIV pathogenesis–one of the top priorities for HIV research by the Office of AIDS Research, National Institutes of Health. Recent studies have shown that piroptus, mediated by Caspaz-1, a highly inflammatory form of programmed cell death, drives the depletion and inflammation of CD4 T cells by HIV. [50] [51] These are two signature events that push the progression of HIV to AIDS. Pyroptos appears to create a pathogenic mecolytic cycle in which dying CD4 T cells and other immune cells (including macrophages and neutrophils) release inflammatory signals that absorb more cells into infected lymphoid tissues to die. The nature of forward feed produces this inflammatory response of chronic inflammation and tissue damage. [53] The identification of pyroptus as a main mechanism that depletes CD4 T cell and chronic inflammation provides exquisite therapeutic opportunities, namely caspaz-1, which controls the pyroptotic pathway. In this regard, pyroptosis of CD4 T cells and secretion of pro-inflmammatory cytokines such as IL-1β and IL-18 can be blocked in HIV-infected human lymphoid tissues by adding the Caspaz-1 VX-765 inhibitor,[50] which has already proven to be safe and well tolerated in the second phase of human clinical trials. [54] These findings could act the development of a whole new class of anti-AIDS treatments that target hosts rather than viruses. Such factors are almost certainly used in combination Art. By promoting the virus's tolerance rather than suppressing its proliferation, VX-765 or related drugs may mimic evolutionary solutions occurring on multiple monkey hosts (such as mangaby-whistle) infected with specific lenotyros of a species that has led to disease deficiency, failure to reduce the number of CD4 T cells, and without chronic inflammation. Solving inflammation of the inflammatory response should be actively terminated when no longer needed to prevent unnecessary spectator damage to tissue. [8] Failure to do so leads to chronic inflammation, and cell destruction. Dissolving inflammation occurs by different mechanisms in different tissues. The mechanisms that serve to end inflammation include: [8][55] short half-life of inflammatory mediators in vivo. Production and beta release of metamorphic growth factor (TGF) from macrophages[57][58] production and release of il-10 (IL-10)[59] mediator production Specialized anti-inflammatory proresolving, i.e. lipoxines, resolins, marcins and neuroprotectins[60][61] regulates the low regulation of pro-inflammatory molecules, such as leukotrins. Upregulation of anti-inflammatory molecules such as the interleukin 1 receptor antagonist or the soluble tumor necrosis factor receptor (TNFR) Apoptosis of pro-inflammatory cells[62] Desensitization of receptors. Increased cell survival in inflammatory regions due to their interaction with extracellular matrix (ECM)[64] low regulation of receptor activity with high concentration of quimoukin cluffing serapides by matrix metalloproteins (MMPs) may lead to the production of anti-inflammatory agents. [65] Acute inflammation is normally resolved by mechanisms that remain somewhat elusive. Emerging evidence now suggests that an active and coordinated program of resolution begins in the first few hours after the inflammatory response begins. After entering the tissues, granulocytes promote the switch of aracidonic acid-derived prostaglandins and leukothrins to lipoxines that initiate the termination sequence. Neutrophil recruitment thus stops and the planned death is involved by apoptosis. At the same time as biosynthesis, these events are polyunsaturated omega-3 fatty acids, from resolvins and protectors that severely shorten neutrophil penetration periods as apoptosis begins. As a result, apoptotic neutrophils are subjected to phage cytose by macrophages, leading to neutrophil cleansing and release of anti-inflammatory