Results: There were 104 patients conducted the study, 93 (89.4%) were men which aged 40 – 49 years old are the highest prevalence (n = 28; 26.9%). Tender hepatomegaly was found in 93 (89.4%) which 87 (83.6%) among them were right lobe liver abscess, right upper abdominal quadrant pain occurred in 98 (94.2%), fever in 89 (85.6%), and leukocytosis in 79 (75.9%) patients. A right lobe hepatic abscess was seen in 87 (83.6%) patients. Tuak was consumed by 91 (87.5%) for 5-10 years. Liver puncture was performed in 79 (76%) patients and we evacuated liquid with variable color, and the bacterial cultures were all negative and the amoeba was not found in all of the samples. All of the patients showed improvement after medication.

Conclusion: The presentations of ALA in Medan are mainly right upper abdominal quadrant pain, fever, tender hepatomegaly, leukocytosis, affect the right hepatic lobe, age 40-49 years old, male, thus a male presented those symptoms with history of tuak consumption increases our suspicion of amoebic liver abscess. Amoeba is highly suspected as the cause of ALA while the medication to this showed improvement.

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Role of vitamin molecules on host gene down regulation and effect on *Leishmania donovani* infection in an experimental animal model

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Background: Vitamins have as long as been recognized for their protective role against tuberculosis. Several studies have shown the role of vitamin D status in tuberculosis patients. Recently, a gene coding for tryptophan-aspartate containing coat protein (TACO) has been recognized to play a crucial role in the survival of *Mycobacterium tuberculosis* within human macrophages and host molecule TACO has been shown to play a important role in the arrest of such a maturation process. Studies showed that synergistic action of Vitamin D3 and Retinoic acid had inherent ability to down-regulate TACO gene transcription in human macrophages and this leads to early maturation of phagosome and pathogen elimination. We are postulating that a similar stable association of TACO with phagosomes would prevent the vacuole from maturation, with a role of TACO in this process in case of *Leishmania*.

Methods: The present study evaluated the effect of treatment with Vitamin D3/Retinoic acid & Chenodeoxycholic acid/Retinoic acid combinations on *Leishmania donovani* infection in BALB/c mouse model. Animals were sacrificed at different time intervals. Parasite load was determined in the impression smears of liver and spleen of control and drug treated animals. histological examinations at different time intervels.

Results: It was found the parasite load and weights of spleen and liver increased till day 28 post infection in control mice, whereas in mice treated with above-mentioned drugs there was a significant decline in weights of the organs and parasite load on days 21 and 28

Conclusion: Our results elucidate that the Vitamin D3/Retinoic acid and Chenodeoxycholic acid/Retinoic Acid treatment led to significant suppression of weights of spleen, liver and parasite load. Healing responses in histological specimens of above molecules treated animals response suggested that the drug treatment down regulated TACO gene expression as was observed in our in-vitro experiments. Vit-D3/Retinoic acid combination showing a significant level of parasite suppression with better immune modulation in comparison with the Chenodeoxycholic acid/retinoic acid combination. These observations suggest that Vit-D3/Retinoic acid and Chenodeoxycholic acid/Retinoic acid may possibly be an important adjunct to the treatment of Leishmaniasis.

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Differentially expressed *Leishmania major* genes might discriminate between clinical isolates of contrasted virulence

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Background: Leishmaniasis is a vector borne parasitic disease affecting millions of people worldwide that causes different clinical manifestations classified as cutaneous, muco-cutaneous, or visceral depending on the infecting specie. Zoonotic cutaneous leishmaniasis (ZCL) caused by *Leishmania* (*L.*) *major* clinically presents as localized self-healing cutaneous sores with a broad range of clinical variations. Whether this clinical spectrum could reflect a differential role played by *L. major* in disease outcome and severity has been very little investigated.

Our hypothesis is that genetic variation among *L. major* parasites, possibly acting at the level of virulence genes expression, might impact, in concert with the host immune response, on the clinical polymorphism of ZCL (severe *vs.* benign disease or asymptomatic infection).

In an attempt to correlate parasite diversity with disease outcome, our goal was to establish a list of selected putative virulence-associated genes that could discriminate between *L. major* isolates collected from the field.

Methods: Human blood derived macrophages were *in vitro* infected with two *L. major* strains expressing contrasted virulence (high *vs.* low virulence) according to their experimental pathogenicity in Balb/C. Our approach was first based on a high throughput transcriptome analysis, namely Serial Analysis of Gene Expression. More than two hundred parasite transcripts showed differential expression between the high- or low-virulent strains at their intracellular infectious stage. Among transcripts that we could confidently assign to their related gene, four (LmjF26.1710, LmjF28.2205, LmjF33.1740 and LmjF33.1750) were tested on human macrophages from donors infected with different parasite strains.