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1: Viral Evasion Mechanisms of the Host Response | Frontiers Research Topic

3. The host-invader interplay: proceedings of the 3rd International Symposium on the Biochemistry of Parasites and Host-Parasite Relationships, Beerse, Belgium, 30 June-3 July, 3.

Publications to most recent first Expression of a bacterial gene in a trypanosomatid protozoan. Evaluation of evolutionary divergence in the genus Naegleria by analysis of ribosomal DNA plasmid restrction patterns. Mol Biochem Parasitol The procyclic acidic repetitive proteins of Trypanosoma brucei: J Biol Chem Variation of tandem repeats in the developmentally regulated procyclic acidic repetitive proteins of Trypanosoma brucei. Mol Cell Biol 9: Biosynthesis of the glycolipid membrane anchor of Trypanosoma brucei variant surface glycoproteins: Biochem Soc Trans Rapid and simple amplification of a specific RNA sequence by the polymerase chain reaction. Small-subunit ribosomal RNA sequence from Naegleria gruberi supports the polyphyletic origin of amoebas. Mol Biol Evol 5: Circular ribosomal RNA genes are a general feature of schizopyrenid amoebae. Discontinuous transcription in Leptomonas seymouri: Nucleic Acids Res Characterization of RNA transcripts from the alpha tubulin gene cluster of Leptomonas seymouri. Amino acid changes linked to pyrimethamine resistance in the dihydrofolate reductase-thymidylate synthase gene of Plasmodium falciparum. Intracellular transport of a variant surface glycoprotein in Trypanosoma brucei. J Cell Biol Detection of a possible trans-splicing intermediate in Trypanosoma brucei. Cloning and transcriptional analysis of a variant surface glycoprotein gene expression site in Trypanosoma brucei. Candidate glycophospholipid precursor for the glycophosphatidylinositol membrane anchor of Trypanosoma brucei variant surface glycoproteins. Glycolipid precursor of Trypanosoma brucei variant surface glycoproteins: Polymorphism in the procyclic acidic repetitive protein gene family of Trypanosoma brucei. Mol Cell Biol 8: Mol Cell Biol 7: Biochemical changes associated with alpha-difluoromethylornithine uptake and resistance in Trypanosoma brucei. Eukaryotic protein modification and membrane attachment via phosphatidylinositol. Direct analysis of the mini-exon donor RNA of Trypanosoma brucei: Effect of 3-aminobenzamide on the frequency of antigen switching in Trypanosoma brucei in vitro. Glycosyl-sn-1,2,-dimyristylphosphatidylinositol is the membrane anchor for Trypanosoma equiperdum and T. Nannomonas congolense variant surface glycoproteins. Developmental regulation of a novel repetitive protein of Trypanosoma brucei. Identification of proteins encoded by variant surface glycoprotein expression site-associated genes in Trypanosoma brucei. Biosynthesis of Trypanosoma brucei variant surface glycoproteins: N-glycosylation and addition of a phosphatidylinositol membrane anchor. Purification and characterization of a novel glycan-phosphatidylinositol-specific phospholipase C from Trypanosoma brucei. Identification of the parasite transferrin receptor of Plasmodium falciparum-infected erythrocytes and its acylation via sn-1,2-diacylglycerol. Structure and organization of the histidine-rich protein gene of Plasmodium lophurae. Primary structure of the histidine-rich protein of Plasmodium lophurae. Analysis of antigen switching rates in Trypanosoma brucei. Variations in the organization of repetitive DNA sequences in the genomes of Plasmodium falciparum clones. Coordinate transcription of variant surface glycoprotein genes and an expression site associated gene family in Trypanosoma brucei. Cysteine eliminates the feeder cell requirement for cultivation of Trypanosoma brucei bloodstream forms in vitro. J Exp Med Glycosyl-sn-1,2-dimyristylphosphatidylinositol is covalently linked to Trypanosoma brucei variant surface glycoprotein. Trypanosoma brucei variant surface glycoprotein has a sn-1,2-dimyristyl glycerol membrane anchor at its COOH-terminus. Acylation of a Plasmodium falciparum merozoite surface antigen via sn-1,2-diacyl glycerol. Release and purification of Trypanosoma brucei variant surface glycoprotein. J Cell Biochem Histidine-rich protein genes and their transcripts in Plasmodium falciparum and P. The complete amino acid sequence of a variant surface glycoprotein VSG from Trypanosoma brucei. J Mol Biol Complete nucleotide sequence of cDNA coding for a variant surface glycoprotein of Trypanosoma brucei. The molecular basis for trypanosome antigenic variation. Molecular cloning of the foot and mouth disease virus genome and nucleotide sequences within the structural protein genes. Variant surface glycoproteins of Trypanosoma brucei are synthesised with cleavable hydrophobic sequences at the carboxy and amino termini.

