genomes, the target sequence for PURα proteins. Interestingly, these GGN-rich sequences are disproportionally distributed in baculoviral genomes and mostly occurred in proximity to the polyhedrin gene. At the same time they encode crucial proline-rich domains in *p78/83*, an essential gene adjacent to the *polyhedrin* gene in the AcMNPV genome. We further demonstrate that the VP1054 protein specifically recognizes GGN-repeats and are currently analyzing the significance of these GGN motifs for DNA packaging. Together, whilst some viruses like human immunodeficiency virus 1 (HIV-1) and human JC virus (JCV) utilize host PURα protein, baculoviruses encode the PURα-like protein VP1054, which is crucial for viral progeny production.

SYMPOSIUM (Special) Thursday, 8:00-10:00

DFG Priority Program Host Parasite Coevolution

Symposium. Thursday, 8:00 **223**

Escaping parasite manipulation: Apoptosis and host-parasite co-evolution in *Apis mellifera*

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Programmed cell death (apoptosis) does not only play an important role in the development of multicellular organisms, but also in the protection against pathogens. Nevertheless, numerous intracellular pathogens have evolved diverse strategies to interfere with and overcome the apoptotic machinery of their hosts. Yet, little is known about the actual mechanisms and how hosts might counter act. We here study the interaction of the intestinal microsporidian parasite *Nosema ceranae* in a susceptible and tolerant honeybee host under laboratory controlled conditions, to understand the importance of apoptosis in this case of host-parasite co-evolution. We visualize apoptotic processes in the gut epithelium using TUNEL assays; relate this to the expression levels of key genes in the apoptotic cascade over the course of the infection, and consequences for metabolic energetics affecting honeybee performance.

Symposium. Thursday, 8:15 **224**

Overcoming external immunity: An increase in virulence as a result of host-parasite coevolution in *Beauveria bassiana*

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An increase in virulence is a trait often observed as a result of host-parasite coevolution. Specific immune responses overcome in order to achieve increased virulence can, however, be difficult to elucidate. We carried out a coevolution experiment with the red flour beetle, *Tribolium castaneum*, and the general entomopathogenic fungus, *Beauveria bassiana*. After just seven host generations of evolution we saw a substantial increase in virulence in all evolved isolates of *B. bassiana*. Furthermore, we were able to show that this increase in virulence was a result of the *B. bassiana* isolates evolving resistance to the external immune defences of the *T. castaneum* beetles, who are able to secrete antimicrobial compounds into their environment. This is a rare example of a virulence increase seen as a result of a coevolution experiment where the exact barrier of host immune defence that the parasite has gained resistance to in order to achieve the increase in virulence has been described.

Symposium. Thursday, 8:30 **225**

Rapid adaptation of *Bacillus thuringiensis* to its nematode host *Caneorhabditis elegans*

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Antagonistic interactions between host and pathogen can produce very high selection intensities. They are often one of the main driving forces during evolution, especially if the interactions persist across time. We specifically assessed the evolutionary impact of these interactions for the pathogen, using evolution experiments with the Gram positive biocontrol agent *Bacillus thuringiensis* and one of its animal hosts *Caenorhabditis elegans*. Our results demonstrate that differences in the experienced selection conditions during the evolution experiment favour distinct characteristics across the pathogen life cycle: (i) pathogen adaptation to a co-evolving host associates with high virulence; (ii) pathogen adaptation to a non-changing host increases infection load; whereas (iii) adaptation without host favours environmental persistence. Concomitant genomic changes in the pathogen were observed at two levels: (i) the different evolution conditions caused clonal selection of distinct, broad-scale genotypes, while (ii) one of these with high virulence showed additional nucleotide changes, including copy number variations of nematocidal toxin genes. Based on one of the most comprehensive data sets collected for an experimentally evolved pathogen, we conclude that sustained coevolution is distinct from other types of selective constraints in shaping pathogen genome and life-history characteristics. Surprisingly, our findings also suggest that sustained virulence, as desired for pest control, may be contingent on the unwanted co-adaptation of the target host.

