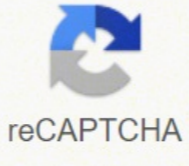
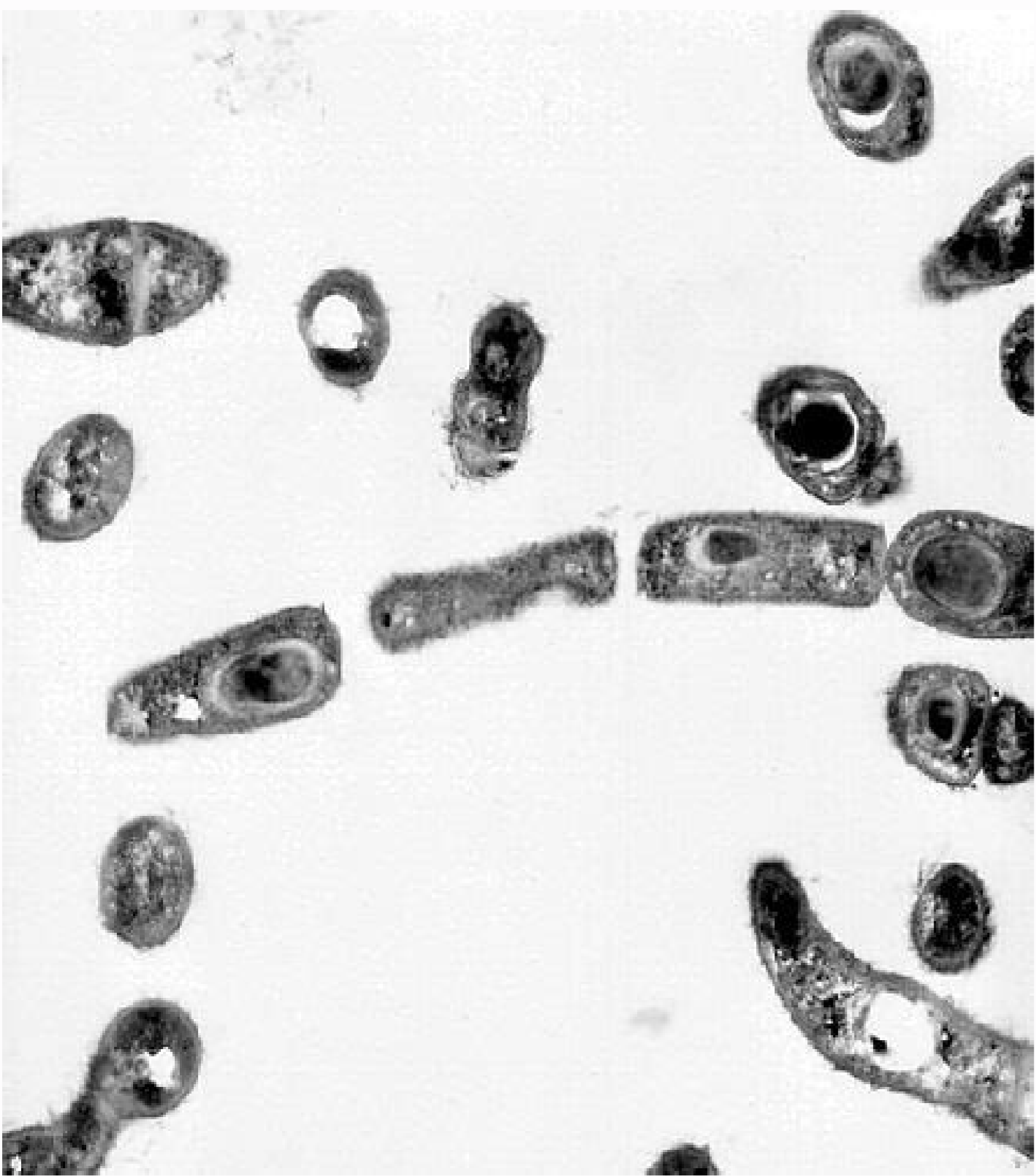
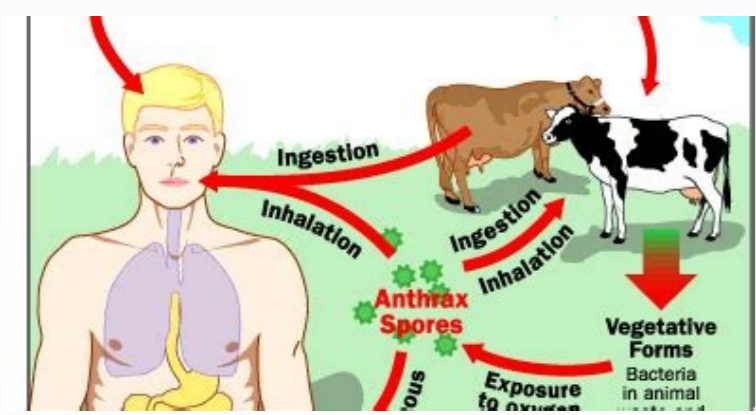