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Nucl Acids Res 9: DNA rearrangements involving the genes for variant antigens in trypanosomes. Antigenic variation of parasites. Glycopeptides from variant surface glycoproteins of Trypanosoma brucei: C-terminal location of antigenically crossreacting carbohydrate moieties. Mol Biochem Parasitol 2: Production of a relapsing infection in rodents. The genes for variant antigens in trypanosomes. An introduction to antigenic variation in trypanosomes. Am J Trop Med Hyg Antigenic variation in clones of animal-infective Trypanosoma brucei derived and maintained in vitro. The genes for variable surface glycoproteins of Trypanosoma brucei. Protective monoclonal antibodies recognising stage-specific merozoite antigens of a rodent malaria parasite. Monoclonal antibodies against the rodent malaria parasite, Plasmodium yoelii. In vitro cloning of animal-infective bloodstream forms of Trypanosoma brucei. Novel expression-linked copies of the genes for variant surface antigens in trypanosomes. Trypanosome variant surface glycoprotein:

2: Iron and Parasites

 This article has been cited by other articles in PMC. In this special issue, we analyze the importance of iron in the host-parasite interplay. Iron is vital for growth of nearly all living organisms, from prokaryotes to humans. Iron plays an important role in several cellular processes, such as respiration, photosynthesis, oxygen transport, and DNA synthesis. Iron is essential but it is not easily bioavailable; ferric iron solubility is low at physiological pH whereas ferrous iron, in aerobic environments, is highly toxic. Therefore, iron is normally bound to proteins and the whole body and cellular iron concentrations have to be regulated in all organisms. Some iron-containing and iron-binding proteins are intracellular such as the oxygen-carrier hemoglobin, the iron-storing protein ferritin, and numerous enzymes. Others are extracellular, mainly transferrin Tf and lactoferrin Lf. Free radicals are deleterious to most macromolecules. Tf and Lf maintain the free iron concentration too low to sustain the parasites growth. Tf is the iron transporter that allows cellular iron uptake; it is mainly found in serum and lymph. Therefore, during infection, there is a constant battle between the host and the invader for iron, in which the invader attempts to have access to host iron and the host arranges complex iron-withholding mechanisms to frustrate the iron stealing. Virtually, all iron-containing proteins in eukaryotes can be used as iron sources by iron-seeking parasites; for that, several elaborate strategies have been developed by parasites to obtain host iron. Thus, capture and uptake of host iron by parasites are considered as virulence determinants. Little information regarding iron acquisition in free-living amoebae has been reported. During the invasion, the microorganism interacts with different tissues such as olfactory neuroepithelium and olfactory bulbs that contain iron-binding proteins. The results show that this protozoan has several cysteine-secreted proteases that cleave iron-binding proteins. Using this strategy, N. Interestingly, although both amoeba variants are able to use B-holo-Lf as an iron source and endocytosed this glycoprotein through clathrin-coated vesicles, the acquisition of iron, binding parameters, and number of protein-binding sites per amoeba are different. In addition, the virulent amoebae also endocytosed B-holo-Lf through a cholesterol-dependent mechanism; thus the B-holo-Lf endocytosis is more efficient in virulent amoebae. They explore the current knowledge about the process that occurs during infection by intracellular pathogens, where the iron is required by both the host cell and the pathogen that inhabits it. Intracellular microorganisms are destroyed by the host tissues through processes that usually involve phagocytosis and lysosomal disruption. However, some intracellular pathogens are capable of avoiding destruction by growing inside macrophages and other cells. Additionally, the implications of these mechanisms for iron acquisition in the host-pathogen relationship are discussed. African trypanosomosis is caused by the parasitic protozoan Trypanosoma brucei. This is a chronic and debilitating disease suffered mainly by people of developing countries. In particular, it examines the effect of iron in gene expression regulation and function of cathepsin L-like and asparaginyl endopeptidase-like CPs as virulence factors. Aspects regarding CPs genomic organization are addressed to offer possible explanations to the fact that only few members of this large gene family are expressed at the RNA and protein levels. Also offers possible ways used to control these particular proteolytic activities. Moreover, all known iron regulatory mechanisms of CPs at transcriptional, posttranscriptional, and posttranslational levels along with new insights into the possible epigenetic and miRNA processes in T. The signal transduction initiated upon ligand binding at the parasite plasma membrane with the process in mammalian cells is compared, based on the large amount of information on the Tf endocytosis. Several signaling pathways participate in Tf trafficking, such as the insertion of membrane vesicles, and the signaling pathways mediated by the inositol-1,4,5-triphosphate and diacylglycerol, MAPK, or growth factors. Some components of these pathways also found in parasites are included, as well as the identification of signaling proteins, useful in the study of essential factors for the parasitic life and as potential targets for the

