prevalent in India, Bangladesh, Southern Sudan, Nepal and Northeast Brazil. The causative organism of VL in the Indian subcontinent and Africa is *L. donovani*. In India it is quite common in Bengal, Bihar and is causing much mortality every year. In Bihar alone 44,000 cases have been registered many of which are drug resistant cases. In Uttar Pradesh recently few active cases have been seen from eastern districts e.g. Varanasi, Gorakhpur, Gonda, Faizabad etc. It is transmitted by an insect vector i.e. Sand fly (*Phlebotomus argentipes*) that lives in warmer places (cervics, tree hole, dung, etc.) where humidity and temperature both are present at regular intervals in a day (humidity during night and temperature at day time). These conditions are essential/necessary for the survival of vector, parasite development and for their distribution. But now a day, due to global climate changes and temperature increases, which support the high degree of sand fly growth, the transmission of disease has increased manifold. The flooding also increase the transmission of larvae from one place to another place thus also increases the distribution of disease.

**PP-170** Incrimination of *Anopheles* mosquitoes as malaria vectors in guinea savanna zone of central Nigeria

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**Background:** The incrimination of the major vectors of malaria pathogens was done in the guinea savanna zone of central Nigeria.

**Methods:** Adult indoor-resting *Anopheles* were caught with mechanical aspirator. They were identified as *An. Funestus*, *An. rufipes*, and *An. gambiae* sl while PCR assays molecularly delineated *An. gambiae* ss and *An. arabiensis* from the *gambiae* sl and S and M forms of *An. gambiae* ss, respectively. All the anophelines were tested for *Plasmodium* sporozoites by ELISA.

**Results:** A total of 1058 anopheline were caught. *An. gambiae* ss was most preponderant (66.64%) than *An. arabiensis* (19.00%), *An. rufipes* (10.68%) and *An. funestus* (3.69%). The S form (60.39%) was significantly higher than M form (39.07%) (P<0.05) of the *An. gambiae* ss population. 2.27% (n = 16) and 0.43% (n = 3) of *An. gambiae* ss were *An. arabiensis* from the *gambiae* sl and S and M forms of *An. gambiae* ss, respectively. All the anophelines were tested for *Plasmodium* sporozoites by ELISA.

**Conclusion:** *An. gambiae* ss and *An. arabiensis* are the major vectors of *Plasmodium* in the study area.

**PP-171** Detection of *Microsporidia* and *Cryptosporidium* in stool specimens from AIDS patients by Modified Trichrome-Blue and Acid-Fast Trichrome staining methods

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**Objectives:** Severe, chronic diarrhea is a frequent complication of human immunodeficiency virus disease. *Microsporidia* and *Cryptosporidium* are important agents of ententeritis, capable of causing severe chronic diarrhea in AIDS patients. In this study Modified Trichrome-Blue (MTS) and Acid-Fast Trichrome (AFT) staining methods were used for detecting these parasites in stool specimens.

**Materials and Methods:** A total of 71 stool specimens from AIDS patients with chronic diarrhea were collected and transferred to parasitology lab. Two slides were prepared from each stool specimen. Slides were stained with MTS and AFT staining, and observed with 100× objective of light microscope.

**Results:** 18 patients (25.35%) were positive for one or both parasites, 9 patients (12.67%) had only infection with *Microsporidia*, 5 patients (7.05%) showed infection with *Cryptosporidium* and 4 patients (5.63%) had both infection.

**Conclusion:** MTS and AFT stain methods were equally useful in the diagnosis of microsporidiosis. Moreover, AFT stain was suitable for detection of *Cryptosporidium*. These findings emphasize the importance of considering both organisms as potential causative agents of diarrhea in AIDS patients.

**PP-173** Protein variations of *Cryptosporidium* spp. from different sources after proliferation in experimentally immunosuppressed mice

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**Background:** Cryptosporidiosis is a parasitic disease caused by *Cryptosporidium* leading to acute/chronic gastroenteritis. Due to severity of infection in immunodeficiency, there is a requirement for modeling of cryptosporidiosis in experimentally immunosuppressed animals. The aim of this study was to proliferate *Cryptosporidium* spp. from different sources in immunosuppressed Balb/c and C57bl/6 mice and to evaluate protein variations of oocysts from Iranian strains.

**Methods:** Stools samples were collected from mice, calves and human, centrifuged by Parasex kit, smears were prepared and stained with acid fast assay. Oocysts were identified, separated and concentrated by sucrose floatation. Balb/c and C57bl/6 mice were immunosuppressed by Dexamethasone injection; immunosuppression was confirmed by lymphocyte proliferation assay, and isolated oocysts from different sources were orally inoculated into mice. After proliferations of parasites, infected animals were humanely killed, entire contents of digestive tract were discharged and the oocysts were counted, homogenized and proteins were separated by SDS-PAGE and analyzed by Western blotting.

**Results:** The results of lymphocyte proliferation assay showed that the C57bl/6 mice were more immunosuppressed; which facilitate them to be more susceptible to the infection than Balb/c mice. Majority (80%) of C57bl/6 mice infected with *Cryptosporidium*, whereas only 20% of Balb/c mice found infected. Western blott results determined that dominant protein bands varied among the isolated strains from mice (48, 55 KD), calves (55, 67 KD), and human (48, 70 KD).

**Conclusion:** This study is the first report of experimental model of cryptosporidiosis in immunodeficiency in laboratory animals in Iran. These findings indicated that immunosuppressed C57bl/6 mice were more susceptible to *Cryptosporidium* spp. infection than Balb/c ones. The current data indicated different patterns in dominant oocyst proteins of *Cryptosporidium* isolates from different hosts, which may refer to their strain variability.