and compensatory cytokines such as growth factor-β1 conversion. The anti-inflammatory program ends with the withdrawal of macrophages through lymphatics. [66] — Charles Serhan connects to depression, there is evidence of a link between inflammation and depression. [67] Inflammatory processes can be initiated by negative cognitions or consequences, such as stress, violence, or deprivation. Negative cognitions can cause inflammation, which can in turn lead to depression. [68] [69] [Suspicious – Discussion] Additionally there is increasing evidence that inflammation can cause depression due to increased cytokines, regulating the brain into a state of disease. [70] Classic symptoms of being physically ill, such as letality, show a lot of overlap in behaviors that determine depression. Cytokine levels tend to increase sharply during episodes of depression in people with bipolar disorder and drop-off during recovery. [71] In addition, clinical trials have shown that anti-inflammatory drugs taken in addition to antidepressants not only significantly improve symptoms but also increase the proportion of people responding positively to treatment. [72] Inflammations that lead to serious depression can be caused by common infections such as infections caused by a virus, bacteria or even parasites. [73] The systemic effects of an infectious organism can escape the immediate tissue range through the circulatory system or lymphatic system, where it may spread to other parts of the body. If an organism is not inhibited by acute inflammation, it may access the lymphatic system through lymph vessels close to the lymphatic system. Lymphatic vein infection is known as lymphangiitis, and the infection of a lymph node is known as lymphadenitis. When lymph nodes cannot eliminate all pathogens, the infection spreads further. A pathogen can access the bloodstream through lymphatic drainage into the circulatory system. When inflammation overwhelms the host, systemic inflammatory response syndrome is diagnosed. When it is due to infection, the term sepsis is applied, with bacterial conditions that are specifically applied for bacterial sepsis and virma specifically to viral sepsis. Vasodilation and organ dysfunction are serious problems associated with widespread infection that may lead to septic shock and death. Inflammation of acute phase proteins also induces high systemic levels of acute phase proteins. In acute inflammation, these proteins prove beneficial, however, in chronic inflammation they can contribute to amyloidosis. [8] These proteins include reactive protein C, serum amyloid A, and serum amyloid P, which cause a range of systemic effects including:[8] Fever increased blood pressure reducing sweating malase loss appetite Somnolence leukocyte inflammation numbers often affect the number of leukocytes found in the body: leukocytosis is often seen during inflammation caused by infection , where it leads to a large increase in the amount of leukocytes in the blood, especially immature cells of leukocyte numbers, usually increases to between 15 and 20 000 cells per microliter, but severe cases can see it approaching 100,000 cells per microliter. [8] Bacterial infection usually leads to an increase in neutrophils, causing While diseases such as asthma, hay fever, and parasite infestations lead to an increase in axiphils and cause hosinophiles. [8] Leukopenia can be induced by certain infections and diseases, including viral infection, ricketsia infection, some protozoa, tuberculosis, and certain cancers. [8] Systemic inflammation and obesity developed with the discovery of interleukins (IL), the concept of systemic inflammation. Although the processes involved with tissue inflammation are the same, systemic inflammation is not encapsulated in a particular tissue but involves endothelium and other organ systems. Chronic