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Commentary

In silco Analysis of *Leishmania donovani* Genome to Understand its Gene Expression Regulation

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DESCRIPTION

Medically significant intracellular parasites called *Leishmania* spp. are responsible for a variety of deadly diseases in humans, from minor cutaneous lesions to more serious and life threatening visceral infections. Because there are currently no vaccines for *leishmaniasis* and just a few chemotherapeutic drugs available for usage, the illness is still a severe public health concern in many nations across the world. The illness, which has two primary clinical forms cutaneous and *Visceral leishmaniasis* is common in tropical and subtropical areas and is known to have zoonotic and human to human transmission patterns.

Leishmania donovani is thought to reside in the phagolysosomes of macrophages and is an obligatory intracellular parasite. Once inside the macrophage, phagosome mutation regulation will aid the parasite's growth and eventually protect it from being destroyed. As a result, nutritional availability will be preferred at the expense of antigen presentation, oxidative damage, apoptosis and immunological activation. The expression of genes in the parasite and the host are both impacted by the parasite's almost exclusive ability to multiply in the phagolysosomes of the infected macrophage. *Leishmania donovani* appears to have two separate genomic characteristics that affect its genetics: either varied chromosome copy counts or varying chromosome dosage among clonal populations of cell.

After further activation with Interferon (IFN), an infection develops a mechanism that reduces macrophages' ability to kill bacteria by interfering with the production of Major Histocompatibility-II (MH-II) molecules. The parasites interact with host cells *via* using a variety of proteins and glycoproteins, causing a long lasting infection that manifests clinically as *Visceral leishmaniasis*. *Visceral leishmaniasis* can cause a number of side effects, such as hepatic cirrhosis, necrotizing oral infections, pulmonary tuberculosis, ocular complications, disseminated intravascular coagulations, nephritic syndrome, glomerulonephritis, neutropenia, immunosuppression and pancytopenia, which can result in concurrent or intercurrent infections and ultimately lead to death. *Leishmaniasis* can be treated with a variety of medications, including first and second line regimens. The usage of the pharmaceuticals that are readily available may be governed by local regulations and differs in different areas.

Transcriptional organisations, such as the Transcriptional Start Sites (TSS), may control changes in a parasite's ability to adapt to its environment, the formation of silent genes and the progression of the parasite through its life cycle (TSS). Through transcriptional and translational controls, *Leishmania donovani* mostly loses its virulence. Therefore, it is important to fully characterise the molecular mechanisms of *Leishmania donovani* in order to develop efficient medications, vaccines and other control strategies. To do this, it is helpful to understand the regulatory elements such as Transcription Factors (TFs), Transcription Factor Binding Sites (TFBSs) and CpG islands in *Leishmania donovani*'s genome that determine its gene expression.

In eukaryotes, transcription regulation that occurred between 50 and 200 bps upstream of the TSS was connected with promotor proximal elements, while it occurred within 25 bps of the core promoter. On the other hand, the distal enhancer promoter was linked to transcription regulation in any direction and orientation far from transcription beginning sites. The results of this study's investigation revealed that the optimal TSS for one promoter sequence was found at a distance of -2 bps from the start codon, where transcription is linked to the core promoter. On the other hand, promotor proximal elements were related with the locations of the best TSSs in seven promoter sequences.

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