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Major Insects of Wheat: Biology and Mitigation Strategies

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1. Introduction

Wheat is one of the major cereal crops with annual global production over 600 MT from about 200 M hectares (FAO 2012). The cultivation of wheat started about 10,000 years ago as part of the Neolithic revolution which state a transition from hunting and gathering of food to settle agriculture. Earlier cultivated forms of wheat were diploid (einkorn) and tetraploid (emmer) with known initial origin of the south-eastern part of Turkey (Dubcovsky and Dvorak, 2007). Subsequent evolutionary adaptation and continuous research produced hexaploid bread wheat that is currently widely adapted in about 95% area of world wheat. Globally, all crop production practices are being highly challenged by biotic and abiotic stresses. Biotic stresses especially insect pests and diseases causes devastating damage in terms of yield and quality. On average pests cause 20-37% yield losses worldwide which translating to approximately \$70 billion annually (Pimentel et al., 1997). In agro-ecosystems, herbivore insects are abundant and likely to colonise within same population and disperse from one crop field to another depending on the availability of plant tissues and feeding behaviour of insects. Quantitative feeding style of the herbivore insect on specific crop resulting significant damage to the crop during the entire life cycle which is believed specific insect as pest of that particular crop. Single pest may attack multiple crops within single growing season that make crop rotation and pest management more challenged. Wheat producing areas encounter with either sucking and piercing pests or plant tissue feeding pests. Regional pests also observed in wheat growing areas as major damaging pests worldwide. The breeding strategy againsts these insects/pests heavily rely on the inheritance of resistance mechanism in the crops under consideration. The insect resistance is mainly governed by three types of mechanisms/genes i.e., oligogenes; where resistance is conferred by single genes as in case of hessian fly in wheat, polygenes; where

several genes having small and additive effect bring about resistance against insects as in case of cereal leaf beetle in wheat and sometime cytoplasmic genes also confer resistance against insects/pest e.g., in maize and lettuce against European corn borer and root aphid respectively.

Large numbers of chemical formulations have been developed as pesticides to chemically control pest problems in different crops, however, control during all stages of insect life i.e. egg, larva, pupa and adult is almost impossible. It is therefore important to understand biology of insect pest simultaneously with the crop biology to understand when, where and what chemical should be used to control specific insect/pest more effectively. In addition, integrated pest management practices can also enhance control measures with minimum input and with no or less environmental hazards.

In this review, we have outlined major insects of wheat along with their biology and control strategies to minimize grain yield losses.

2. Wheat aphids

There are six species of aphids that damage cereals. These species include *Rhopalosiphum padi*, *Schizaphis graminum*, *R. maidis*, *Metopolophium dirhodum*, *Sitobion avenae* and *Diuraphis noxia*. Two of the species commonly known as Russian Wheat Aphid (*Diuraphis noxia*) and Bird Cherry-Oat Aphid (*Rhopalosiphum padi*) are considered notorious for their direct and indirect losses.

Russian Wheat Aphid (RWA) is known to be a sporadic insect causing significant yield losses by spreading out from its origin. The centre of origin for RWA is considered to be the central Asian mountains of Caucasus and Tian Shan. The species could now be found in South Africa, Western United States, Central and Southern Europe and Middle east (Berzonsky et al., 2003). The RWA was first reported in South Africa in 1978 (Walters 1984), in Mexico during 1980 (Gilchrist et al., 1984), in United States in 1986 and Canadian Prairie Provinces during 1988 (Morrison et al., 1988). RWA is present in almost all significant wheat producing areas of the world except Australia (Hughes and Maywald 1990). RWA attacks most of the cereals including wheat, barley, triticale, rye and oat. Alternate hosts for RWA are cool season (crested) and wheat grasses (*Agropyron spp.*). The economic impact of RWA includes direct and indirect losses that have been estimated to be \$893 million in Western United States during 1987 to 1993 (Morrison and Pears, 1998) whereas 37% yield losses in winter wheat have been reported in Canadian Prairies (Butts et al., 1997). Direct losses have also been assessed as an increased input cost due to insecticides and indirect losses include reduced yield due to RWA infestation.