Symposium. Thursday, 8:45 **226**

Intra-host parasite interactions between co-infecting *Bacillus thuringiensis* strains

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Hosts and parasites are expected to influence each others evolution due to antagonistic interactions, potentially leading to host-parasite coevolution. However, many studies focus on the interactions between hosts and parasites, ignoring that within one host different parasite genotypes may interact and may thus feed-back on the coevolution between host and parasite. The interactions between parasite genotypes may range from competition between genotypes for limited host resources to cooperation for more efficient host exploitation. Using *Caenorhabditis elegans* as host and the bacterial microparasite *Bacillus thuringiensis* we found indications for diverse interaction strategies between the bacteria, ranging from public good to spiteful bacteriocin production. However, it remains unclear how stable these strategies are over the course of time, i.e. when hosts have to be repeatedly infected and when hosts may also adapt to these parasite strategies. For this reason, we performed a laboratory-based selection experiment in which either single *B. thuringiensis* genotypes or a mixture of strains coevolved with hosts. After 10 host generations, we found differences between the evolution treatments. Most interestingly, mixed infections strongly lost virulence. Whether this is caused by a trade-off between host-exploitation and bacterial competition or by division of labour between bacterial clones remains to be shown. Importantly, these results have strong implications for epidemiology, since the evolution of bacteria and its consequences for the host depend on the multitude of infection.

Symposium. Thursday, 9:00 **227**

Experimental evolution *in silico*: host-parasite coevolution versus parasite adaptation

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Bacillus thuringiensis is a widely distributed natural pathogen of invertebrates and plays an important role in (agricultural) pest management. The bacteria kill the host by CRY toxins and other virulence factors. Recent experimental studies on the evolution of virulence revealed that one-sided adaptation of *B.t.* with non-evolving hosts (*Caenorhabditis elegans*, *Tribolium castaneum*) selects for intermediate or no virulence, sometimes coupled with parasite extinction. In contrast, host-parasite co-evolution selects for high virulence and for hosts with strong resistance against *B.t.* However, a sound theoretical explanation is missing. Here, we propose a new mathematical model that mimics the experimental set-up. We consider two bacterial strains, a virulent “toxin producer” and an avirulent “non-toxin producer”. Bacterial evolution is modeled as an iterated process of intra-host dynamics and bacterial transmission between hosts. The intra-host dynamics are described as a two-phase process, where the first phase covers the period from beginning of infection until host death and the second phase the period from host death until depletion of host resources. Increase in host resistance is simulated by extending the first phase. Our model analysis revealed, in general, the same basic trends as the above-mentioned experimental studies. Especially, we could show that resistant hosts select for highly virulent bacterial strains. Moreover, we found (1) that the evolved level of virulence is independent of the initial level of virulence, and (2) that the bacterial dosage significantly affects the evolution of virulence with low dosage selecting for highly virulent strains. These predictions can be tested in future experiments.

Symposium. Thursday, 9:15 **228**

Immune priming with *Bacillus thuringiensis* in *Tribolium castaneum*

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There is accumulating evidence for a memory-like phenomenon in the immune defence of invertebrates. Such 'immune priming' can be rather specific, and might be transmitted from parents to offspring. Invertebrates do not possess the machinery of the vertebrate adaptive immune system, and the mechanistic underpinnings of immune priming are still largely unknown. In the red flour beetle *Tribolium castaneum*, immune priming for resistance against the entomopathogen *Bacillus thuringiensis* has been demonstrated, both within and across generations. Immune priming arose after septic 'pricking' as well as oral pathogen exposure. Moreover, not only mothers, but also fathers were able to transmit such resistance to their offspring. In this talk I will present our recent approaches to deepen our understanding of the evolutionary relevance and mechanistic underpinnings of immune priming in this host-pathogen system.

Symposium. Thursday, 9:30 **229**

Rapid reciprocal adaptation between the red flour beetle and *Bacillus thuringiensis* bacteria during experimental coevolution

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The antagonistic interaction between hosts and parasites is a powerful evolutionary force that may drive rapid evolutionary adaptation. It can lead to coevolution by reciprocal adaptation and counter-adaptation of hosts and parasites. However, in natural populations, it is very difficult to exclude other selective forces that may influence the interaction and to identify true coevolution. We thus performed experimental coevolution in the laboratory between the red flour beetle and *Bacillus thuringiensis* bacteria. We made use of an experimental design that included control treatments in which either of the antagonists was allowed to adapt to a non-evolving host or parasite, respectively, and we also controlled for a possible adaptation to laboratory conditions. We here report on evolved differences in the phenotypes of host and parasite,

and in particular an observed increase in parasite virulence and host resistance. Moreover, we found a potential for parasite local adaptation under coevolution.