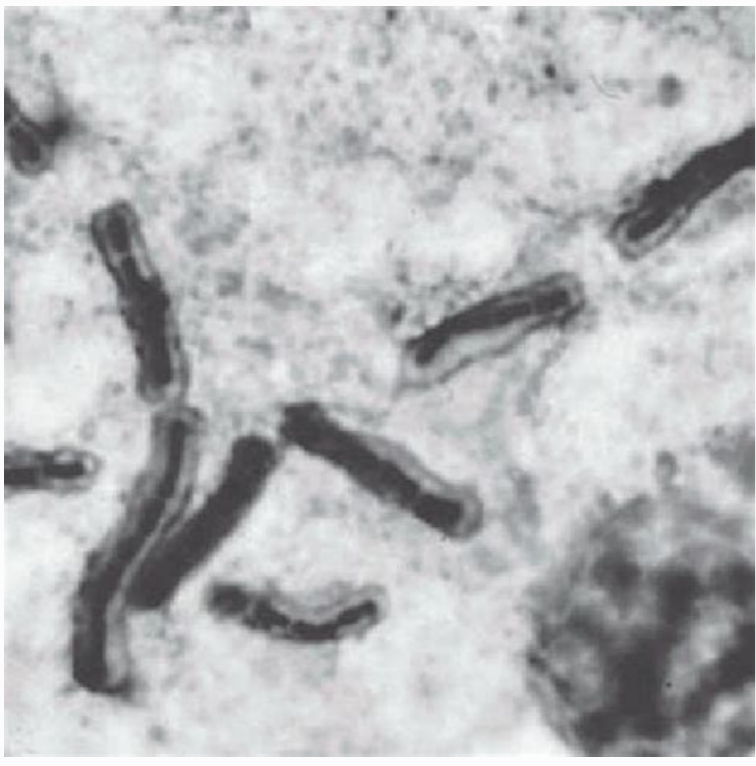




I'm not robot



Next



WHO GETS IT?

- Animals primarily
- Common in...