2.1. Biology

Climatic conditions and temperature in particular, plays a significant role in population dynamics of the aphids. A warmer temperature can potentially accelerate the aphid's growth both in terms of number and size, yet, the extreme temperatures can possibly limit the survival and spread of the aphids. RWA is known to be present in its three different morphological

types—immature wingless females, mature wingless females and mature winged females. Winged mature females or *alates* spread the population and infection to the surrounding host plants whereas the wingless types or *apterous* cause damage by curling and sucking the young leaves. Heavily infested plants may typically look prostrated and/or stunted with yellow or whitish streaks on leaves. These streaks, basically, are formed due to the saliva injected by the RWA (Kazemi et al., 2001). The most obvious symptoms in heavy infestations can be reduced leaf area, loss in dry weight index, and poor chlorophyll concentration. Plant losses could be attributed mainly due to reduced photosynthates availability to plants and reduced photosynthetic activity due to RWA infestation (Millar et al., 1994; Burd and Elliott 1996). The photochemical activity of the plants have been reportedly inhibited by the RWA feeding from leaves and disruption in electron transport chain is likely to be the main cause of the reduced activity (Haile et al., 1999). Spikes can have bleached appearance with their awns tightly held in curled flag leaf. RWA can feed from main stem, flag leaf sheath and/or even developing kernels at flowering, resulting in shrivelled/empty grain or spike death (Peairs 1998a). In the event of sever attack; the wheat tiller can have purplish streaks. Approximately 1% to 0.67% yield losses per percentage of the infested tillers are reported at two tiller stage in Montana and Washington respectively (Archer et al., 1998). Yield losses can greatly vary due to infestation at different growth stages, duration of infestation and climatic conditions (wind patterns and temperature). A number of biotypes for RWA have been reported to be present throughout the cereal production areas of the world. These biotypes are classified due to significant genetic differences among them (Weng et al., 2007).

2.2. Strategies to mitigate RWA

A number of strategies have been deployed to mitigate RWA. Among these strategies, the host plant resistance has been the most effective and economic method to induce antixenosis, antibiosis and/or tolerance against RWA. RWA host plant resistance is well known to be qualitative in nature, and about nine resistance genes have been documented so far. These genes are: *Dn1*, *Dn2*, *dn3*, *Dn4*, *Dn5*, *Dn6*, *Dn7*, *Dn8*, and *Dn9* (Du Toit 1989, Nkongolo et al. 1991a, Saidi and Quick 1996, Marais and Du Toit 1993, Marais et al. 1994, Elsidaig and Zwer 1993). A gene-for-gene model supposedly induces resistance against RWA. In this mechanism the resistant gene produces a protein containing nucleotide binding site-leucine rich repeat (NBSLRR) domain (Feuillet et al. 2003, Botha et al. 2005 Jones). This NBSLRR domain first recognizes and then interacts with cognate Avr protein produced by the respective insect (Keen, 1990). Another domain (serine / threonine-protein kinases: STKs) has also been reported to be produced by *Dn* genes to confer resistance against RWA (Boyko et al. 2006). A general practice to introgress resistance in commercial cultivars is a combination of two resistant genes; however there are reports with single resistant gene or a combination of three genes conferring all three types of resistances in small grain cereals. *Dn4* reportedly have been most extensively used gene in breeding for resistant cultivar development (Quick et al., 2001). Rye and common progenitors of wheat (*T. Tauschii* or goatgrass) has served as resistance source for number of genes. *Dn7* gene was introduced in hexaploid wheat through translocation from the rye chromosome 1R to wheat chromosome 1B, and this gene has exhibited the resistance against all the known biotypes of RWA in North America and Africa (Lapitan et al. 2007, and Zaayman

et al. 2008). Pyramiding the resistance genes would be ideal to minimize the development of resistant biotype of aphids, yet, at present there is no such differential series of pure lines available to be used as breeding material. Marker assisted selection could be deployed potentially to confer a long term resistance against RWA. A number of alternate methods to control RWA has been suggested and practiced that include cultural, biological and chemical control methods. Cultural control strategies involve eradication of volunteer and alternate host plants is generally recommended. Another strategy is grazing the volunteer plants which significantly reduce the RWA infestation (Walker and Peairs 1998). Adjusting planting dates to de-synchronize the insect population dynamics and favourable environmental conditions of any particular area can also help to control RWA (Butts 1992). The enhanced fertigation of infested field, and biological control of RWA is also possible with 29 different species of insects and 6 fungus species (For further detail the readers are encouraged to read Hopper et al. (1998). Of the predator insects, 4 different species of wasps have become adopted to United States. Besides these cultural practices, chemical control method is also widely practiced with equivocal cost efficiency.

