antimicrobial peptides (AMPs), whose antibacterial properties have been extensively studied. However, anti- fungal peptide's activity has not attracted a lot of attention so far, especially the *in vivo* activity of such peptides. The mealworm beetle, *Tenebrio molitor*, expresses an antifungal peptide, the tenecin3, which is a glycine-rich peptide, closely related to plant defensins and drosomycin, with no antibacterial activity and of unknown mode of action. Tenecin 3 has been proven to have *in vitro* antifungal activity against *Candida albicans*, but its activity against entomopathogenic fungi has never been studied. We used a gene knock-down approach to study the effect of tenecin 3 on the survival of *T. molitor* after an infection by two different strains of *B. bassiana* and one strain of *B. pseudobassiana*. Interestingly, the mortality pattern varied among the three strains whether tenecin3 was down-regulated or not.

CONTRIBUTED PAPERS — Wednesday, 13:30-15:30 - ***Vouvay***

Virus 5 - *Ikbal Agah Ince & Sassan Asgari*

Contributed paper. Wednesday, 13:30, **152-STU**

Interactions between the salivary gland hypertrophy virus and its host immune system

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Tsetse flies (Diptera; Glossinidae) are naturally infected by *Glossina pallidipes* salivary gland hypertrophy virus (GpSGHV; *Hytrosaviridae*); a large dsDNA virus specifically pathogenic to *Glossina* spp. GpSGHV infections are largely asymptomatic in most of the tsetse species. In *G. pallidipes* asymptomatic infection can convert to symptomatic infection that is characterized by overt salivary gland hyperplasia (SGH). This syndrome also leads to reproductive dysfunction of infected flies. We hypothesised that GpSGHV infection is maintained at low levels by dsRNA-mediated gene silencing, such that only few viral genes are expressed during asymptomatic infections. To test this hypothesis, we first investigated whether host-mediated dsRNA mechanisms are involved in asymptomatic virus infection by comparative analyses of *Argonaute* (*Ago*) and *Dicer* (*Dcr*) gene expression levels in asymptomatic and symptomatic *G. pallidipes*. We found that both *Ago* and *Dcr* were significantly up-regulated in symptomatic compared to asymptomatic flies. Furthermore, short RNA sequence analyses indicated that more small RNAs (19 miRNAs) were produced during symptomatic infections compared to asymptomatic infections (8 miRNAs). When mapped onto the host (*Glossina*) genome, the miRNAs in the asymptomatic flies mapped onto several genes with a putative relation to regulation of transcription, translation, macroautophagy, immunity, apoptosis and tumour suppression. In symptomatic flies, majority of miRNAs mapped to metabolic-related genes and a few to transcription genes. We recently set up knock-down bioassays to investigate the involvement of the miRNA targeted genes in regulating GpSGHV infection in *G. pallidipes* flies.

Contributed paper. Wednesday, 13:45, **153-STU**

Host range of Glossina pallidipes salivary gland hypertrophy virus (GpSGHV)

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The salivary gland hypertrophy virus (SGHV) is a dsDNA virus (family: *Hytrosaviridae*), and It has been reported in many species of tsetse fly (Diptera: Glossinidae). Generally, the virus infection is asymptomatic but in certain tsetse species i.e. *Glossina pallidipes* (Gp), the virus infection can convert to symptomatic and cause the salivary gland hypertrophied (SGH) symptoms. The high prevalence of SGH in tsetse colony is associated with a reduction of flies fecundity and fertility, which may cause colony collapse. To understand the molecular mechanism controlling the development of SGH in Gp and its rare presence/absence in other tsetse species, we attempted to analyse the host range of the GpSGHV in other tsetse species. The GpSGHV virus collected from SGH of Gp and injected into tsetse adults and 3rd instar larvae of Gp, *G. fuscipes* (Gf) *G. brevibalpis* (Gb), *G. p. gambiensis* (Gpg), *G. m. morsitans* (Gmm) and *G. m. centralis* (Gmc) Virus quantification at different times post injection indicated an increase of virus titre in the adults of all injected species except Gb. Dissection of both injected flies and F1 generation showed no development of SGH except *G. pallidipes* F1 generation (46%). Dissection of the flies 10 days post-emergence from injected larvae indicated the presence of SGH in Gp (67 %), Gf (26 %), Gpg (18 %), Gmm (9%), Gmc (6 %) and Gb (0 %). The hypertrophied salivary glands observed in the heterologous species were smaller than SGH normally found in Gp. These results indicate that (i) the GpSGHV can replicate in other tsetse species and (ii) the development of SGH requires a component from immature stages.

Contributed paper. Wednesday, 14:00, **154**

Highjack of intracellular signalling pathways and robust immune responses explain the hytrosavirus-induced differential pathologies in two *Glossina* model species

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Glossina pallidipes salivary gland hypertrophy virus (GpSGHV; *Hytrosaviridae*) is exclusively pathogenic to tsetse flies, vectors of African trypanosomoses. GpSGHV infection is largely latent, but can switch to a symptomatic infection state leading to salivary gland hyperplasia (SGH) and reproductive dysfunction. Of all tsetse species, *G. pallidipes* is the most susceptible to overt SGH symptoms. Whilst in naturally infected *G. pallidipes* SGH occurrence is the exception rather than the rule, SGH is only apparent in the F1 progenies of artificially infected *G. pallidipes*