S E M I N A R

Department of Biological Sciences

NUS National University of Singapore

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Hosted by Dr Cynthia He

Small interference RNAs originated from pseudogene and natural antisense transcripts regulate gene expression in African *Trypanosoma brucei*

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Pseudogenes have been shown to acquire unique regulatory roles from more and more organisms. We reported the observation of a cluster siRNAs derived from pseudogene of *Trypanosoma brucei* using high through-put analysis. We also presented genome-wide investigation of the other siRNAs, such as natural antisense transcripts (NATs), which were considered to play an important regulatory role at the level of post-transcription according to the transcripts annotation and the deep-sequencing results. Some of these siRNAs, including pseudogene-derived siRNAs and NATs-derived siRNAs were proved to suppress gene expression through RNA interference. Specially, hundreds of NAT pairs forming complex regulatory networks in *T. brucei* were yielded and most of them were only present in the bloodstream form. This discovery of functional siRNAs originated from pseudogenes and NATs provided insights into the new origin of the non-coding small RNAs and the novel function of pseudogene and NATs in *T. brucei*. And the specificity of NATs in the bloodestream form indicated their regulatory potential on the control of differentiation of *T. brucei*.

Trypanosoma lewisi, a considered species-specific rat trypanosome, is a human pathogen?

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Trypanosoma lewisi has widely been considered as a non-pathogenic rat trypanosome. However, more and more cases of humans infected with *T. lewisi* have been reported around the world, indicating that it can infect humans in some undetermined circumstances. Human-infectious trypanosomes such as *Trypanosoma cruzi, T. brucei rhodesiense* and *T. b. gambiense* can be discriminated from animal ones by their resistance to normal human serum (NHS). These parasites naturally resist trypanolysis induced by the human-specific pore-forming serum protein apolipoprotein L1 (apoL1). Here we demonstrate that *T. lewisi* is resistant to lysis by either NHS or recombinant apoL1, both *in vitro* and *in vivo.* Together with recent reports of human infection by *T. lewisi*, these data allow the conclusion that *T. lewisi* is a human pathogen.

Artwork by Ann Nee, DBS, NUS