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Risk factors of Rift Valley fever in central Sudan

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Background: Rift Valley fever (RVF) is one of the most serious Transboundary, infectious disease. It is a mosquito-borne viral zoonotics disease, causes periodic severe epidemics, characterized by high mortality rate of young animals and abortion in pregnant ruminants.

Many risks factors are associated with the emergence of RVF

Movement of infected animals,Environmental conditions.The modification of the Ecosystem.Demographic change of human.

Objective: To determine potential risk factors associated with RVF occurrence in Central Sudan.

Methods & Materials: Study area: The study was conducted on sheep, goats and cattle herds in Central Sudan.

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Study design: Study population

The study was primarily focused on sheep and goats herd as reference or target population.

Sampling frame and sample size Determination: Cross sectional study design was carried out to determine the seroprevalence of RVF in small ruminants in 2007.

Serum sample was collected, using multistage random sampling technique.

Blood samples were collected identifying sheep and goats from designated geographic localities in the study area.Serological diagnosis was carried out on collected specimens to demonstrate the presence of IgM immunoglobulines by ELISA.

RVF sero-prevalence was estimated by calculating the proportion of the positive cases by the test as nominator and sampled population of sheep and goats as denominator in the study area at given point in time.

Statistical data analysis: The collected data was organized and managed using relevant statistical software package for social sciences (SPSS). To investigate risk factors associated with RVF, the data was analyzed by univariate analysis using the Chi Square test.

Results: States Involved in the Study, Samples Tested, Caprine, Ovine and Bovine by IgM Captured ELISA

State	species									Total tested
	Caprine			Ovine			Bovine			
	sample tested	+ve	PP*	sample tested	+ve	PP*	sample tested	+ve	PP*	
ALgezira	103	63	61%	74	38	51%	43	10	23%	220
The White Nile	22	4	18%	12	6	50%	29	5	17%	65
Sinnar and The Blue Nile				7	1	14%	31	7	22%	38

* PP: positive percentage for Elisa test.

Conclusion: The overall prevalence of RVF in Central Sudan was 40.2%. The highest prevalence was recorded in Gezira state as well as Sinnar state, while the lowest prevalence was recorded in Blue Nile state. Univariate analysis revealed that there is now significant association of state and species with sero-positivity of RVF. While multivariate model showed that goats are associated with sero-positivity of RVF.

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Opposing roles of host IFN-γ signaling during a parasite infection of the mouse

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Background: The protozoan phylum Apicomplexa consists of about 5000 parasite species of medical and veterinary importance, nearly all of which are obligate intracellular pathogens of diverse host cell types. The most prominent genera include *Toxoplasma*, *Plasmodium, Eimeria, Cryptosporidium* and *Theileria*. Intracellular parasites reprogram host functions for their survival and reproduction, which are often preceded by an adaptive and subversive modulation of the host transcriptome. Conversely, the infected host attempts to defend the microbial insult. The extent and relevance of parasite-mediated host responses *in vivo* remains poorly studied, however.

Methods & Materials: We utilized *Eimeria falciformis*, an obligate intracellular parasite completing its entire life cycle in the mouse intestinal epithelium, to identify and validate host determinants of the parasite infection using the contemporary *ex vivo* transcriptomics and classical parasitology methods.

Results: Most prominent mouse genes induced during the onset of asexual and sexual growth of parasite comprised of IFNγ-regulated factors, *e.g.*, immunity-related GTPases (IRGA6/B6/D/M2/M3), guanylate-binding proteins (GBP2/3/5/6/8), chemokines (CxCL9-11) and several enzymes of the kynurenine pathway including indoleamine 2,3-dioxygenase 1 (IDO1). These results indicated a multifarious innate defense (tryptophan catabolism, IRG, GBP, chemokine signaling), and a consequential adaptive immune response (chemokine-cytokine signaling, lymphocyte recruitment). The inflammation- and immunity-associated transcripts were increased during the course of infection, following influx of B-cells, T-cells and macrophages to the parasitized caecum tissue. Consistently, parasite growth was enhanced in animals inhibited for CxCr3, a major receptor for CxCL9-11 present on immune cells. Interestingly, despite a prominent induction, mouse IRGB6 failed to bind and disrupt the parasitophorous vacuole, implying an immune evasion by *E. falciformis*. Furthermore, oocyst output was impaired in IFNγ-R$^{-/-}$ and IDO1$^{-/-}$ mice, both of which suggest a subversion of IFNγ-signaling by the parasite to promote its growth.

Conclusion: Collectively, our study in the *Eimeria*-mouse model identifies a retinue of host determinants regulated by IFNγ, some of which are protective, while others are subverted or even exploited by the parasite. The work also illustrates the value of *ex vivo*