development of chemotherapeutic approaches. We hope that researchers enjoy the reading of this special issue related to parasites and one of the most important chemical elements, iron. Undoubtedly, the acquisition of host iron by a pathogen is a crucial step during the development of infection and is determinant in its outcome.

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Variation of the surface glycoprotein VSG of T. The pathologic consequences of trypanosomiasis caused by T. The severity of these disease symptoms varies among hosts, which, by this definition, are referred to as more trypano-resistant or more trypano-susceptible. Recent studies provide reasonable grounds to doubt the above concept. In both strains of mice plasma cells arose with the same kinetics, reached similar numbers in all lymphoid organs examined and synthesized and secreted similar amounts of antibodies of the same Ig classes, including antibodies specific for exposed VSG-epitopes on the infecting organisms. Hence it was inferred that there was less antibody bound per trypanosome, leading to failure of the susceptible mice to clear parasites from the bloodstream. VSG-specific antibody absorbed by trypanosomes is endocytosed and degraded D. Failure to clear trypanosomes from the bloodstream leads to a prolonged parasitaemic wave, rapid destruction of lymphoid organ architecture and concomitant loss of ability to mount efficient humoral immune responses. Accessory studies showed that the higher levels of parasitaemia reached in infected susceptible, as opposed to resistant, mice correlated with slower parasite differentiation to committed non-dividing trypanosomes in the bloodstream,5 an event that is probably controlled by antibody-independent host responses. The rate of parasite differentiation to committed non-dividing T. Axenic culture systems7 were exploited to identify host-derived macromolecules required for the multiplication of T. Two different serodemes of T. No major biochemical adaptations were thus required to transit between the two environments and hence the in vivo and in vitro T. High-density lipoproteins HDL; density 1. It was shown that both culture-adapted T. Black, unpublished took up lipoprotein-lipids without talking up or degrading apolipoproteins. The uptake process did not discriminate between HDL and LDL, was independent of exogenous divalent ions and was not influenced by exogenous weak bases 20 mM ammonium chloride, 20 m M chloroquine8. The uptake mechanism was thus utterly different from receptor-mediated endocytosis of LDL as practiced by mammalian cells. X63 obtained the lipids from LDL only and normal mouse spleen cells did not take up the lipids. This observation suggests that lipoprotein-derived phospholipids and cholesterol esters might enter T. In contrast, bile acids and conjugated and unconjugated bile salts inhibited the uptake by culture-adapted and normal bloodstream T. Different cholesterol conversion products had differing efficiencies to inhibit T. Two possibilities present themselves. The cholesterol conversion products might prevent the uptake by T. Alternatively, the cholesterol conversion products might compete with an interaction between lipoprotein-associated cholesterol ester and a T. In the absence of the bile acid, the T. Uptake of the cholesterol ether was inhibited by the bile acid Vandeweerd and Black, unpublished. Because cholesterol esterases are unable to cleave the ether bond11 and because cholesterol ether cannot diffuse through cell membranes, we conclude that T. Alternatively, cholesterol esters and phospholipids might be sequestered in chylomicrons and VLDL in such a way that they cannot interact with T. As a preliminary step to distinguishing between these possibilities, it is of some importance to define the maximum size of molecules that can enter the T. In contrast, uptake by T. We therefore consider it likely that uptake of the H cholesteryl linoleate is mediated by binding to a protein, whereas uptake of H dipalmitoyl phosphatidyl choline is not. It is tempting to suggest that release of the lipoprotein-associated phospholipid occurs in the T. The uptake by T. Concentrations of bile acids 5 to 15 m M , and conjugated and unconjugated bile salts 50 to m M , which inhibit uptake by T. Although the inclusion of LPD-FBS in the incubation mixture reduces the short-term toxicity of cholesterol conversion products, it does not completely abrogate their effects. Concentrations of bile acids m M can be chosen that prevent multiplication of T. Because conjugated bile salts and bile acids are found in normal plasma, it is an attractive idea that these molecules might have a role to play in protection against African trypanosomes. Clearly, quantitative data are required on the bile acid, and conjugated and unconjugated bile salt concentrations in the plasma and interstitial fluids of normal and infected trypano-susceptible and trypano-resistant hosts. Equally clearly, quantitative data are required on the