- South & Central America
- Southern/Eastern Europe
- Asia
- Africa
- Middle East



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[5]]7[Until the twentieth century, anthrax infections killed hundreds of thousands of people and animals every year. [14] Anthrax has been developed as a weapon by a number of In herbivor animals, infection occurs when they eat or breathe the spores while grazing. [11] Animals can become infected by eating infected animals. [11] Etymology The name in English comes from the Antrax (Å "Å¼½", the Greek word for carbon, [15]]16[possibly having Egyptian etymology, [17] due to the characteristics Black skin injuries developed by victims with a cutaneous anger infection. The black central debris surrounded by a vivid red skin has been recognized for a long time as a typical of the disease. The first recorded use of the word " Anthrax "in English is in a translation 1398 by Bartholomaeus Anglicus' Work by Proprietiubus Rem (about the properties of things, 1240). [18] The Intrax was historically known for a wide variety of names that indicate their Symptoms, location and groups considered more vulnerable to infection. They included Siberian plague, Cumberland's disease, carbon, splient fever, malignant edema, Woilsorter's disease and Bradford's disease. [19] Signs and Symptoms The lesion of the skin of the Antrax skin ventrax lesion in the cutaneous neck of the neck, also known as Hide-Porter's disease, is when the Antrax occurs in the skin . It is the most common way (> 90% of the ventrax cases). It is the least dangerous (low mortality with treatment, 23.7% of mortality without). [20]]5[The cutaneous Intrax presents as a skin lesion in the form of boiled that eventually forms a ultrasound with a black center (eschar). Black scarce is often shown as a large and painless necrochorate (starting as an irritating lesion or blister and skin that is dark and generally concentrated as a black spot, a little resembling the bread mold) in the infection site. In general, Cutaneous are formed within the penetration site of spores between two and five days after the exhibition. Unlike bruises or most of the other injuries, infections in the cutaneous ventrax usually do not cause pain. Close lymph nodes can become in Redjeid, swollen and painful. Soon a crust is formed on the lesion, and it falls into a few weeks. The complete recovery may take longer [21]. The cutaneous ventrax is typically caused when the spores of B. anthracis enter through cuts on the skin. This form is most commonly when human beings manipulate animals or infected animal products. [22] Injection In December 2009, there was an outbreak of an ventrax among the consumers of heroine by injection in the areas of Glasgow and Stirling, in Scotland, which caused 14 deaths [23]. It is believed that the source of the Intrax was the dilution of heroine with bone flour in Afghanistan [24]. The injected ventrax may have symptoms similar to the cutaneous ventrax, and it can also cause a deep infection in the muscle and spread more fast. [25] Lungs The Inhalation Intrax is usually developed within a week after the exhibition, but it can take up to 2 months. During the first days of illness, most people have a fever, chill and fatigue. These symptoms can be accompanied by coughing, difficulty breathing, painful pain and nausea or vomiting, which causes caruncuket by inhalation to distinguish from flu and extra-hospital pneumonia. This is often described as the prodromic period. [26] During the next day, the lack of breath, cough and chest pain becomes more common, and complaints that do not involve chest as nausea, vomiting, altered mental state, sweats and headache are developed in A third or more of people. Symptoms of the upper respiratory tract occur only in a quarter of people, and muscle aches are rare. The altered mental state or lack of breath generally leads people to medical attention and marks the fulminating phase of the necessary.) It first infects the lymph nodes in the chest, rather than the lungs themselves, a condition © called hemorrhagic mediastinitis, causing the bloody lupus to accumulate in the thoracic cavity, thus causing shortness of breath. The second (neumonÅa) (neumonÅa) Noittic [ylipitum airetcab eht erehw, scitahpmyl eht ot noitalucric eht yb daerps neht dna seussit eht otni yrtne fo etis eht ta etanimreg serops eHT .skeow ro syad wef a nihtiw tsoh eht lik ylaicpyt dna namuh ro lamina eht edisni gnylplitum nigeb airetcab eht, dnuow nepo na ni decalp ro detsegni ec©O .nevorp neeh ton sah siht lghuoh, seussit eht otni serops lairetcab eht fo yrtne gnittimrep, tcart IG eht nihtiw sduow esuac ot dezisehtopyh neeb sah noitategev eht; noitategev ykips ro, Tattirri, hquor gnitae