positive for *hla, hld;* 6 harbored *hlg* and 1 isolate carried *hlb;* prevalence of *sea, sec* was predominant (16% and 7%). Nematode killing assay revealed that LD_{50} of *C. elegans* L4-larvae ranged from 6–12 hrs for HA-MRSA and 12–60 hrs for CA-MRSA strains. Following severe intestinal infection and prolonged paralysis, complete killing of animals occurred between 66–114 hrs.

Conclusion: The present study documents the prevalence of various virulence factors among MRSA isolates. This preliminary study using *C. elegans* model has indicated higher pathogenesis and rapid killing of HA-MRSA strains which showed higher prevalence of leukocidins (*pvl, lukD, lukE*) and enterotoxins (*sea, sec*) than CA-MRSA and so could serve as a model for pathogenesis testing for *S. aureus*.

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Modeling of cerebral tuberculosis in BALB/c mice using clinical strain from patients with CNS -TB infection

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Background: This study describes development of a TBM animal model in mice using C3 strain isolated from CSF of TBM patients which can be used to study pathogenesis of C3 strain in mice.

Methods & Materials: Female BALB/c mice aged 6–10 weeks were housed in a BSL- 3 facility. Two groups of mice (n = 12) were challenged intravenously through tail vein with 2×10^7 of C3 strain. Control group of mice (n = 10) was separately maintained and injected with sterile saline. Mice were killed at 30 and 50 days after development of infection, for estimating number of mycobacteria colonizing in brain,lungs and for histopathological changes. The mycobacterial burden was determined by plating and counting the number of CFUs. Serially diluted homogenates of individual lungs and brains were plated onto Middlebrook 7H11 medium. CFU's were counted after 3–4 weeks of infection. Histological sections were stained using hematoxylin and eosin.

Results: Mice infected with C3 strain showed prominent edema of the brain in left hemisphere at 30 days, which increased at later stage compared to control mice. Histopathological examination of brain showed swelling of neurons along with lymphocytic infiltration which was progressively more at 50 days. The lung section at 30 days revealed >40 lession in lung parenchyma which improved at 50 days, with lession reduced to <20. Brain and lungs of control mice showed no major changes. Infection with C3 strain showed significant levels of mycobacterial load in brains with progress in infection. Analysis of CFU count revealed significantly high load in lungs (6.80 ± 0.1) at 30 days post infection, however the mycobacterial burden gradually decreased in lungs (5.8 ± 0.2) with progress in infection at 50 days. Analysis of CFU load in brain showed significantly high mycobacterial load at 50 days (4.0 ± 0.2) compared to infection at 30 days (5.0 ± 0.1). No mycobacterial load was observed in brains and lungs of control mice.C3 strain infection was associated with reduced survival-40% and high mortality rate -60%compared to control mice.

Conclusion: Our present study demonstrated that intravenous inoculation by C3 strain from TBM patient's leads to progressive dissemination and development of TBM disease in mice.

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Genome wide differential host response to highly or low pathogenic H5N1 avian influenza virus infection in ducks

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Background: The underlying molecular mechanisms of pathogenesis and outcome of disease to different pathotypes of H5N1 influenza infection in ducks remain unclear.

Methods & Materials: Hence, we studied genome wide host gene expression of duck lung tissues infected with A/duck/India/02CA10/2011 (AD2011) and A/duck/Tripura/103597/2008 (AD2008) H5N1 viruses using custom designed microarray. AD2011 is highly pathogenic whereas AD2008 is low pathogenic to ducks.

Results: Comparative analysis of differentially expressed genes revealed that 688 genes were commonly expressed, 877 and 1556 genes are uniquely expressed to infection with AD2011 and AD2008 virus isolate, respectively. The up-regulation of cytokine (IL17), chemokines (CCL4 and CXCR4) and IFN stimulated genes (OAS, IFITM2, STAT3, TGFB1 and TGFB2) in the lungs tissues possibly caused high mortality in ducks infected with AD2011 virus. The expression of important antiviral immune genes IFIT5, IFITM5, RSAD2, EIF2AK2, Mx, β-defensins, TRIM23 and SLC16A3 in AD2008 infection, but not in AD2011 infection, might fine-tune the innate immune responses and prevent cytokine storms and tissue damage. Several immune related gene ontology terms and pathways activated by both the viruses were qualitatively similar but quantitatively different.

Conclusion: Based on these findings, we conclude that subtle differences in host immune responses may determine the different outcome of H5N1 infection in ducks.

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