sensitivity of different trypanosome clones, serodemes and species to the toxic effects of cholesterol conversion products in the presence or absence of blood. In The Host-Invader Interplay. Van den Bossche, Ed.:

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The Host-Invader Interplay: 3rd International Symposium on the Biochemistry of Parasites and Host-Parasite Relationships In vitro cloning of animal-infective bloodstream forms of Trypanosoma brucei.

Correspondence and Footnotes Abstract Several lines of evidence have shown that Trypanosoma cruzi interacts with host extracellular matrix ECM components producing breakdown products that play an important role in parasite mobilization and infectivity. Increased expression of ECM components has been described in the cardiac tissue of chronic chagasic patients and diverse target tissues including heart, thymus, central nervous system and skeletal muscle of experimentally T. ECM components may adsorb parasite antigens and cytokines that could contribute to the establishment and perpetuation of inflammation. Since persistent "danger signals" triggered by the parasite and its antigens are required for the establishment of inflammation and ECM alterations, therapeutic interventions that control parasitism and selectively modulate cell migration improve ECM abnormalities, paving the way for the development of new therapeutic strategies improving the prognosis of T. Trypanosoma cruzi, Extracellular matrix, Cell adhesion, Cell migration, Adhesion molecules, Chemokines Introduction As a common consequence of a combinatorial interplay between invader and host, inflammatory processes are established leading to invader control but frequently contributing to the establishment of chronic inflammatory disease. Inflammatory responses to tissue infection require cell migration, a widespread process that comprises cell-cell adhesion and cell-extracellular matrix ECM interaction and is orchestrated by cell adhesion molecules and integrin receptors, ECM components, matrix metalloproteinases and chemoattractant molecules. Migration is also a prerequisite for infectious agent spreading. Direct or bridge molecule-mediated interactions occur between invaders and host ECM and cells, being a crucial event for successful infection. Trypanosoma cruzi-host cell interaction The parasite talks The protozoan T. The understanding of the molecular basis of the host cell invasion process by T. Several lines of evidence demonstrate that the ECM glycoprotein fibronectin FN promotes adhesion and uptake of trypomastigotes by macrophages and fibroblasts 1,2 and amastigotes by murine or human macrophages 3. Subsequent studies showed that T. It is commonly believed that as a prerequisite for host cell invasion, T. Recently, it has been reported that the binding of T. Also, a new class of T. In vivo, cruzipain also binds to the FN network present in the heart tissue of T. Sites that are cryptic or hidden in the native ECM molecules may become activated or exposed upon disassembly or degradation of the ECM component structure by proteolytic enzymes that are released by interstitial parasites or, considering the whole event, secreted by tissue-invading leukocytes. As a biological consequence, the ECM breakdown products may facilitate invasion of target cells by the parasite. In fact, FN cleavage fragments were shown to provide a growth factor-like activity for the differentiation of trypomastigotes to amastigotes, a more efficient invasive form In addition, in view of the ongoing T. The inflamed myocardium is exposed to parasite antigens either released into the cardiac interstitial spaces by extracellular parasites or from killed organisms. More recently, we have shown that proteins mainly ranging from 85 to kDa released by T. A cysteine proteinase, cruzipain, but not trans-sialidase, was detected among the cell-bound antigens. As a consequence of the adsorption of released antigens the sensitized cells became targets for anti-T. Thus, the in vivo released T. Recent studies have shown that an kDa serine protease located inside a vesicular compartment close to the flagellar pocket was found to hydrolyze FN and, more importantly, to be a potential target for a potent selective inhibitor that blocks T. Also, a new member of the T. Together with parasite-binding sites for host thrombospondin, heparin sulfate and FN, this LN-binding site has been proposed to be a potential target for inhibition of invasion of host cells by T. Taken together, these data led to the concept that specific interactions between T. Moreover, these findings provide evidence that ECM-binding sites present on the T. Trypanosoma cruzi antigen cruzipain green is detected associated with fibronectin fibers red in the myocardium of chronically days post-infection infected mice. The animals were infected with trypomastigotes of the Colombian strain of T. The arrows indicate the superposition of the labeling for fibronectin and cruzipain yellow. The pathogenesis of tissue remodeling, particularly that which causes the unique apical aneurysm found in chagasic patients, remains