3. Bird cherry-oat aphid

Bird cherry oat aphids can saliently be characterised due to their high adaptive biological plasticity and transmission of viral diseases—*Barley yellow Dwarf* (BYD) virus in particular (Stern 1967). Bird cherry oat aphid is native to almost all over the world (Vickerman and Wratten 1979) and is abundantly found in Northern Europe (Wikteliu, 1982), North America, and New Zealand (Kieckhefer & Gustin, 1967; Kieckhefer, 1975). Bird cherry oat aphid can adopt a number of species as an alternate host including oat, wheat, cereal and other grasses and even on species of families *Juncaceae* and *Cyperaceae* (Rautapaa, 1970) with primary host being Bird Cherry (*Prunus padus* L.) and closely related tree species. Yield losses caused by Bird cherry oat aphid can vary greatly depending upon the time of infestation in relation to plant growth. It is one of the serious pests of in wheat growing areas of the world due to: a) its longest span of presence from early spring to late autumn (Dedryver 1978), b) ability to overwinter as an egg and/or parthenogenetic individuals and c) vectoring the *Barley yellow Dwarf* (BYD) virus.

3.1. Biology

Bird cherry oat aphid has the ability to multiply parthenogenically for one or more than one generation and subsequently undergo sexual reproduction. Bird cherry oat aphid alates fly to the primary host during autumn to mate and produce eggs. Change in environmental conditions stimulates the reproductive growth in Bird cherry oat aphid, to overwinter as eggs (Lees 1966), although it can survive in the regions of mild winter (Carter et al., 1980) and/or by descending down beneath the soil surface and feeding from the base of stalks (Wikteliu, 1987). An equivocal role of temperature in the survival of eggs has been reported in literature with a number of studies reporting the positive correlation between bird cherry oat aphid population and warm winters (Pierre 1987). However, certain clones adaptive to a site of cooler

temperatures have shown considerable ability to withstand winter temperatures (Griffiths and Wratten 1979). Therefore, it could be very tempting to conclude a strong positive correlation between temperature and increase in population of Bird Cherry Oat Aphid.

The feeding symptoms of bird cherry oat aphids are almost absent. Direct yield losses caused by bird cherry oat aphid are greatly dependent upon plant growth stage; as 24-65% losses can occur in case of infestation at seedling stage, and very low or non-significant yield losses from booting or later stages have been reported (Kieckhefer et al., 1995; Voss et al., 1997). Indirect yield losses are caused by transmitting viral diseases *e.g.* causing one of the important viral disease, *Barley yellow Dwarf* (BYD), in cereals. Sucking the sap and transmitting the BYD simultaneously can cause even more losses than alone (Riedell, 1999, 2007). The yield losses caused by sucking the plant sap can reduce the grain yield by 15%. The yield losses caused by BYD virus were estimated to be as high as 70% in the individual field of Idaho, with an average loss of 22% in different years depending upon the severity of infestation (Bishop and Sandvol 1984).

3.2. Strategies to mitigate bird cherry oat aphid

Number of studies have produced contrary results in the perspective of host plant resistance against bird cherry oat aphid. This might have happened due to very high biological plasticity of bird cherry oat aphids, presence of number of clones and related species in different geographical regions and different plant traits conferring resistance. Comprehensive and effective resistance against bird cherry oat aphid is typically possible when one has a detailed understanding of plant resistance mechanism to a particular growth stage of bird cherry oat aphid life cycle. In this scenario, numerous experiments have been designed to explore the most effective stage in the life cycle to limit the population of bird cherry oat aphid and its relationship to the extent of plant damage (Rauttapaa 1970; Markkula and Roukka 1972; Lowe 1980). Plant traits or mechanisms that induce nymphal mortality, elongated development at seedling stage and reduce birth rate at flowering are reportedly the most effective mechanisms to manage bird cherry oat aphid (Wikteliuss and Pettersson 1985). Plant traits that can prevent the bird cherry oat aphid inoculating the phloem and can reduce the proportional production of winged females, can limit the BYD dispersal to other plants (Gibson and Plumb 1977).

