BSP 2026 Abstract – Federica Giordani

Molecular mechanisms of *Trypanosoma evansi* resistance to trypanocidal drugs

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Trypanosoma evansi is the causative agent of surra, a veterinary disease affecting a variety of domestic animals including camels, buffalos, horses and cattle. Surra is endemic across tropical and subtropical regions, where it is mainly transmitted by hematophagous insects. To treat *T. evansi* infections, the same trypanocides widely used to kill African animal trypanosomes are used. However, poorly regulated regimens and widespread use have inevitably raised concerns about drug resistance selection. Despite the severe and wide-ranging negative consequences of surra on livestock and the agricultural economy, data on trypanocide resistance and its determinants in *T. evansi* remain scarce, hampering our understanding of the extent of the problem and a more rational deployment of drugs.

Although *T. evansi* could be classified as a subspecies of *T. brucei*, the absence of the kinetoplast, an important target for many trypanocidal drugs, allows for a more targeted evaluation of their mode of action and resistance in the former. We are investigating the molecular basis of resistance to a panel of trypanocides (diminazene, isometamidium, suramin and the new valyl ester benzoxaboroles) in *T. evansi* AnTat 3/3, to evaluate the mechanisms underlying resistance development in this akinetoplastic trypanosome strain. Resistant cells are selected *in vitro* by stepwise exposure to increasing concentrations of trypanocides, and genomic DNA is then extracted and analysed by whole-genome sequencing. The identification of resistance determinants is initially guided by established knowledge from *T. brucei*. However, we also aim to identify features that might be unique to *T. evansi*, as comparative analysis may uncover distinct resistance mechanisms that merit further investigation.