unclear. Fibrosis is one of the most important features of the chronic chagasic cardiomyopathy with multifocal scarring frequently observed in the absence of significant inflammation or parasitized myocytes More recently, it was shown that, compared with dilated cardiomyopathy, chronic chagasic myocarditis presents a distinctive pattern of ECM abnormalities However, focal connective tissue damage with multiple areas devoid of ECM components was observed in inflamed atria of early acutely infected mice It is possible that ECM degradation can precede or concurrently occur with enhanced fibrosis, and account for remodeling of cardiac tissue. In fact, alterations of the ECM may contribute to ventricular dysfunction and geometric shape changes observed in human and experimental T. These include loss of ECM components through degradation or mechanical destruction producing a thin-walled, dilated, and poorly contracting ventricle, even in the absence of significant myocyte injury Abnormal expression of ECM components seems to be a widespread event during T. A progressive and intense increase in the intralobular network containing FN and type IV collagen was observed in the atrophic thymus of T. In skeletal muscle diffuse inflammation composed mainly of mononuclear cells is initially detected in the early acute phase. It was paralleled by ECM abnormalities that are observed in areas with inflammatory infiltrates and areas not affected by myositis, and related or not to the presence of parasite pseudocysts and antigens. In this experimental model, CD8-mediated myocarditis is established during the early acute infection Figure 3 and persists during the chronic phase Also, CD8-mediated meningoencephalitis restricted to the acute phase is observed in this animal model Moreover, these data support the idea that during T. Laminin expression was significantly increased in the cardiac tissue of T. Similarly, in the inflammation of the central nervous system of acutely T. Our findings do not rule out this possibility; however, it is possible that after transmigration through the endothelial layer, inflammatory cells may lose or down-modulate the expression of VLA-6 Interestingly, we have shown that the FN mesh present in the inflamed heart of chronically T. Moreover, mononuclear cells expressing VLA-4 and CD44 were detected immunohistochemically in the myocardium of chronic chagasic patients 31 , suggesting that these inflammatory cells are activated and able to interact with ECM components. The role of FN and its receptors in the physiopathogenesis of the chronic T. In fact, it has been reported that T. The distribution of variant forms of FN in anatomic sites in vivo remains unknown but it has been suggested that VLAdependent co-stimulation of T cells may be restricted to certain critical anatomic sites in vivo It is reasonable to propose that interactions of T cells with the fine FN filamentous network present in the inflamed myocardium via VLA-4 molecules may provide an anchor for invading cells and not only function as a pathway in the T cell migration processes but may also influence antigen-specific T cell recognition, proliferation, survival and effector activity In addition, FN and other ECM components may also act as chemoattractants for additional invading cells or provide a substrate for the anchoring and stabilization of chemoattractant molecules 35,36 , contributing to the perpetuation of inflammation in this tissue. Further as discussed above, T. Indeed, it has been shown that damaged ECM activates macrophages to secrete IL and chemokines 37,38 , cytokines present in the inflamed heart of chronic chagasic patients 24,25 , and in acute and chronic experimentally T. These cytokines play a central role by controlling tissue parasitism during T. With respect to putative sources of ECM components in T. However, the possibility that myocytes and other cell types present in these tissues could also contribute to ECM production cannot be ruled out. In fact, our results show an increased expression of FN and LN by infected and noninfected neurons and glial cells astrocytes and microglia 28 of acutely T. Also, in vitro studies have shown that several other cell types when infected with T. In this respect, in addition to a remarkable increase in ECM production by T. Also, this modified heparan sulfate proteoglycan binds more efficiently to acidic fibroblast growth factor. Moreover, the ECM produced by infected endothelial cells can direct the synthetic patterns of noninfected cells in a manner uniquely observed in infected endothelial cells, suggesting a plausible pathway by which infection of only a few cells can ultimately result in the coordinate responses of neighboring noninfected cells Since that gene expression is regulated by the surrounding ECM 38 , these results could partially explain the drastic alterations of ECM expression as well as other products observed during chronic chagasic myocardiopathy in the presence of only few parasites. In view of the data described above, the ECM produced by T. Further, our findings showing that adsorption of T. It should also be considered that soluble factors such as cytokines, chemokines and growth factors produced

