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Erik Vassella

African trypanosomes, which cause human sleeping sickness, are responsible for severe medical and economical problems in large areas of sub-Saharan Africa. There are currently 500'000 cases of human sleeping sickness per year as estimated by the WHO. The disease is fatal if untreated, there is no vaccine and the few drugs that are available are either extremely toxic or only effective for a restricted form of the disease. Hence, this disease, which is neglected by pharmaceutical industry, is one of the major health concerns in many developing countries. The parasite depends on the tsetse fly for its dissemination. During cyclical transmission, it undergoes differentiation into distinct life-cycle stages, which are adapted to their respective environment. Bloodstream forms proliferate in the blood of the mammalian host. This environment provides optimal growth conditions for bloodstream forms since it is warm and rich in nutrients. However, trypanosomes have to fight against elimination by the host immune system. In contrast, the gut of the tsetse fly is rather cold and poor in nutrients. In addition, parasites of this compartment have to cope with digestive enzymes.

How can trypanosomes sense their environment and how can they adapt to these dramatic changes during cyclical transmission? The aim of my project supported by the Hans Sigrist Foundation was to identify extracellular signals controlling differentiation of trypanosomes. In addition, I was interested in elucidating the molecular mechanisms mediating the differentiation program and resulting in changes in gene expression. Compounds that interfere with a specific differentiation process may be lethal for the parasite and thus may be used as a drug against human sleeping sickness. This work led to several publications and will be an integral part of my habilitation.

Contact address:
Dr. Erik Vasella, Institut für Pathologie, Murtenstrasse 31, 3010 Bern,
E-mail: erik.vasella@pathology.unibe.ch