inflammation is widely observed in obesity. [74] Obese people usually have many high markers of inflammation, including:[76][77] IL-6 (Interleukin-6)[78][79] IL-8 (Interleukin-8) IL-18 (Interleukin-18)[78][79] TNF-α [78] CRP (C-reactive protein)[78][79] insulin[78][79] blood glucose[78][79] leptin[78][79] low-grade chronic inflammation with an increase of two to three times the systemic concentration of cytokines such as TNF-α , IL-6, and CRP. [80] Waist circumference has a significant relationship with systemic inflammatory response. [81] Loss of white adipose tissue reduces levels of inflammation markers. [74] The relationship between systemic inflammation and insulin resistance and type 2 diabetes, and with atherosclerosis, is under preliminary investigation, although rigorous clinical trials have not been conducted to confirm such relationships. [82] C-reactive protein (CRP) is produced at a higher level in obese people, and may increase the risk of cardiovascular disease. [83] The results of the present wounds on the skin, evidence of fibrosis and wound healing result in a specific condition will be determined by the tissue where the damage occurred and the cause of the damage that caused it. Here are possible results to inflammation:[8] ResolutionThe complete repair of inflamed tissue returns to a normal condition. Inflammatory measures such as vasodilation, chemical production, and stopping the penetration of leukocytes, and damaged paranchymal cells are regenetrable. This is usually the result when limited or short-term inflammation has occurred. Amounts of large fibrosis degrade tissue, or damage in tissues that are unable to regeneenet, cannot be completely regeeneneted by the body. Fibrous ulcers occur in these areas of damage, forming a wound composed primarily of collagen. The wound will not contain any specialized structures such as paronchymal cells, so functional impairment may occur. The Abscess formationA cavitation contains pus, opaque liquid containing dead white blood cells and bacteria formed with general residues of destroyed cells. Chronic inflammation in acute inflammation, if the pathogen remains then chronic inflammation will follow. This process, specified by It takes days, months or even years, it may lead to the formation of a chronic wound. Chronic inflammation is determined by the dominant presence of macrophages in the affected tissue. These cells are powerful immune agents of the body, but released

toxins (including reactive oxygen species) are damaged for the organism's own tissues as well as invasive agents. As a result, chronic inflammation is almost always associated with tissue degradation. Examples of inflammation are usually shown by adding extensions, as shown below. However, some conditions such as asthma and asthma do not follow the convention. More examples are available on the list of types of inflammation. See acute appendicitis acute infectious meningitis acute tonsilitis as well as anaphylatoxin anti-inflammatory essential fatty acid interactions inflammatory reflux interleukin-lipoxine neurogenic inflammation substances P references ^ Ferrero-Miliani L, Nielsen OH, Andersen PS, Girardin SE; Nielsen; Andersen; Girardin (February 2007). Chronic inflammation: The importance of NOD2 and NALP3 in the interleukin-1 generation. klein . Exp. Immunol. 147 (2): 227–35. doi:10.1111/j.1365-2249.2006.03261.x. PMC 1810472. PMID 17223962.CS1 maint: multiple names: authors list (link) ^ Abbas A.B.; Lichtman A.H. (2009). Ch.2 Innate Immunity. In Saunders (Elsevier) (ed.). immunology . Functions and disorders of the immune system (3rd ed.). ISBN 978-1-4160-4688-2. ^ a b c d Hall, John (2011). Guyton and Hall textbook of medical physiology (12th ed.). Philadelphia, Pa.: Saunders/Elsevier. p. 428. ISBN 978-1-4160-4574-8. ^ Granger, D. Neil; Senchenkova, Elena (2010). Leukocyte–Endothelial Cell Adhesion. Inflammation and microsycrolation. Physiology of integrated systems—from cell to function. Morgan & Claypool Life Sciences. ^ Piira, Olli-Pekka; Miettinen, Johanna A.; Hautala, Arto J.; Huikuri, Heikki V.; Tulppo, Mikko P. (2013). Physiological responses to emotional excitement in healthy individuals and patients with coronary artery disease. Selfnomic neuroscience. 