nehv yllaicepse, gnizarg elihw detcefni netfo era serovibreH .etats siht ni sedaced rof evivrus nac dna, lios eht ni mrof erops ni ster yllamron muiretcab ehT] 92 [.esuum a otni meht tup dna, airetcab eht detalosi, woc detcefni na morf elpmas doolb a koot eh NEHW 6781 NI HC©K Trebor YB EsaesID ESUAC OT NWOHS SAW T]] 2 [.ezis Ni Må¼åå¼ Å, 9 YB 1 Tuoba MuiretCab Ciboreana Evitatlucaf, Evitisop-Marg, Depahs-Dor a Si Sicarhnta Sullicab Sicarhnta Sullicab: Elictra Niam Esaesid Xarhnta eht fo esuac eht, sicarhnta sullicaB muiretcab eht fo niats marG a fo hpargorcimotohP airetcab esuac] dedeen noitatic [.snixot ekam ot gnunuitnoc elihw, ydob eht tuohguorht dna maertsdoolb eht ot sdaerps ti, metsys lanitsetniortsag eht sedavni muiretcab eht refA .taorht dna htuum eht ni dna sentisetni eht ni dnuof neeb evah snoiseL .rucco nac doolb fo gnitimov lanoisacc©] 82 [.etiteppa fo ssl dna, tcart lanitsetni eht fo noitammafni etuaca, sniap lanimodba, doolb htiw yllaitnetop, aehrraid yb deziretcaraha si dna taem detcefni-xarhnta gnimusnoc yb desuac netfo tsom si noitcefni) Ig (lanitsetniortsagG lanitsetniortsag] 72 [.sesac lataf ni sruoh 84 nihtiw htaed dipar dna, Cchohs, htaerb fo ssentrohs emerixe, revef hgih edulcni smotpmYs .egats tsrif eht refA syad ro sruoh mihw yinedudus poleved egats dnoces eht fo smotpmys .sgnul EHT ©T SEDON HPMYL EHT MORF SDAERPS NOITCOPNI EHT NEHW Srucco The production of two powerful exotoxins and lethal toxin by the bacteria causes death. Veterinarians can often say a possible death induced by the idler by its sudden appearance, and by dark and non-coagulant blood that refuses from body holes. Most of the ventrax bacteria within the body after death are overcome and destroyed by anaerobic bacteria in minutes at post mortem. However, the ventrax vegetative bacteria that escape from the body through the blood that ruffing or through the opening of the canal can form resistant spores. These vegetative bacteria are not contagious.]30[A spore is formed by vegetative bacteria. The triggers of the spore formation are not known, although the tension of oxygen and the lack of nutrients can play a role. Once formed, these spores are very difficult to eradicate. [Mecouragement required] Infection of herbivores (and occasionally human) by the inhalation route usually begins with inhaled spores that are transported through the respiratory trails towards the tiny bags air (alv olos) in the lungs. Spores are then collected by carrine cells (macrophages) in the lungs and are transported through small vessels (lymphatics) to lymph nodes in the central cavity of the chest (mediastinum). The damage caused by the spores of the idle and the bacilli in the central cavity of the chest can cause pain in the chest and difficulty breathing. Once in the lymph nodes, spores germinate in active bacilli that multiply and finally burst the macrophages, releasing many more bacilli in the bloodstream to be transferred to the whole body. Once in the bloodstream, these bacilli release three proteins called lethal factor, factor of and protective antigen. All three are not toxic © themselves, but their combination © not extremely lethal to humans.[31] The protective antagonist combines with these other two factors to form a lethal toxin and an edema toxin, respectively. These toxins are the main agents of tissue tissue Bleeding, and death of the huÅ © sped. If antibiotics are administered too late, even if antibiotics eradicate bacteria, some fumes still die of toxemia because the toxins produced by bacilli remain in their systems at lethal dose levels. [Mecouragement required] Bacillus Anthracis Electronc sweep micrograph enhanced with color shows the spleen tissue of a monkey with inhalation idle; Bacils in the form of a rod (yellow) and a gram-positive caruncuket bacteria (pushed rods) erythrocyte in the cerebrospinal liquid: if present, a gram negative bacterial species would appear pink. (The other cells are white globules). Exposure The spores of the idyr are capable of surviving in hard conditions during decades or even centuries. [32] These spores can be found on all continents, including antartic. [33] It is known that the disturbed severity of the infected animals cause the infection after 70 years [34]. Historically, the inhalation idler was called disease of wool classifiers because it was an occupational risk for people who classified wool. [35] Today, this form of infection is extremely rare in advanced nations, since there are almost no infected animals left. [Appointment required] Occupational exposure to infected animals or their products (such as skin, wool and meat) is the usual way of Exposure for humans. Workers who are exposed to animals and dead animal products run the