4. Greenbug

Schizaphis graminum Rondani or greenbug is a warm season perennial pest, causing substantial losses to cereal crops and wheat in particular. Greenbug was first reported on oat during early 20th century and also has colonized successfully in sorghum during 1960s (Harvey and Hackerott 1969). Greenbug is known to be originated from Virginia, North America (Hunter 1909), with a contradictory report that it might have originated from Italy (Michuad, 2010). Webster and Amosson (1995) reported 41% dryland and 93% irrigated area under wheat cultivation in Western US was infested with greenbug. A notorious periodic outbreak during 1976 in Oklahoma caused estimated losses exceeding \$80 million (Starks and Burton 1977). Large populations of greenbug shift onto sorghum during summer when wheat is harvested

and colonize in masses. In absence of sorghum, they can shift to wild grasses which can rarely accommodate larger populations (Anstead et al., 2003).

4.1. Biology

Greenbug is a light green, small size (about 3 mm in length), and sap sucking arthropod. It injects its stylet in sieve tubes, by secreting proteanacious saliva to facilitate penetration. Greenbug passively feed on sap upon a successful connection to the sieve tube (Miles 1999). Yellow to red lesions surrounded by a large chlorotic area can be readily identified on leaf surface, which turn necrotic with time. A seven-days feeding of 30 aphids per culm reportedly caused 40% grain weight losses on winter wheat (Kieckhefer and Kantack 1988). Greenbug is also reported to significantly reduce root length (Burton 1986) and hence limiting the plant capability to withstand drought stress. Greenbug has also been confirmed to vector *Barley yellow dwarf virus*. It can multiply asexually year round in a warm area as cold temperatures can significantly limit its survival. Occasional sexual reproduction, supposedly, has lead to the emergence of different biotypes of greenbug, which were eventually identified due to their differential response to resistant cultivars and pesticides (Ullah and Peters 1996; Rider and Wilde 1998). Wood (1961), identified greenbug damage on resistant line DS 28A, and described it as a different biotype which was named as Biotype B. The biotype to which DS 28A was resistant was called Biotype A. Similarly, biotype C was discovered on sorghum due to greenbug substantial damage on cultivar named 'Piper' resistant to biotype B. So far, eleven biotypes has been identified and named biotype A to K. Most prevalent biotypes in Oklahoma and Kansas, which is the area of its most economic threat, are I, E and K (Kindler et al., 2001) whereas biotype G is reported to be more prevalent on non-cultivated grasses in Southern Great Plains (Anstead et al., 2001).

4.2. Strategies to mitigate greenbug

A regular detection of new greenbug biotypes has more or less necessitated the use of two strategies to mitigate its severe outbreaks: the chemical control method and host plant resistance. A number of chemicals have been used against greenbug including dimethoate, parathion, methyl parathion, chlorpyrifos, imidacloprid and malathion with varying doses depending upon the threshold on a specific growth stage. Extensive use of chemicals had not only induced insecticide resistance in the greenbug, but also has environmental concerns in addition to the extra cost. Therefore, the researchers continuously looked for host plant resistance against the greenbug. Qualitative inheritance of resistance conferred by both dominant and recessive genes is well documented in literature with gene symbols as: *gb1*, *Gb2*, *Gb3*, *Gb4*, *Gb5*, and *Gb6*. *Gb6* is the most potent gene conferring resistance against biotypes B, C, E, G and I and was recovered from a wheat-rye translocation germplasm by Porter et al., 1994. Theoretically, gene pyramiding could possibly ensure a broad spectrum and long-lasting resistance against greenbug. Porter et al., (2000), designed a study to verify the resistance conferred by more than one gene, and reported no additional protection conferred by more than one gene compared to their single counterpart; and suggested for a sequential release of resistant gene with complete monitoring of greenbug biotypes prevailing in a particular area.