177 (2): 280–5. doi:10.1016/j.autneu.2013.06.001. PMID 23916871. S2CID 19823098. ^ a b Stedman's Medical Dictionary (Twenty-fifth ed.). Williams & Wilkins. 1990. ^ Rather, L. J. (1971). Disturbance of function (functio laesa): the legendary fifth cardinal sign of inflammation, added by Galen to the four cardinal signs of Celsus. Bull N Y Acad Med. 47 (3): 303–322. PMC 1749862. PMID 5276838. ^ a b c d e f g h i j k l m n o p q r Cotran; Kumar, Collins (1998). Robbins pathology is the basis of the disease. Philadelphia: W.B Saunders Company. ISBN 978-0-7216-7335-6. ^ Kumar, Rukmini; Clermont, Gilles; Vodovotz, Yoram; Chow, Carson C. (21 September 2004). The dynamics of acute inflammation. Journal of Theoretical Biology. 230 (2): 145–155. arXiv:q-bio/0404034. doi:10.1016/j.jtbi.2004.04.044. PMID 15321710. S2CID 16992741. ^ a b c d Parakrama Chandrasoma; Clive R. Taylor (2005). Part A. General Pathology, Section II. The Host Response to Injury, Chapter 3. The Acute Inflammatory Response, sub-section Cardinal Clinical Signs. 3) آسیب شناسی پنهانrd ed.). مک گرو هیل ISBN 978-0-8385-1499-3. OCLC 150148447. Retrieved 5 November 2008. ^ Werner, Ruth (2009). 4) ماساژ درمانگر راهنمای آسیب شناسیth ed.). ولترز کلوور ISBN 978-0781769198. ^ Vogel, Wolfgang H.; Berke, Andreas (2009). تاریخچه مختصر بینایی و پزشکی چشمی. انتشارات کوگلر. p. 97. ISBN 978-90-6299-220-1. ^ Porth, Carol (2007). ملزومات pahtophysiology: مفاهیم دولت های بهداشتی تغییر یافته. Hagerstown, MD: Lippincott Williams & Wilkins. p. 270. ISBN 978-0-7817-7087-3. ^ Dormandy, Thomas (2006). بدترین شرارت ها: مبارزه انسان با درد. New Haven, Conn: Yale University Press. pp. 22. ISBN 978-0-300-11322-8. ^ a b Herrington, Simon (2014). 15) کتاب درسی آسیب شناسی مویر ed.). CRC Press. p. 59. ISBN 978-1444184990. ^ Cevikbas, Ferda; Kempkes, Cordula; Buhl, Timo; ظروف سرباز یا مسافر، مسیحی؛ Buddenkotte, Joerg; Steinhoff, Martin (2014). Carstens, E.; Akiyama, Tasuku (eds.). خارش: مکانیسم ها و درمان. مرزها در علوم اعصاب. Boca Raton (FL): CRC Press/Taylor & Francis. ISBN 9781466505438. PMID 24830021. ^ Caughey, George H. (1 June 2007). Mast cell tryptases and chymases in inflammation and host defense. 154–141 :(1) 217 .بررسی های ایمونولوژیک. doi:10.1111/j.1600-065x.2007.00509.x. PMC 2275918. PMID 17498057. ^ Caughey, George H. (5 May 2016). Mast cell proteases as pharmacological targets. European Journal of Pharmacology. 55–44 :778. مدولاسیون فارماکولوژیک سلول های دکل و بازوفیل ها. doi:10.1016/j.ejphar.2015.04.045. PMC 4636979. PMID 25958181. ^ Libby, P (19–26 December 2002). Inflammation in atherosclerosis. 74–868 :(6917) 420 .طبیعت. Bibcode:2002Natur.420.. 868L. doi:10.1038/nature01323. PMID 12490960. S2CID 407449. ^ Wiedermann U, et al. (1996). کمبود ویتامین A پاسخ های التهابی را افزایش می دهد. اسکن J Immunol. 44 (6): 578–584. doi:10.1046/j.1365-3083.1996.d01-351.x. PMID 8972739. S2CID 3079540. ^ Hargrave, B. Y.; Tiangco, D. A.; Lattanzio, F. A.; Beebe, S. J. (2003). در سلول های قلبی NF-κB کوکائین، نه مورفین، باعث تولید گونه های واکنش پذیر اکسیژن و فعال شدن cotransfected. 151–141 :(2) 3 سمیول قلبی. doi:10.1385/CT:3:2:141. PMID 14501032. S2CID 35240781. ^ Montiel-Duarte, C.; Ansorena, E.; López-Zabalza, M. J.; Cenarruzabeitia, E.; Iraburu, M. J. (2004). نقش گونه های فعال اکسیژن، گلوتاتیون و NF-κB در آپوپتوز ناشی از 3,4-متیلن دی اکسی مت آمفتامین (اکستازی) بر روی سلول های استلات کبدی. بیوشیمیای فارماکول . 