by T. In this context, a fibroblast-stimulating factor was identified in cell-free conditioned media from T. More recently it was shown that T. Thus, it is possible that interactions of cytokines and chemokines with FN and other ECM components or enzymatically modified ECM may affect the distribution and pleiotropic activities of these molecules in inflamed tissues, where the cytokines may act as proadhesive molecules, strengthening cell binding to the ECM, thereby promoting cell activation 36, Hence, presentation of cytokines and chemokines by ECM molecules in the inflamed tissue of T. It is also possible that products of the inflammatory cells such as IFN-g, TNF-a and chemokines detected in the heart tissue of chagasic patients 24,25 and heart and central nervous system of animals experimentally infected with T. Furthermore, systemic hormones such as glucocorticoids 53 and cytokines such as IFN-g 54 and TNF-a 55 detected in elevated levels in the serum of T. Further, local and systemically produced proinflammatory cytokines may modulate the expression of adhesion molecules such as ICAM-1 and VCAM-1 on the endothelial cells 56,57 and hence promote cellular infiltration of lymphocytes and other effector cells into the inflamed tissues. More recently, we have shown that during experimental chronic chagasic infection there is a remarkable increase in the expression of VCAM-1 on the endothelial cells of heart blood vessels In addition, ICAM-1 expression that was low on endothelial cells of cardiac tissue of noninfected animals was upregulated in T. Using murine models susceptible and resistant to the development of acute T. Moreover, the heart tissue of T. The findings discussed above provide evidence that profound alterations in the expression of ECM, cytokines, and adhesion and chemoattractant molecules are observed in several target tissues as a consequence of T. All of these molecules may play a beneficial role leading to activation, recruitment and migration of cells involved in the control of the parasite but may also contribute to the establishment of adverse consequences such as chronic inflammation and organ dysfunction. Amastigote nest containing hundreds of parasites in the absence of inflammatory infiltrates at 35 days post-infection inset. B, Flow cytometric analysis of mononuclear cells obtained from skeletal muscle of T. Immunohistochemical analysis showed that compared with normal mice C , chronically T. LN is not detected surrounding the inflammatory cells red. Immunohistochemical analysis showed that the increased expression of LN is restricted to the basal lamina of myocytes and basement membrane of blood vessels C. Panel B depicts an immunohistochemical analysis of the cardiac tissue of T. Immunohistochemical analysis showed that compared with normal mice C , acutely 28 days post-infection T. The arrow indicates the presence of an amastigote nest inside a cardiomyocyte. Since the parasite is apparently required for the continuous supply of "danger signals" that result in damage to cardiac tissue 59 , the elimination of the parasite may cause clearance or partial regression of inflammatory lesions in target tissues. It has been shown that specific chemotherapy leads to reversibility of cardiac fibrosis in mice chronically infected with T. Interestingly, the improvement in ECM abnormalities in benznidazole-treated mice correlated with a drastic reduction in inflammation Thus, we may propose that an immunomodulatory treatment that selectively blocks or diminishes the entry of deleterious inflammatory cells into the cardiac tissue of T. Importantly, the treatment of acutely T. Thus, we propose that a therapeutic intervention ultimately leading to modulation of ECM expression could contribute to improving the prognosis of T. Fibronectin enhances macrophage association with invasive forms of Trypanosoma cruzi.