Development of molecular markers flanking these resistant genes is underway to arm the modern molecular techniques to exploit the resistance potential at its maximum.

5. Cereal leaf beetle

Cereal leaf beetle is an insect of cereal or small grain grasses. The particular origin of the insect is still unknown, however, it is considered to be a native insect of Europe and Asia. It is a serious insect in Eastern and South-Eastern Europe including Hungary, Yugoslavia, Poland, and Rumania. It is now considered to be present all over the Europe. In Asia, it is reported to be present in Pakistan, India and Iran. In America, it was probably introduced in early 1960s when it was first identified as a serious insect in Michigan, in 1962. It is now present in most of the states, and in Canadian Prairies—Alberta, Saskatchewan, and Manitoba (Kher et al., 2011). Cereal leaf beetle feeds on oat, wheat, barley in particular and on many other cultivated and non-cultivated grasses (Wilson and Shade 1966). The economic losses caused by cereal leaf beetle greatly vary among the crops, regions and timing and level of infestation. Buntin et al., (2004) reported a maximum loss of 40%; whereas Herbert et al., (2007) reported about 15% wheat yield losses in Virginia due to cereal leaf beetle.

5.1. Biology

Cereal leaf beetle adult is about 5mm long, bluish black head and elytra, and burgundy red thorax and legs. Adult feeding usually does not cause economic losses to the crops. However, the larvae, which is also about 5mm in length and shiny black in colour, feeds on photosynthetic tissues of the leaf, leaving behind the leaf skeleton only (Buntin et al. 2004). This results in significant loss of photosynthetic activity of the plant, giving it a frosted look. Hence, the plant fails to produce expected yield and quality (Merrit and Apple 1966, Grant and Patrick 1993). Cereal leaf beetle generally has one generation per year, however a small second generation is also reported in Virginia (McPherson 1983b). A typical cereal leaf beetle life cycle span is about 46 days, but can be as short as 10 days and as long as 90 days depending upon the environmental conditions and temperature (Guppy and Harcourt 1978, Metcalf and Metcalf 1993). Highest yield losses can be anticipated by the cereal leaf beetle larvae feeding the flag leaf. The losses vary greatly in different regions *e.g.* in Poland the yield losses reported are 3-8% (Ulrich et al., 2004), and could be as high as 95% in The Netherlands (Daamen and Stol 1993) and on different grain crops *e.g.* wheat yield losses in North America can reach up to 55% (Royce 2000), whereas these losses can be 75% in oat and barley (Webster and Smith 1979).

5.2. Strategies to mitigate cereal leaf beetle

Chemical control has long been practiced to control cereal leaf beetle, even before its identification and recognition as a threatening pest. Pesticides have both been applied as granules to soil (Carbofuran) and as a foliar spray (Endosulfon, methomyl, methyl parathion, etc). Non-selective insecticides have indiscreetly killed the natural enemies and the parasitic species. Biological control has also been an effective method to mitigate cereal leaf beetle. A number of species

parasitic to larvae and eggs have been reported as *T. julis*, *Diaparsis carinifer*(Thomson) and *Lemophagus curtus*(Townes) (Hymenoptera: Ichneumonidae), *Anaphes flavipes*(Foerster) (Hymenoptera: Mymaridae)(LeSage et al., 2007, Haynes and Gage 1981). Host plant resistance against cereal leaf beetle has been most effective in wheat, mainly due to trichomes (pubescence) produced on leaf surface. A positive correlation between the resistance and trichome length and intensity is reported (Wellso 1973). Non-preferential behaviour for oviposition and first larval instar feeding deterrence are the mechanisms conferring resistance. Oat and Barley have shown lesser resistance against cereal leaf beetle relative to wheat (Hahn 1968). Host plant resistance could not be exploited to its maximum due to variety of reasons: very few resistance sources, lesser adaptation and a negative correlation between resistance and yield are some of them (Kostov 2001).