greatest risk, especially in countries where the idi is more common. The ventrax in the cattle grazing to open field where they are mixed with wild animals still occasionally occasionally in the United States and elsewhere. [Required quote] Many workers dealing with wool and leathers of are routinely exposed to low levels of ntrax spores, but the greater number of exposure levels © not sufficient to develop ntrax infections. It is reported that a lethal © infection is the result of inhalation © about 10,000 Å 20,000 spores, although this dose varies among the hosts There is little documented evidence available to verify the exact or average number of spores needed for © infection. Infection mode © Inhaling, mediastunic, and widening ntrax The ntrax can enter the human body through the intestines (ingestion© n), the lungs (inhalation© n), or the skin (cutÅ nea), and causes different unique symptoms depending on © place of entry. In general, an infected human is quarantined. However, anthrax does not usually spread from an infected human to an uninfected human[37]. However, if the disease is fatal to the person's body, its mass of antrax bacilli becomes a potential source of infection © others and special precautions must be taken to prevent further contamination© Inhalation anthrax, if not treated until obvious symptoms occur, is usually fatal[37]. Anthrax can be contracted in laboratory accidents or by handling infected animals, their wool or their bodies.[38] It has also been used in biological warfare agents and by terrorists © intentionally infect, as exemplified by the 2001 Anthrax attacks.[39] Mechanism The lethality of Anthrax disease is due to the two main virulence factors of the bacterium: the poly-D-glutÅ ic acid capsule, which protects the bacteria from phagocytosis by the neutro© filos, and the tripartite protein toxin, called the ntrax toxin. Anthrax components: Protective antigen (PA), edema factor (EF) and lethal factor (LF).[40] PA mãÅo LF produces lethal toxin, and PA mãÅo s EF produces edema toxin. These toxins cause death and inflammation © the tissues (edema), respectively. To enter the cells, the edema and lethal factors use another proteÅna produced by B. anthracis called protective antigen, which binds to two surface receptors in the Lawn. ©, a cell protease divides the AP into two fragments: PA20 and PA63. PA20 dissociates in the extracellular medium, without any additional role in the © cycle. PA63 is oligomerized with six six Fragments PA63 that form a hepty-american ring structure called a PREPORE. Once in this way, the complex can unite competitively up to three EFS or LF, forming a resistant complex. [31] The receptor-mediated endocytosis occurs below, providing comprehensive comprehensive access to the interior of the HuÅ © sped cell. The acidified environment within the endosoma triggers the heptamer to release the LF and / or EF in the cytosol. [41] It is unknown to exactly the complex results in the death of the cell. The edema factor is an adenylate dependent on calmodulin. ADENILATE CYCLASA Catalyzes the ATP conversion in cyclic amplifier (camp) and pyrophosphate. The complexation of adenylate cyclase with calmodulin eliminates the calmodulin of stimulating the signs activated by calcium, inhibiting the immune response. [31] To be specified, LF inactive neutrophils (a type of phagocytic cell) by the process that has just been described so that they can not phagocystic bacteria. Throughout history, it was presumed that the lethal factor caused the macrophages to make TNF-alpha and interleukin 1, beta (IL1B). TNF-Alpha is a cytokine whose main role is to regulate immune cells, as well as to induce inflammation and apoptosis or programmed cell death. Interleukin 1, Beta is another cytokine that also regulates inflammation and apoptosis. The overproduction of TNF-ALPHA and LL1B finally leads to the SÅ © Pictic Shock and Death. However, the recent evidence indicates that it is also aimed at the endothelial cells that line the serious cavities, such as the pericamnet cavity, the pleural cavity and the peritoneal cavity, the lymph vessels and the blood vessels, which causes vascular leaks of fluid and cells, and ultimate instance, hypovolegical shock and selbthic shock. Diagnosis© Possible edema and necrosis in a case of anulment © injection© Several techniques can be used for the direct identification © B. antracis in classical material. First, the specimens may be stained. Bacillus spp. They are quite large in size (3 to 4 ¼m long), they can grow in long chains, and Spot Gram-positive. To confirm that the body is B. anthracis, rapid diagnostic techniques can be used, such as-based tests Å Å

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