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5: DI-fusion Cloning and characterization of cDNA sequences coding for two

Both HDL and LDL from a number of different species, including cattle, African buffalo, eland, rabbits and rats, were as able as FBS-HDL or FBS-LDL to support T. brucei multiplication. 8 The observations fit well with the published requirement of bloodstream T. brucei for exogenous lipids 9 and suggest the presence of a lipid scavenging mechanism suited to a parasite with a wide host range.

Additional Information In lieu of an abstract, here is a brief excerpt of the content: Indeed, of the six major tropical diseases targeted for action by the World Health Organization, malaria, schistosomiasis, filariasis, trypanosomiasis, leishmaniasis, and leprosy, five are parasitic diseases [I]. Yet, despite decades of intensive effort on the part of researchers, public health planners, and government agencies, control measures are barely effective, and the eradication of these diseases remains a distant dream. The development of vaccines against tropical diseases has been an elusive goal [2], and the resurgence of drug-resistant organisms [3] raises the specter that even our sophisticated arsenal of pharmaceuticals may not be adequate to meet the challenge in the future. Any attempt to manipulate an organism, whether in laboratory research or in clinical practice, must be predicated on a sound knowledge of the experimental animal [4]. In planning approaches to control parasitic diseases, it is particularly useful to frame questions about the The material presented in this paper was developed from lectures given by the author at the Uniformed Services University of the Health Sciences, Bethesda, Maryland. The author is grateful to J. Michelson for their useful suggestions with regard to this topic, and to M. DiBari for editorial assistance. The opinions or assertions contained herein are the private ones of the author and are not to be construed as official or reflecting the views of the U. Department of Agriculture or the Department of Defense. Copyright is not claimed for this article. Such an outlook, connoting as it does the melodramatic images of doctors locked in mortal combat with microbes, is not proposed simply for romantic purposes. Instead, as will be shown, this approach yields a very useful way to analyze parasitic behavior. It is well accepted that animals tend to adapt to their environment over time, and parasites are no exception. For the parasite, however, the situation becomes more complicated because the environment is another living organism, the host. The microenvironment terminology of Dogiel [7] of the parasite includes not only temperature, pH, oxygen concentration, and availability ofnutrients but also the specific responses of the host that are directed toward elimination of the parasite. Adaptation to such a potentially hostile environment has involved the evolution of what can only be described as ingenious methods to evade those responses. There is even mounting evidence that some parasites have encountered the immune response as simply another host resource to be exploited [8]. Many of the mechanisms used by parasites to deal with the host immune system bear a striking resemblance to the tactics used by armies on the battlefield. A Tactical Approach to Infection and Immunity By definition, an infectious organism is an "invader," and, whether the invasion is active or passive, it represents an assault, an act of aggression. The invader seeks lebensraum, a place to live, within the host. In response , the host mobilizes an army of cells from the immune system. One does not have to stretch the imagination too far to picture lymphocytes constructing a hasty defense at the site ofinfection or macrophages mounting a counterattack. Infection stimulates blastogenesis and results in an increase in the number ofcirculating white blood cells, particularly eosinophils [9, 10], which is certainly analogous to mobilizing the reserves or reactivating the draft. Such a battle may have any of several outcomes: The parasite may be entirely repulsed, as happens in natural resistance of rats to Schistosoma mansoni [11] and mice to Hymenolepis diminuta [12] infections, or in the "self-cure" phenomenon of Haemonchus contortus in sheep [13]. If not eliminated, the parasite may be sequestered You are not currently authenticated. View freely available titles:

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7: Trypanosomiasis: Host Susceptibility and Trypanosoma brucei Lipid Uptake

Benzimidazole anthelmintics may act by interfering with the microtubule system in Ascaris www.enganchecubano.comg of benzimidazole anthelmintics, and their inactive metabolites, to A. suum tubulin was demonstrated by the inhibition of

intestinal extracts of the nematode to bind [3 H]colchicine.

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