The Mite – Make it Right

Project 1: The final hurdle to chromosome level assembly of *T.urticae* genome
Project 2: Steps towards a richer molecular-genetic understanding of diapause

This document contains snippets from both papers. It is more background then I will discuss during lab meeting since my main goal is to give an overview of the main results of each project. **Not required reading.** This is just extra background if anyone is interested.

Project 1 Introduction (unedited first paragraph of manuscript)

Arthropod herbivores represent a good system to study evolutionary processes as they show a high adaptive potential towards novel host plants and pesticides (Gassmann et al., 2009; Schoonhoven et al., 2006; Strong et al., 1984). Even though host plant use is a complex trait that enables the herbivore to recognize, assimilate and detoxify plant metabolites, arthropod herbivores can adapt to a new and biochemically distinct host in just a limited number of generations. Due to host plant adaptation, the host plant range of arthropods is dynamic and may shift, expand or contract, depending on the genetic correlations of host-associated performance across different plant species (Savolainen et al., 2013). The accumulative adaptive differentiation between populations adapting to different plants often leads to reproductive isolation, in a process called ecological speciation (Schluter, 2001). As a result, many arthropod species actually consist of a group of genetically diverging host races specialized to different host plants. However, despite the evolutionary trend to specialize to certain hosts, true generalist arthropod herbivores exist and are able to feed and adapt to a wide range of hosts belonging to various plant families (Bernays & Graham, 1988; Schoonhoven et al., 2006; Strong et al., 1984). Like host plant use, pesticide resistance in arthropods is a highly evolvable trait and is typically achieved by an increased insensitivity of the pesticide targets and/or an increased metabolic detoxification of the pesticide. Pesticides differ in their mode of action and enforce different selection pressures on arthropod herbivores, resulting in the evolution of different resistance mechanisms (Knowles, 1997). Although pivotal in our understanding of evolutionary biology, the genetic basis of these and other adaptive processes is still poorly understood and heavily discussed (Orr, 2005). This discussion mainly centers on the relative importance of a mono- vs polygenic basis of adaptation and whether adaptation is based on standing genetic variation or newly arisen genetic mutations. Research on the genetic basis of adaptation using arthropod herbivores has shown great potential. For instance, genetic studies show that under certain circumstances, adaptation to pesticides in field populations typically involves the selection of single genes with a large phenotypic effect (french-Constant et al., 2004; McKenzie et al., 1992). Unfortunately, the genetic architecture of polygenic controlled pesticide resistance and host plant adaptations of arthropod herbivores is far less explored, partly due to varying and **often low mapping resolution (my part of the story)** in traditional linkage studies (Alexandre et al., 2013; Jaquiery et al., 2012; Jones, 1998; Oppenheim et al., 2012). The recent availability of genomic resources for various plant feeding arthropod species

opens up a new and promising research avenue. Analysis of genomic divergence between host races within phytophagous insect species complexes has confirmed a polygenic basis of host plant adaptation. Within these regions of insect genomes, genome annotation identified candidate causative genes that code for enzymes that are predicted to be involved in recognition and detoxification pathways of plant compounds (Duvaux et al., 2015; Smadja et al., 2012; Soria-Carrasco et al., 2014; Yassin et al., 2016). Unfortunately, within these divergent genomic regions between host races, genes controlling host plant use cannot be readily disentangled from other differentiating genes that underpin traits that responded to other divergent environmental factors and contributed to reproductive isolation.

Project 2 Introduction

Diapause or developmental arrest is a type of animal dormancy that offers arthropods a mechanism of coping with unfavorable conditions. Diapause cues redirect the organism from its normal course of development and reproduction to a physiological state more suited for surviving low temperatures, declining food supply, high population densities, predators, and other stress factors. Two characteristics set diapause apart from quiescence (Hodek, 2002; Koštál, 2006). First, diapause happens in response to environmental stimuli that inform of upcoming unfavorable conditions, whereas quiescence results from an acclimation response to unfavorable conditions themselves. Second, and more importantly, diapause is a centralized, hormonally regulated program that dramatically reduces an organism's metabolism and arrests life cycle progression (e.g., metamorphosis, yolk production). In contrast, quiescence only affects certain aspects of an organism's physiology and is often regulated as an on/off switch. Diapause is known to be limited to one particular life stage and can occur during the embryonic, larval, pupal or adult stage. Besides developmental arrest, diapause induction triggers a whole set of adaptations including a decrease in metabolism (Harvey, 1962; MacRae, 2010), an increase in stress tolerance (King & MacRae, 2015), 3 changes in behavior (Veerman, 1985), and reduced energy use (Hahn & Denlinger, 2011). Nevertheless, diapause in insects and mites is incredibly diverse. Further elucidating the underlying mechanisms is crucial to grasp the bigger picture of arthropod diapause.