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Genomics of the Asian rice gall midge and its interactions with rice

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Understanding virulence and manipulative strategies of gall formers will reveal new facets of plant defense and insect counter defense. Among the gall midges, the Asian rice gall midge (AGM) has emerged as a model for studies on plant–insect interactions. Data from several genomics, transcriptomics and metabolomics studies have revealed diverse strategies adopted by AGM to successfully invade the host while overcoming its defense. Adaptive skills of AGM transcend from its genomic and transcriptomic make-up. Information arising from studies on genetics, mitochondrial genome and miRNAs, amongst other parameters, highlights AGM's capacity to maneuver the host defense, reorient host metabolome and redirect its morphogenesis.

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Introduction

Biotic invaders constantly challenge plants and a significant number of these are microbes and herbivores. The dependence of these microbes and herbivores on their host plant is for food, shelter and protection from their respective predators. These associations are mostly detrimental to the host or in few rare instances both host and the intruder may co-survive. Plant defense against pathogenic microorganisms and herbivores is well elaborated to be a two-tier system [1•]. Innate immunity or basal defense is based on the plant receptors that recognize pathogen/microbe or herbivore associated molecular patterns (PAMPs/MAMPs/HAMPs), or damage associated molecular patterns (DAMPs). Receptors on plant cell surface recognize these patterns and mount pattern-triggered immunity (PTI) that is broad-spectrum and race non-specific. However, a range of invaders has evolved elicitors or effectors that neutralize PTI and successfully colonize the plant host. Second tier of defense of plants is mediated through several classes of resistance (R) genes that detect the specific effectors from a strain of pathogen and mount effector-triggered immunity (ETI) [1•]. This defense is rapid and race-specific. It is also generally accompanied with localized tissue necrosis at invading site called hypersensitive reaction (HR) and subsequent induction of systemic acquired resistance (SAR).

Plant gall formers are taxonomically diverse organisms [2] but all share the capacity to manipulate their host plants for their advantage. These induce galls of various plant organs for protection and nutrition. Plants, in turn, gain advantage of limiting the herbivore damage to these gall sites and often buy time for other natural forces to limit the populations of gall formers. During the interaction gall formers take over the process of morphogenesis, metabolism and differentiation of their hosts to create gall chambers for protection and feeding. Among the insect herbivores, gall formers act as 'con artists' (term adapted from Keen [3]) creeping under the carpet of plant defense. This is in contrast to other defoliators or stem borers causing extensive tissue damage and act as 'thugs' [4]. The sap-suckers, which directly suck host for food supply and water, occupy an intermediate status. Understandably, plant defense has evolved in diverse directions against these diverse guilds of herbivores.

The Asian rice gall midge (AGM), *Orseolia oryae* (Wood-Mason) (Diptera:Cecidomyiidae), causes significant yield loss across rice growing nations of Asia. In India, based on the economic damage caused by AGM, it is rated as the third most important pest after stem borers and planthoppers [5]. The pest population build up is strongly influenced by the prevailing weather and pest attack is generally restricted to wet rice growing season. Whenever the conditions are favorable for the pest, the crop damage can be severe and yield losses significant. The availability of resistance genes in rice germplasm accessions and ease of transferring resistance through classical breeding into elite cultivars have made host-plant resistance the main means to contain the pest [6]. However, prevalence and rapid evolution of virulent populations or biotypes of the

Table 1

Virulence spectra of the Asian Rice Gall midge biotypes in India[†]

Gall midge biotypes	Rice gall midge resistance (R) genes										
	Gm1	*Gm2*	*gm3*	*Gm4*	*Gm5*	*Gm6*	*Gm7*	*Gm8*	*Gm9*	*Gm10*	*Gm11*
GMB1[a]	avir	avir	avir	avir	avir	avir	avir	avir	avir	avir	avir
GMB2[b]	**vir**	avir	avir	avir	avir	avir	avir	avir	avir	avir	avir
GMB3[c]	avir	**vir**	avir	avir	avir	avir	avir	avir	avir	avir	avir
GMB4[d]	**vir**	**vir**	avir	avir	**vir**	**vir**	**vir**	avir	**vir**	**vir**	avir
GMB4M[e]	**vir**	**vir**	avir	avir	**vir**	**vir**	**vir**	avir	**vir**	**vir**	**vir**
GMB5[f]	avir	avir	avir	**vir**	avir	avir	avir	**vir**	avir	avir	avir
GMB6[g]	avir	**vir**	**vir**	**vir**	**vir**	**vir**	**vir**	**vir**	**vir**	**vir**	**vir**

