Stripe rust fungus *Puccinia striiformis* (*Ps*) is a great threat to cereal production in the world. Extensive efforts have been made to study the disease, including the biology of the pathogen and the epidemiology and management of the disease. However, the molecular mechanisms underlying the *Ps* pathogenicity to different cereals and virulence to different cultivars have not been well understood. Taking the advancement of whole-genome sequencing technologies, the objectives of this project were to i) identify and characterize avirulence genes (*Avr*) in *Ps* and ii) decipher the genomic basis of host adaptation of *Ps* at the forma specialis level. Firstly, we revealed putative pathogenicity mechanisms in *P. striiformis* f. sp. *tritici* (*Pst*, the causal agent of wheat stripe rust) by characterizing the secretome and comparing the *Pst* secretome with those of other rust fungi. We detected a large portion of species-specific secreted proteins that may have specific roles when the fungus interacts with the wheat host. Candidate avirulence genes were identified by incorporating virulence phenotypes in a correlation analysis by whole-genome sequencing fourteen *Pst* isolates with balanced virulence profiles. Secondly, we generated genomes with high continuity for *Pst* and *P. striiformis* f. sp. *hordei* (*Psh*, the causal agent of barley stripe rust) to study the genomics of the formae speciales. These genomes provide high-quality resources for deciphering the genomic basis of rapid evolution and host adaptation, identifying genes for avirulence and other important traits, and studying host-pathogen interactions. Thirdly, we compared the genomes of *Pst* and *Psh* to study the genomic basis of host adaptation. We found that host adaptation of *Ps* at the forma specialis level is a complex trait, involving not only virulence-related genes but also other genes. Gene loss, which might be driven by transposable element activities and adaptive to different hosts, provides genomic basis for host adaptation of different *Ps* formae speciales. Fourthly, we generated a segregating population by self-fertilizing a *Pst* isolate to genetically map *Avr* genes. A genetic map was constructed through whole-genome sequencing of the parental isolate and 117 progeny isolates. Quantitative trait loci analysis mapped six *Avr* genes to the genetic map. Through referring the molecular markers to the high quality *Pst* genome, we identified secreted protein genes that could be candidates for further cloning the avirulence genes. Our studies significantly advanced the understanding of the genomic basis for the rapid evolution of virulence in the *Ps*-cereal pathosystems.