67 :(6) 33–1025. doi:10.1016/j.bcp.2003.10.020. PMID 15006539. ^ Hendrik Ungefroren; سبنس؛ دانیل سیدل؛ هندریک لهنرت؛ Ralf Haas (2011). Interaction of tumor cells with the microenvironment. 18) 9 (ارتباط سلولی و سیگنالینگ. doi:10.1186/1478-811X-9-18. PMC 3180438. PMID 21914164. ^ Coussens, L.M.; Werb, Z. (2002). التهاب و سرطان. 7–860 :(6917) 420 .طبیعت. Bibcode:2002Natur.420.. 860C. doi:10.1038/nature01322. PMC 2803035. PMID 12490959. ^ Gunn, L; Ding, C; Liu, M; Ma, Y; Qi, C; Cai, Y; Hu, X; آگگروال، د؛ ژانگ, HG; Yan, J (15 September 2012). نقش های مخالف برای مکمل جزء C5a در پیشرفت تومور و میکرو ویروس تومور. Journal of Immunology. 189 (6): 2985–94. doi:10.4049/jimmunol.1200846. PMC 3436956. PMID 22914051. ^ Copland, JA; Sheffield-Moore, M; Koldzic-Zivanovic, N; Gentry, S; Lamprou, G; Tzortzatou-Stathopoulou, F; Zoumpourlis, V; Urban, RJ; Vlahopoulos, SA (June 2009). گیرنده های استروئید جنسی در تشخیص اسکلتی و تئوپلاسی اپی تلیال: آیا مداخله خاص بافت ممکن است؟. BioEssays. 31 (6): 629–41. doi:10.1002/bies.200800138. PMID 19382224. S2CID 205469320. ^ Colotta F, Allavena P, Sica A, Garlanda C, Mantovani A; Allavena; سیکا؛ Garlanda; Mantovani (July 2009). Cancer-related inflammation, the seventh hallmark of cancer: links to genetic instability. 81–1073 :(7) 30 .کارسینوژنز (بازبینی). doi:10.1093/carcin/bgp127. PMID 19468060.CS1 maint: multiple names: authors list (link) ^ a b c d Coussens LM, Werb Z (2002). 7–860 :(6917) 420 .التهاب و سرطان. طبیعت. Bibcode:2002Natur.420.. 860C. doi:10.1038/nature01322. PMC 2803035. PMID 12490959. ^ a b c d e f g h Chiba T, Marusawa H, Ushijima T (2012). التهاب مرتبط با توسعه سرطان در اندام های گوارشی: مکانیسم ها و نقش ها برای مدولاسیون ژنتیکی و اپی ژنتیک (PDF). 563–550 :(3) 143 .گوارش. doi:10.1053/j.gastro.2012.07.009. hdl:2433/160134. PMID 22796521. ^ Mantovani A, Allavena P, Sica A, Balkwill F (2008). Cancer-related inflammation (PDF). 44–436 :(7203) 454 .طبیعت. Bibcode:2008Natur.454.. 436M. doi:10.1038/nature07205. hdl:2434/145688. PMID 18650914. S2CID 4429118. ^ Larsen GL, Henson PM (1983). Mediators of inflammation. 59–335 :1 . آنو . برگرد ایمونول. doi:10.1146/annurev.iy.01.040183.002003. PMID 6399978. ^ a b c d Shacter E, Weitzman SA (2002). التهاب مزمن و سرطان. انکولوژی (Williston Park, N.Y.). 16 (2): 217–26, 229, discussion 230–2. PMID 11866137. ^ Yu Y, Cui Y, Niedernhofer LJ, Wang Y (2016). Occurrence, Biological Consequences, and Human Health Relevance of Oxidative Stress-Induced DNA Damage. Chem. Res, Toxicol. 29 (12): 2008–2039. doi:10.1021/acs.chemrestox.6b00265. PMC 5614522. PMID 27989142. ^ Dizdaroglu M (2012). Oxidatively induced DNA damage: mechanisms, repair and disease. 47–26 :(2–1) 327 . سرطان لت. doi:10.1016/j.canlet.2012.01.016. PMID 22293091. ^ Nishida N, Kudo M (2013). اکسیداتیو and epigenetic instability in human hepatocarcinogenesis. Dig Dis. 31 (5–6): 447–53. doi:10.1159/000355243. PMID 24281019. ^ a b Ding N., Maiuri A. R., O'Hagan H.M. (2017). نقش نوظهور اصلاح کننده های اپی ژنتیک در ترمیم آسیب DNA مرتبط با بیماری های التهابی مزمن. تحقیقات جهش. 780 :81–69. doi:10.1016/j.mrrev.2017.09.005. PMC 6690501. PMID 31395351.CS1 maint: multiple names: authors list (link) ^ Kawanishi S, Ohnishi S, Ma N, Hiraku Y, Oikawa S, Murata M (2016). Nitrative and oxidative DNA damage in infection-related carcinogenesis in relation to cancer stem cells. 26 :38 .ژن های انیورون. doi:10.1186/s41021-016-0055-7. PMC 5203929. PMID 28050219. ^ O'Hagan HM, Wang W, Sen S, Destefano Shields C, Lee SS, Zhang YW, Clements EG, Cai Y, Van Neste L, Easwaran H, Casero RA, Sears CL, Baylin SB (2011). Oxidative damage targets complexes containing DNA methyltransferases, SIRT1, and polycomb members to promoter CpG Islands. سلول سرطانی 19–606 :(5) 20 .. doi:10.1016/j.ccr.2011.09.012. PMC 3220885. PMID 22094255. ^ Maiuri AR, Peng M, Sriramkumar S, Kamplain CM, DeStefano Shields CE, Sears CL, O'Hagan HM (2017). Mismatch Repair Proteins Initiate Epigenetic Alterations during