6. Wheat stem sawfly

The stem sawfly of wheat, *Cephuscinctus* Norton (Hymenoptera: Cephidae), is a phytophagous insect of wheat and other cereal crops including barley, rye and triticale. It is of serious concern in different parts of world especially in northern hemisphere (Shanower and Hoelmer 2001). The *C. cinctus* is considered to be a single specie; however differences in virulence have been detected due to genetic variability. Its larvae under different environmental conditions such as similar to North Dakota and Montana differed in duration of post diapause development that might be due to climatic variability. It is one of the major pests of spring wheat in USA. The cropping system like summer fallowing and strip cropping is the main reason to make sawfly as a potential pest causing significant losses. The historical background revealed that *C. cinctus* is indigenous to North America and it exhibits a relationship with Siberian species (Ivie and Zinovjev 1996). Its spread in North America could have occurred due to transport of straw or crown from plants containing live larvae (Ivie 2001). The case of severe infestation of wheat stem sawfly (WSS) was recorded in 1922 in Canada which was due to absence of natural enemies of the sawfly that could result a severe threat to food security. The outbreaks of WSS were short lived because host plants were immediately eliminated due to rust epidemics but the continuous development of rust resistant genotypes lead to progression development of WSS population. Strip farming to control soil erosion is another reason for dissemination of WSS from one field to another.

The biology of WSS revealed that adults of both sexes are weak fliers and cannot fly long distances. The adult feed on exudate moisture and on nectar while resting on plant stem with head in downward position and legs aligned with its body. The life cycle of WSS is synchronised with the phenology of host plant and all growth and development occurs within the host plant except the last stage. The timing of its emergence is greatly influenced by temperature and adults become active during warm season when wind speed is minimum. The cloudy, windy and rainy conditions have an inverse relationship with the activity of WSS. Adult males become visible first as compared to female to ensure mating of females so that most of eggs oviposited in the early flight will be fertilized whereas eggs at the end of flight remained unfertilized. The haploid male will be produced from unfertilized eggs whereas fertilized eggs lead to the development of diploid female. The adults are sexually mature and ready for copulation and oviposition. The female lay 30-50 eggs during her entire life. The egg stage of

WSS consists of 5-7 days in length while larval development last for one month. On completion of developmental phase, the larvae start feeding and filling the stem with excreted plant tissue called frass that ultimately lead to the stem splitting. The larvae then descend down to the base of stem creating a V shape furrow that results in complete cutting of the stem. The larva constructs a thin cellophane structure to get protection. This sealed cocoon help larva to remain protected from environmental hazards and predation.

The protected larvae can survive for months and it passes most of its winter in the crown root since temperature remained higher as compared to ambient temperature. The rate of mortality of larvae becomes high if it is exposed to low temperature. However, pupation occurs if there is rise in temperature and weather is dry. The pupal development depends on climatic conditions like drop in temperature. The pupa is white and as pupal development proceeds, wings start emerging/developing followed by pigmentation in the body that results in a mature adult. The insect remained in soil during winter and under favourable environmental conditions, it emerges out and ready for flight. The distribution of WSS is spatial and temporal. As soon as they emerge from stubbles, they start migrating to the nearby wheat plants. The infestation might be severe if females oviposit first within field margin that often results in the uniform distribution of eggs as the flight progress (Nansen et al. 2005). The release of signalling compounds from plants attracts WSS that often lead to severe infestation. However female is unable to differentiate between damaged and healthier plants.

The mature WSS cause little injury but boring action of larvae is very destructive and is a major cause of severe losses. The declined in photosynthetic activities due to destruction of parenchyma and vascular tissues is one of the main damage caused by larvae. The stem will be hollow in a week as larvae feeds up and down.

