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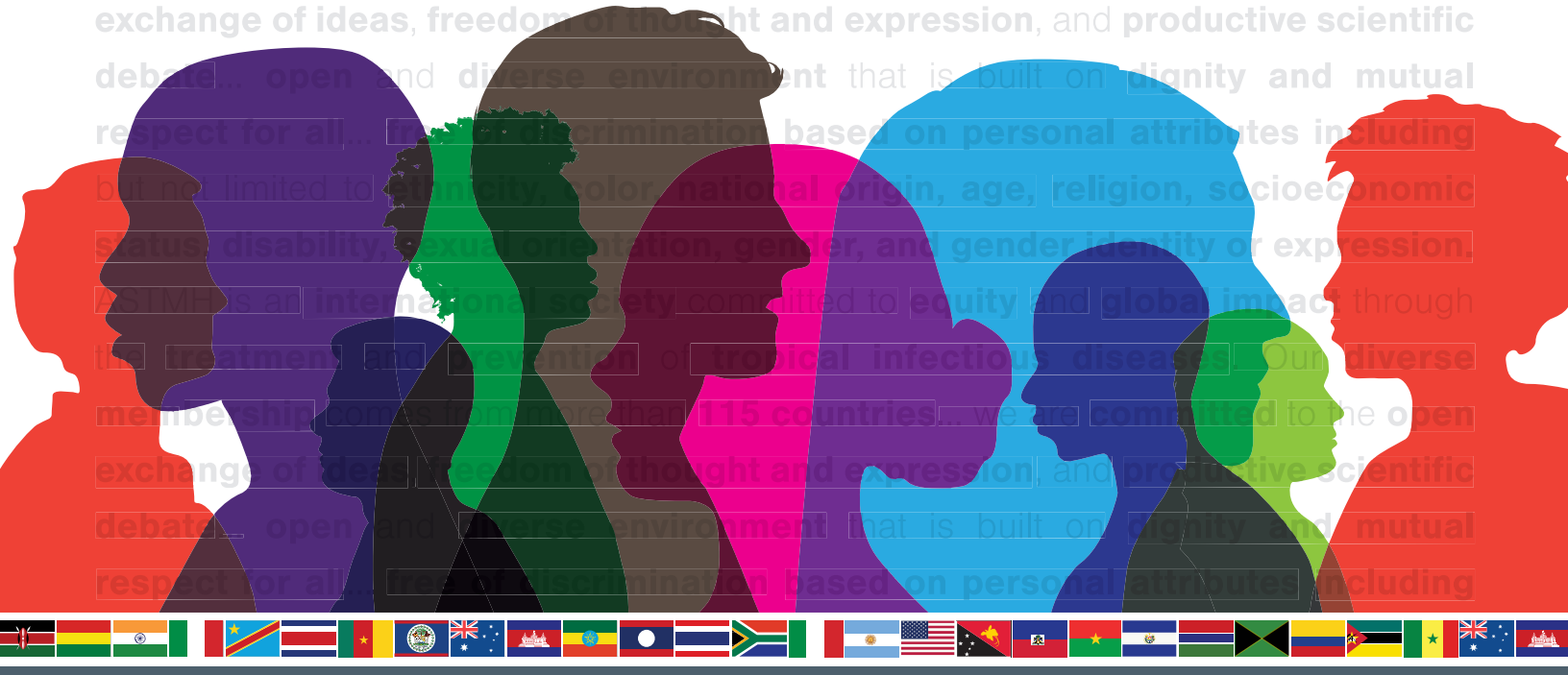
ABSTRACT BOOK



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spiked a fever of 102F, and was admitted for further evaluation and treatment. He was found to have a CD4 of 76 (prior CD4 was 379 four months earlier) with an undetectable HIV viral load. Chest X ray showed bilateral infiltrates, and laboratory testing showed mild transaminase level elevation. Next-generation sequencing (NGS) of cell-free DNA was performed. Within 72 hours, the result came back with detection of Aspergillus fumigatus, Hepatitis B and CMV. Sputum cultures eventually grew Aspergillus fumigatus, and subsequent confirmatory testing revealed elevated serum aspergillus antigen and beta-D-glucan levels. Hepatitis B DNA of >100 million IU/ml and CMV of 256 IU/ml were also detected. Despite previous Hepatitis B vaccination with negative Hepatitis B surface antigen and positive surface antibody titers one year earlier, he was found to have lost his Hepatitis B immunity, and demonstrated positive Hepatitis B surface antigen, core antibody and E antigen. CT scan of the chest revealed multilobar pneumonia with a four-centimeter cavitary lesion of left lower lobe. MRI of brain revealed multiple ring-enhancing lesions likely representing abscesses from Aspergillosis. LP was unremarkable. He subsequently improved with voriconazole and micafungin treatment, and was discharged home. This case illustrated the potential utility of using NGS of cell-free DNA to rapidly diagnose multiple co-infections in severely immunocompromised HIV infected patients without the need for invasive diagnostic procedures.

