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Evaluation of p27 gene knockout mutant of Leishmania major (MRHO/IR/75/ER) with light and electron microscopy

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Leishmaniasis as a parasitic disease with manifestations ranging from cutaneous to fatal visceral infection is caused by several *Leishmania* species. These protozoan parasites in the gut of a sandfly vector or in culture media replicate as extracellular, flagellated promastigotes and inside the parasitophorous vacuole of vertebrate host macrophages as amastigotes. In Iran Zoonotic Cutaneous Leishmaniasis (ZCL) caused by *Leishmania major* (*L. major*). *L. major* p27 is an important gene encoding cytochrome c oxidase (COX) component and is a mitochondrial membrane protein. It expresses a 27 kDa protein that has potential role in ATP synthesis. This study aimed to evaluate morphology of *L. major* mutant (deficient in the p27 gene) with light and Scanning electron microscopy (SEM) imaging methods and its pathogenicity in BALB/c mice.
Evaluation of *L. major* mutant promastigotes with invert light microscope (400X) showed that proliferation and differentiation is slower than virulent ones but in term of morphology with light microscope (1000X) and Gimsa staining, we didn't see any difference in nucleus, kinetoplast, body and flagella between them. Results of Scanning electron microscopy also demonstrated no difference between body and flagellum although the parasites have a highly resolution in cell shape and form. Also in BALB/c mice the mutant didn't cause any lesion and in 75% of them we didn't see any amastigote in their smears (1000X). In conclusion, there is no difference between Knockout and Virulent *L. major* with light microscopic and SEM methods. It seems Transmission electron microscopy (TEM) is more useful in analysis of morphology and differentiation of produced p27 gene knockout mutant.