Symposium. Thursday, 9:45 **230**

Means of fast virulence adaption: the plasmid and prophage equipment of selected *Bacillus thuringiensis* strains

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Strains of *Bacillus thuringiensis* (Bt) are used since decades as pest control in crop protection. A descriptive feature of the species is the existence of paracrystal bodies, which consist of δ-endotoxins, acting against specific classes of invertebrates. Over the years a solid amount of research has been achieved on the activity of δ-endotoxins on invertebrates as well as on the diversity of cry-toxin genes. In contrast surprisingly little is known on the genomic loci which encode this diversity of δ-endotoxins. Furthermore the knowledge on other invertebrate virulence factors encoded by Bt as well as on host adaptation factors is rather fragmentary. The observation of phenotypes that differ between strains indicates that they are encoded within the pan-genome of *Bacillus thuringiensis*. Since a pan-genome consists of the genes that are not shared by all members of species many of them are encoded on strain specific extra chromosomal elements. Here we present a comparative analysis of more than 40 extra chromosomal replicons such as plasmids and prophages of three nematocidal and two insecticidal Bt strains.

SYMPOSIUM 8 (Cross-Divisional) Thursday, 14:00-16:00

Host – Pathogen Ecology at the Molecular Level: Gene Regulation and Environment Sensing

Symposium. Thursday, 14:00. **231**

The *Bacillus thuringiensis* way of life: communicate to kill and survive in the insect host

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At the end of exponential growth, bacteria of the *Bacillus cereus* group (*ie. B. thuringiensis* and *B. cereus*) produce virulence factors allowing the bacteria to invade their host. In the insect gut, genes controlled by the PlcR quorum sensor allow the bacteria to damage the intestinal barrier and to gain access to the haemocoel. After the death of the insect, PlcR activates transcription of a gene encoding a second quorum sensor, NprR. NprR induces production of degradative enzymes and of a biosurfactant allowing the bacteria to survive in the insect cadaver and eventually to sporulate. The development of the sporulation process is controlled by the master regulator Spo0A, whose activity is regulated by Rap proteins. PlcR, NprR and Rap are quorum sensing regulators belonging to the RNPP family. Their activity depends on the signalling peptides PapR, NprX and Phr, respectively. Altogether our results indicate that these three cell-cell communication systems, acting sequentially, coordinate virulence and adaptive properties with the general physiology of the bacterial cells. The PlcR-PapR complex induces the production of virulence factors allowing the bacteria to kill the insect. NprR-NprX activates transcription of genes allowing the bacteria to switch from a virulence state to that of survival in the host cadaver. Ultimately, the inhibition of the Rap proteins by the Phr signalling peptides triggers sporulation, thus allowing the bacteria to disseminate and to persist in the environment.

Symposium. Thursday, 14:30. **232**

The interplay of Paenibacillus larvae with honey larvae during infection

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Honey bees are attacked by numerous pathogens, some of them just causing covert infections others causing overt disease symptoms and even death of individuals and entire colonies. Among the latter group is the bacterium *Paenibacillus larvae*, the etiological agent of the epizootic American Foulbrood of honey bees (AFB). As the name suggests, AFB is a bacterial disease affecting only the larval stages of honey bees. *P. larvae* is an obligate killer because death of larvae and conversion of larval biomass into bacterial biomass are prerequisites for disease transmission within and between colonies. Hence, *P. larvae* must have evolved effective means to attack larvae, to circumvent the larval immune response and to finally kill and decompose larvae. We recently identified and characterized some of these virulence factors of *P. larvae*. We will present a model for molecular pathogenesis of *P. larvae* infections built upon these novel findings in order to further the understanding of the molecular basis of pathogen-host-interactions in American Foulbrood disease.

Symposium. Thursday, 15:00. **233**

Antimicrobial defense and persistent infection in insects revisited

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Antimicrobial peptides are mainly produced and used by multicellular organisms such as insects to interact with pathogenic and mutualistic micro-organisms. Antibiotics are mostly produced by single cell eukaryotes and bacteria. Here we provide a possible explanation for this dichotomy. Our hypothesis is based on the observation that antibiotics elevate bacterial mutation rates and we show that AMPs do not elevate bacterial mutation rates. Nevertheless we also found that bacterial resistance evolves readily against single AMPs in vitro, but the situation is already more complicated by the simultaneous action of two AMPs. I will contextualize these findings in the light of the immune responses of the beetle Tenebrio molitor and will use these findings to discuss some of the multiple roles AMPs have in host-microbe interactions: policing and killing.