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A SYSTEMATIC REVIEW OF VIRULENCE FACTORS IN THE *LEISHMANIA* GENUS

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Parasite-determined factors play a complementary role in the pathogenesis of leishmaniasis, a disease caused by protozoans of the genus *Leishmania* with diverse and species-specific clinical manifestations. Virulence factors (VFs), or pathogen moieties facilitating disease, can potentiate host cell damage by Leishmania species via increased expression, host cell invasion, stress tolerance, and modulation of the host immune system. Due to large eukaryotic genomes in Leishmania species, there is a wide array of VFs which contribute to different aspects of pathogenesis. Here we conduct a comprehensive, systematized review of the literature around VFs in *Leishmania* spp. and construct a complete picture of parasite-determined contributors to the pathogenesis of various clinical forms of leishmaniasis. PubMed (NCBI), MEDLINE (OVID), EMBASE (OVID), Web of Science, and LILACS (VHL) were searched from inception to July 2018 using combinations of the search terms "virulence factor*", "*Leishmania*", and "Leishmaniasis*", while accounting for unique database syntax. Iterative inclusion and exclusion of search terms was employed to maximize relevant article extraction. For the systematic review, we will include primarily molecular and mechanistic pathogenesis studies in various model systems, observational studies, review studies, cohort studies, as well as clinical trials. Of 2620 articles remaining after title and abstract screening, some major VFs identified in the Leishmania genus are: heat shock proteins (HSP23, HSP70), cysteine peptidases (CPB), mannose phosphate isomerases (MPI), metalloproteases (GP63), and elongation factors (EF1-alpha), among many others. Data will be grouped and summarized by species, geographic region of endemicity, and VFs. This systematic compilation of mechanistic VF data will add to the large body of work in molecular pathogenesis of kinetoplastids and enhance our understanding of species and regional variations in Leishmania pathogenesis.

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NEW *LEISHMANIA* SPECIES AND ITS POTENTIAL NEW VECTOR, RESPONSIBLE FOR CUTANEOUS LEISHMANIASIS IN SOUTHEASTERN GHANA

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An outbreak of human cutaneous leishmaniasis (CL) was observed in Ho District in the Volta Region of South-eastern Ghana in 1999, which was we proved to be leishmaniasis and isolated the causative protozoan from patients with skin ulcers. The study aimed at identifying the *Leishmania* species responsible for CL and their potential vectors. Aspirates from patients with cutaneous ulcers were cultured *in vitro* in *Leishmania* growth medium (Sloppy Evan's and M199). Isolates of the "yet-to-be-named" *Leishmania* species were amplified, and genomic DNA was extracted. Sequence analysis of RPL23, ITS1, RNAPolII genes were performed with reference genomes. The Ghanaian *Leishmania* isolate clade closely with the pathogens *L. orientalis*, *L. martiniguensis*, *L. marcopodum*, belong to *Leishmania (Mundinia) enriettii* complex on the evolutionary tree. The phylogenetic analysis of Ghanaian isolates and other available sequences revealed it's a newly species. To incriminate the vector transmitting the Ghanaian isolates, two potential vectors, *Lutzomyia longipalpis* (sand fly) and *Culicoides sonorensis* (biting midge) were infected with Ghana *Leishmania* isolate and maintained for more than 10 days. Representative vectors were dissected daily to check infectivity. Heavy infections were characterised in both vectors at the blood meal stage, until the blood meals were digested. Infections in *Lu. longipalpis* decreased to 0, 3 days post-bloodmeal. Infections in the *C. soronensis* were retained beyond 10 days to ≈80%, colonising the midgut and stomodeal valve. A newly identified human pathogenic *Leishmania* species responsible for CL in Ghana was able to heavily infected *C. sonorensis*, colonizing the midgut and stomodeal valve up to 10 days and beyond. Although most *Leishmania* spp. causing human disease are transmitted by a sand fly vector, this result leads us to hypothesize that midges could be vectors of this new *Leishmania* species in Ghana. Further proof is required to demonstrate successful transmission of the parasite by *C. sonorensis* to mammalian host.

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ACCURACY OF DIAGNOSTICS IN TEGUMENTARY LEISHMANIASIS: A SYSTEMATIC REVIEW

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Tegumentary leishmaniasis (TL) is characterized by cutaneous and mucocutaneous ulcerative skin lesions, caused by Leishmania parasites, that can potentially disfigure the midface. The clinical presentation of TL is similar to that of epidemiologically overlapping fungal and mycobacterial infections, thereby necessitating confirmatory diagnostics to inform appropriate treatment. Laboratory diagnostic techniques for TL include