Year of report and incidence locations (State) in India.
a: 1970–83, Hyderabad, Warangal, Maruteru (AP), Sambalpur (Orissa), Raipur (MP).
b: 1970–83, Cuttack, Bhubaneswar (Orissa), Mangalore (Karnataka), Goa, Sakoli (Maharashtra).
c: 1970–83, Ranchi (Bihar), Wangbal (Manipur).
d: 1985–89, Srikakulam*, Vishakhapatnam* (AP), Sakoli* (Maharashtra).
e: 2006, Laboratory selection from Warangal population.
f: 1994, Moncompu (Kerala).
g: 1995–98, Wangbal* (Manipur).
*A shift in biotype status reported during the period.
avir: avirulent against rice varieties harboring respective R gene.
vir: virulent against rice varieties harboring respective R gene.
[†] Adapted from Prasada Rao *et al.* [41] and Bentur *et al.* [23••].

pest capable of overcoming host resistance is a matter of great concern. Evolution of virulent biotypes resulted in cyclic outbreaks of the pest, referred to as boom-and-bust cycles, that have caused devastating pest damage at various time intervals.

Host defense and corresponding counter defense by the pest in this interaction is highly specialized in nature and mode of action. Gall midge maggots upon hatching wriggle down the space between leaf sheaths to feed on the apical meristem and thereby inducing nutritive tissue development through secretions of the salivary gland [7]. This leads to development of a gall chamber, around the maggot, which elongates to form a tubular leaf sheath gall called silver shoot. On pupation, pupa wriggles upward in the gall tube to rise above the standing water level in the field. The pupa drills an exit hole, with its cephalic horns, at the apex of the gall tube and eventually the metamorphosed adult flies out of this hole. This is the feature of a compatible interaction between the plant and the insect. In contrast, during an incompatible interaction, resistant genotypes of rice react strongly to kill the invading maggots within 24–48 hours of initiation of feeding [8•]. Feeding on resistant rice varieties leads to maggot mortality and is mostly accompanied by tissue necrosis (hypersensitive response; HR+) or sometimes without tissue necrosis (HR-) at the feeding site [9]. AGM shares several of its features with the Hessian fly [*Mayetiola destructor* (Say)]—an economically important Cecidomyiid pest of wheat. Expression of HR as part of host resistance is also known in wheat–Hessian fly interactions [10,11].

Genetics of host resistance against AGM

While resistance in rice against AGM was discovered several decades ago, it was only in the 1980s that genetics of inheritance of resistance to AGM was clearly established. Studies documented that a single major gene was responsible for resistance to AGM. Also, the presence of AGM biotypes was recorded prior to the above studies (see Table 1). Subsequently, a gene-for-gene relationship (Box 1) between plant *R* gene and AGM *avr* was

Box 1 Gene-for-gene hypothesis

Genetics of plant resistance and virulence of pathogen (the ability to infect host plant) was first demonstrated by Harold Flor [38]. His model proposed that for each resistance (*R*) gene in the host there is a corresponding gene in the pathogen responsible for pathogenicity. Resistance in plant is encoded by dominant '*R*' gene that recognizes the product of a dominant '*Avr*' gene in the pathogen that leads to initiation of plant surveillance system and induction of resistance. Susceptibility of plants and virulence in pathogens are recessive traits. Though, this hypothesis explained many aspects of multiple plant-pathogen interactions it could however, not explain all interactions and therefore further refined. Though Flor hypothesized the existence of a dominant *Avr* gene, it was not clear as to why it should exist in the pathogen when it was proving to be disadvantageous to the pathogen.

Hence a new model, based on Flor's model, was proposed that also accounted for 'adaptive' and 'basal' immune responses. The modified hypothesis states that '*R*' gene products provide adaptive immunity by perceiving specific effectors of the pathogen to activate effector-triggered immunity (ETI). On the other hand, 'basal' immune response uses transmembrane pattern recognition receptors (PRRs) to identify generic features of the pathogen and thereby inducing pattern-triggered immunity (PTI) [39••,40••]. This model satisfactorily explains the presence of numerous *R* genes (*i.e.*, with nucleotide binding site and leucine rich repeats) and evolution of multiple gene families coding for effectors in plants and pathogens, respectively.