Inflammation-Driven Tumorigenesis. سرطان Res. 77 (13): 3467–3478. doi:10.1158/0008-5472.CAN-17-0056. PMC 5516887. PMID 28522752. ^ Yamashita S, Kishino T, Takahashi T, Shimazu T, Charvat H, Kakugawa Y, Nakajima T, Lee YC, Iida N, Maeda M, Hattori N, Takeshima H, Nagano R, Oda I, Tsugane S, Wu MS, Ushijima T (2018). تغییرات ژنتیکی و اپی ژنتیک در بافت های طبیعی اثرات دی دی دیال بر خطر ابتلا به سرطان در میان بافت ها. Proc. Natl. Acad. Sci. U.S.A. 115 (6): 1328–1333. doi:10.1073/pnas.1717340115. PMC 5819434. PMID 29358395. ^ Raza Y, Khan A, Farooqui A, Mubarak M, Facista A, Akhtar SS, Khan S, Kazi JI, Bernstein C, Kazmi SU (2014). Oxidative DNA damage as a potential early biomarker of Helicobacter pylori associated carcinogenesis. پاتول . انکول . Res. 20 (4): 839–46. doi:10.1007/s12253-014-9762-1. PMID 24664859. S2CID 18727504. ^ a b Deeks, Steven G. (1 January 2011). HIV infection, inflammation, immunosenescence, and aging. 155–141 :62 .بررسی سالانه پزشکی. doi:10.1146/annurev-med-042909-093756. PMC 3759035. PMID 21090961. ^ Klatt, Nichole R.; چومونت، نیکولا؛ Douek, Daniel C.; Deeks, Steven G. (1 July 2013). Immune activation and HIV persistence: implications for curative approachs to HIV infection. 342–326 :(1) 254 .بررسی های ایمونولوژیک. doi:10.1111/imr.12065. PMC 3694608. PMID 23772629. ^ Salazar-Gonzalez, J. F.; Martinez-Maza, O.; Nishanian, P.; عزیز، ن.؛ Shen, L. P.; Grosser, S.; Taylor, J.; Detels, R.; Fahey, J. L. (1 August 1998). افزایش و افزایش بار ویروس سرم در عفونت ویروس نقص ایمنی انسان CD4 T فعال سازی ایمنی پیش از نقطه هجوم سلول های The 430–423 :(2) 178 .از بیماری های عفونی. doi:10.1086/515629. PMID 9697722. ^ Ipp, Hayley; Zemlin, Annalise (1 February 2013). هنگامی که التهاب مضر می شود HIV: پارادوکس پاسخ ایمنی در عفونت. Clinica Chimica Acta; International Journal of Clinical Chemistry. 416: 96–99. doi:10.1016/j.cca.2012.11.025. PMID 23228847. ^ Nasi, Milena; Pinti, Marcello; Mussini, Cristina; Cossarizza, Andrea (1 October 2014). التهاب مداوم در عفونت HIV: 8–184 :(2) 161 .مفاهیم تثبیت شده، دیدگاه های جدید. نامه های ایمونولوژی. doi:10.1016/j.imlet.2014.01.008. PMID 24487059. ^ Milush, Jeffrey M.; Mir, Kiran D.; Sundaravaradan, Vasudha; Gordon, Shari N.; Engram, Jessica; Cano, Christopher A.; Reeves, Jacqueline D.; آنتون، الیزابت؛ O'Neill, Eduardo (1 March 2011). دو منفی همراه است T قابل توجهی با سلول های CD4+ T با از دست دادن سلول SIV عدم وجود ایدز بالینی در منگابی های سوت آلوده به. The Journal of Clinical Investigation. 121 (3): 1102–10. doi:10.1172/JCI44876. PMC 3049370. PMID 21317533. ^ Rey-Cuillé, M. A.; Berthier, J. L.; Bomsel-Demontoy, M.C.; Chaduc, Y.; Montagnier, L.; Hovanessian, A. G.; Chakrabarti, L. A. (1 May 1998). Simian immunodeficiency virus replicates to high levels in sooty mangabeys without inducing disease. Journal of Virology. 72 (5): 3872–86. doi:10.1128/JVI.72.5.3872-3886.1998. PMC 109612. PMID 9557672. ^ Chahroudi, Ann; Bosinger, Steven E.; Vanderford, Thomas H.; Paiardini, Mirko; Silvestri, Guido (9 March 2012). Natural SIV hosts: showing AIDS the door. 93–1188 :(6073) 335 .علم. Bibcode:2012Sci... 335.1188C. doi:10.1126/science.1217550. PMC 3822437. PMID 22403383. ^ a b Doitsh, Gilad; Galloway, Nicole L. K.; Geng, Xin; یانگ، ژیوان؛ Monroe, Kathryn M.; Zepeda, Orlando; هانت، پیتر دبلیو. هاتانو، هیرویو؛ Sowinski, Stefanie (23 January 2014). مرگ سلولی توسط pyroptosis درایوهای CD4 کاهش سلول T در عفونت HIV-1. 514–509 :(7484) 505 .طبیعت. Bibcode:2014Natur.505.. 509D. doi:10.1038/nature12940. PMC 4047036. PMID 24356306. ^ Monroe, Kathryn M.; یانگ، ژیوان؛ Johnson, Jeffrey R.; Geng, Xin; Doitsh, Gilad; Krogan, Nevan J.; Greene, Warner C. (24 January 2014). IFI16 DNA sensor is required for death of lymphoid CD4 T cells abortively infected with HIV. 432–428 :(6169) 343 .علم. Bibcode:2014Sci... 343..428M. doi:10.1126/science.1243640. PMC 3976200. PMID 24356113. ^ Galloway, Nicole L. K.; Doitsh, Gilad; Monroe, Kathryn M.; یانگ، ژیوان؛ Muñoz-Arias, Isa; Levy, David N.; Greene, Warner C. (8 September 2015). انتقال سلول به سلول HIV-1 مورد نیاز است به ماشه مرگ Pyroptotic سلول های لنفوئید بافت مشتق از CD4 T. 63–1555 :(10) 12 . گزارش هاي سلولي. doi:10.1016/j.celrep.2015.08.011. PMC 4565731. PMID 26321639. ^ Doitsh, Gilad; Greene, Warner C. (9 March 2016). Dissecting How CD4 T Cells Are Lost During HIV Infection. Cell Host & Microbe. 19 280–291. doi:10.1016/j.chom.2016.02.012. PMC 4835240. PMID 26962940. ^ Study of VX-765 in Subjects With Treatment-resistant Partial Epilepsy - Full Text View - ClinicalTrials.gov. clinicaltrials.gov. Retrieved 21 May 2016. ^ Eming, S. A.; Krieg, T.; Davidson, J.M. (2007). التهاب در ترمیم زخم: مکانیسم های مولکولی و سلولی. Journal of Investigative Dermatology. 127 (3): 514–525. doi:10.1038/sj.jid.5700701. PMID 17299434. ^ Ashcroft, G. S.; یانگ، ایکس؛ Glick, A.B.; Weinstein, M; Letterio, J. L.; Mizel, D. E.; Anzano, M; Greenwell-Wild, T; Wahl, S.M.; دنگ، سی؛ Roberts, A.B. (1999). موش فاقد Smad3 نشان می دهد بهبود زخم شتاب و اختلال در پاسخ التهابی محلی. Nat Cell Biol. 1 (5): 260–6. doi:10.1038/12971. PMID 10559937. S2CID 37216623. ^ Ashcroft, G. S. (1999). تنظیم دو راهی تابع ماکروفاژ توسط TGF-β. 82–1275 :(15) 1 .میکروب ها آلوده می شوند. doi:10.1016/S1286-4579(99)00257-9. PMID 10611755. ^ Werner, F; Jain, M. K.; Feinberg, M. W.; Sibinga, N. E.; Pellacani, A; Wiesel, P; Chin, M. T.; Topper, J. N.; Perrella, M. A.; Lee, M. E. (2000). میانجیگری می شود Smad3 مهار فعال سازی ماکروفاژ از طریق β1-تبدیل فاکتور رشد. J Biol Chem. 275 (47): 36653–8. doi:10.1074/jbc. M004536200. PMID 10973958. ^ Sato, Y.; Ohshima, T.; Kondo, T. (1999). نقش تنظیمی اینترلوکین-10 اندوژن در پاسخ التهابی جلدی بهبود زخم مورین. Biochem Biophys Res Commun. 265 (1): 194–9. doi:10.1006/bbrc.1999.1455. PMID 10548513. ^ Serhan, C. N. (2008). Controlling the resolution of acute inflammation: a new genus of dual anti-inflammatory and proresolving mediators. J Periodontol. 79 (8 Suppl): 1520–6. doi:10.1902/jop.2008.080231. PMID 18673006. ^ Headland SE, Norling LV (2015). 60–149 :(3) 27 .حل التهاب: اصول و چالش ها. سمینارها در ایمونولوژی. doi:10.1016/j.smim.2015.03.014. PMID 25911383. ^ Greenhalgh, D. G. (1998). The role of apoptosis in wound healing. Int J Biochem Cell Biol. 30 (9): 1019–30. doi:10.1016/S1357-2725(98)00058-2. PMID 9785465. ^ Jiang, D; Liang, J; Fan, J; Yu, S; Chen, S; Luo, Y; Prestwich, G. D.; Mascarenhas, M.M.; Garg, H. G.; Quinn, D. A.; هومر، ر. ج؛ Goldstein, D. R.; Bucala, R; Lee, P. J.; Medzhitov, R; Noble, P. W. (2005). تنظیم آسیب ریه و ترمیم توسط گیرنده های عوارض مانند و هیالورنان. Nat Med. 11 (11): 1173–9. doi:10.1038/nm1315. PMID 16244651. S2CID 11765495. ^ Teder, P. (2002). Resolution of lung inflammation by CD44. 8–155 :(5565) 296 .علم. Bibcode:2002Sci... 296..155T. doi:10.1126/science.1069659. PMID 11935029. S2CID 7905603. ^ McQuibban, G. A.; گونگ، جی. Tam, E.M.; McCulloch, C. A.; کلارک-لوئیس، من؛ به طور کلی c.M. (2000). Inflammation dampened by gelatinase A cleavage of monocyte chemoattractant protein-3. 6–1202 :(5482) 289 .علم. Bibcode:2000Sci... 289.1202M. PMID 10947989. ^ Serhan CN, Savill J; Savill (2005). Resolution of inflammation: the beginning programs the end. 7–1191 :(12) 6 نات ایمونول. doi:10.1038/ni1276. PMID 16369558. S2CID 22379843. ^ Berk, M; Williams, L. J.; Jacka, F. N.; O'Neil, A; Pasco, J. A.; Moylan, S; Allen, N.B.; Stuart, A. L.; Hayley, A.C.; Byrne, M. L.; Maes, M (2013). پس افسردگی یک بیماری التهابی است، اما التهاب از کجا می آید؟. BMC Medicine. 11: 200. doi:10.1186/1741-7015-11-200. PMC 3846682. PMID 24228900. ^ Cox, William T. L.; Abramson, Lyn Y.; Devine, Patricia G.; Hollon, Steven D. (2012). Stereotypes, Prejudice, and Depression: The Integrated Perspective. 449–427 :(5) 7 .دیدگاه هایی در مورد علوم روان شناسی. doi:10.1177/1745691612455204. PMID 26168502. S2CID 1512121. ^ Kiecolt-Glaser, Janice K.; Derry, Heather M.; Fagundes, Christopher P. (November 2015). Inflammation: Depression Fans the Flames and Feasts on the Heat. American Journal of Psychiatry. 172 (11): 1075–91. doi:10.1176/appi.ajp.2015.15020152. PMC 6511978. PMID 26357876. ^ Williams, Caroline (4 January 2015). آیا افسردگی نوعی واکنش آلرژیک است؟. نگهبان. ^ Brietzke, Elisa; استرتز، لورا؛ Fernandes, Brisa Simões; Kauer-Sant'Anna, Marcia; Mascarenhas, Marcello; Escosteguy Vargas, Andréia; Chies, José Artur; Kapczinski, Flávio (2009). مقایسه سطوح سیتوکین در بیماران افسرده، مانیک و یوتمیک مبتلا به اختلال دوقطبی. Journal of Affective Disorders. 116 (3): 214–7. doi:10.1016/j.jad.2008.12.001. PMID 19251324. ^ Müller, N; Schwarz, M J; Dehning, S; Douhe, A; Cerovecki, A; Goldstein-Müller, B; Spellmann, I; Hetzel, G; Maino, K; Kleindienst, N; Möller, H-J; Arolt, V; Riedel, M (2006). مهارکننده سیکلوکسیژناز-2 سلکوکسیب اثرات درمانی در افسردگی عمده دارد: نتایج یک مطالعه دو سوکور، تصادفی، دارونما کنترل شده، مطالعه آزمایشی افزودنی به ریبوکستین. روانپزشکي مولکولي . 11 :(7) 4–680. doi:10.1038/sj.mp.4001805. PMID 16491133. ^ Canli, Turhan (2014). Reconceptualizing major depressive disorder as an infectious disease. Biology of Mood & Anxiety Disorders. 4: 10. doi:10.1186/2045-5380-4-10. PMC 4215336. PMID 25364500. ^ a b Parimisetty A, Dorsemans AC, Awada R, Ravanan P, Diotel N, Lefebvre d'Hellencourt C (24 March 2016). صحبت مخفی بین بافت چربی و سیستم عصبی مرکزی از طریق عوامل ترشح شده- مرز در حال ظهور در تحقیقات نورودژنراتیو. J Neuroinflammation (Review). 13 (1): 67. doi:10.1186/s12974-016-0530-x. PMC 4806498. PMID 27012931. ^ Kershaw, E. E.; Flier, J. S. (2004). بافت چربی به عنوان عضو غدد درون ریز. J Clin Endocrinol Metab. 89 (6): 2548–56. doi:10.1210/jc.2004-0395. PMID 15181022. ^ Bastard J, et al. (2000). بالا رفتن سطح اینترلوکین 6 در بافت چربی سرم و زیر جلدی زنان چاق پس از کاهش وزن کاهش می یابد. J Clin Endocrinol Metab. 85 (9): doi:10.1210/jc.85.9.3338. PMID 10999830. ^ Mohamed-Ali V, et al. (2001). beta-Adrenergic regulation of IL-6 release from adipose tissue: in vivo and in vitro studies. J Clin Endocrinol Metab. 86 (12): 5864–9. doi:10.1210/jc.86.12.5864. PMID 11739453. ^ a b c d e f g Loffreda, S; Yang, S. Q.; Lin, H. Z.; Karp, C. L.; Brengman, M. L.; Wang, D. J.; Klein, A. S.; Bulkley, G.B.; Bao, C; نجیب، پی. دبلیو. Lane, M. D.; Diehl, A.M. (1998). Leptin تنظیم پاسخ های ایمنی التهابی. FASEB J. 12 (1): 57–65. doi:10.1096/fasebj.12.1.57. PMID 9438411. ^ a b c d e f g Esposito, K; Nappo, F; Marfella, R; Giugliano, G; Giugliano, F; Ciotola, M; Quagliaro, L; Ceriello, A; Giugliano, D (2002). غلظت سیتوکین التهابی به شدت توسط هیپرگلیسمی در انسان افزایش می یابد: نقش استرس اکسیداتیو. گردش. 106 :(16) 72–2067. doi:10.1161/01.CIR.0000034509.14906.AE. PMID 12379575. ^ Petersen, A.M.; Pedersen, B. K. (2005). The anti-inflammatory effect of exercise. J Appl Physiol. 98 (4): 1154–62. doi:10.1152/japplphysiol.00164.2004. PMID 15772055. S2CID 4776835. ^ Rogowski, O; شاپیرا، من؛ Bassat, O. K.; Chundadze, T; Finn, T; Berliner, S; Steinvil, A (2010). دور کمر به عنوان عمدتا به پاسخ میکرو التهابی در سندرم متابولیک: یک مطالعه مقطعی. Journal of Inflammation. 7: 35. doi:10.1186/1476-9255-7-35. PMC 2919526. PMID 20659330. ^ Goldfine, Allison B.; Shoelson, Steven E. (3 January 2017). رویکردهای درمانی هدف قرار دادن التهاب برای دیابت و خطر قلبی عروقی مرتبط. Journal of Clinical Investigation. 127 (1): 83–93. doi:10.1172/jci88884. ISSN 0021-9738. PMC 5199685. PMID 28045401. ^ Choi, J.; Joseph, L.; Pilote, L. (2013). Obesity and C-reactive protein in various populations: a systematic review and meta-analysis. 244–232 :(3) 14 .بررسی چاقی. doi:10.1111/obr.12003. ISSN 1467-7881. PMID 23171381. S2CID 206227739. پیوندهای خارجی ویکی مدیا کامنز دارای رسانه های مربوط به التهابات است. التهاب در کتابخانه ملی پزشکی ایالات متحده عنوان موضوع پزشکی (MeSH) بازیابی شده از