The mitigation strategies might include cultural control (strip planting and alternative planting strategies), early forecasting system, simulation modelling for long term planning, biological control, chemical control and development of host plant resistance (gene deployment, resistant cultivar development and cultivar blends). The pheromone monitoring and host-plant semiochemicals techniques could be used as an effective strategy to minimize damage of WSS. However, the future research needs to involve multi scale collaborative efforts among different disciplines to develop a holistic approach to control any outbreak of WSS. Cultural methods are critical to control WSS, therefore, it's important to encourage producers to adopt such procedures which can minimize the WSS population and increase beneficial insects. The use of resistant genotypes having solid stem can contribute to minimize the damage to a greater extent. The use of cultivars blends, IPM (integrated pest management) and ICM (integrated crop management) could be considered as management tools for the control of WSS.

7. Wheat midge

The major pest of spring wheat in most part of world is Wheat midge (WM) which can cause 30% reduction in wheat yield resulting in an economic loss of 30 million dollar. It is also called orange wheat blossom midge and it is the periodic pest of wheat crop in the northern hemi-

sphere and cause significant damage when climatic conditions favours its growth. It's the main pest of China, Europe and North America where winter and spring wheat is being cultivated. WM is serious pest in Canada (Lamb et al. 2000) that has resulted in widespread use of insecticides. The origin of WM was first detected during 1741 in England. The dispersal of WM mainly take place from Europe to North America and then to Asia. Its dissemination is through larvae which remain in the spikes and then stored in the seed after harvesting with combine harvester. WM hibernate in the soil and during spring season it multiply and pupate. The hatching of cocoon depends on soil temperature and moisture that result in higher numbers. At the ear emergence, the adult WM mates and females then move to wheat crop where it starts laying eggs. The flight of females takes place at evening and if wheat crop is absent laying of eggs take place at barley or weed grasses. The hatching of larvae from eggs takes place after a week and produces alpha-amylase enzyme to release sugars from the grain. The larvae then drop to the soil after feeding for few weeks and made a cocoon around itself. The generation of WM completes in one year and it passes winters in soil as larvae. The high temperature terminates the diapause of larvae and it comes out from cocoons and spends some time at soil surface (Doane and Olfert, 2008). The damage to the crop starts at grain development stages causing shrivelling and crack which ultimately reduces yield and quality of crop.

The development of WM is highly dependent on soil moisture and temperature. The termination of larval diapause occurred in phases: firstly, larvae required cool temperature for three months; secondly, larvae enter into moisture sensitive phase which remained for 5-6 weeks. However, if soil is dry it remained in diapause for one year while on the other hand if moisture is sufficient, larvae's terminated diapause, pupated and emerged as adults within five weeks. The adult's stage is last stage of WM and basically it is small orange fly with length of 2-3mm. It has two large black eyes with size equivalent to mosquito and has three pairs of legs which are larger in size. The wings are oval shaped and transparent. The adults will prefer to remain in crop canopy where the environment is humid and when conditions become favourable the female become active and comes at the top of canopy starting laying eggs on newly emerged spike. Therefore, WM larvae compete directly with humans for the grain and destroy the grain by causing shrivelling. The infestation of WM can be seen on all parts of spike and feeding of larvae is greater on small seeds as compared to larger one (Lamb et al. 2000). The intensity of damage could be determined from the feeding place of larvae. If it feeds closer to the grain embryo, the attack will be very severe. Usually, the seed is attacked by larger number of larvae but if four or more is present attack will be of serious nature. The body size of larvae might be affected significantly if they are present on one seed because of competitions between them. The damage caused by larvae to the wheat seed can be calculated by dividing mass lost by the seed to the mass gain by the larvae (Lamb et al. 2000) called as efficiency index of WM larvae. The activity of WM larvae decreases when wheat seeds have lost 75% of their mass. WM feeding has resulted in maximum impact among feeding insects that feed on crops belong to Poaceae family (Gavloski and Lamb 2000). The damage of WM to crop adversely affects the agronomic performance like resistance to sprouting, yield, germination and seedlings early vigor. It also affects grain quality resulting to change in seed protein levels and decline in the drought resistance patterns of crop. The quality might be further deteriorated due to carrying of harmful microorganism with WM and attack by the semolina after the WM.

The WM could be controlled by inspecting field at heading stage and by the application of insecticide to minimize the damage. If infestation of WM is identified at early stage by regular monitoring at heading and flowering then WM attack could be minimized to a greater extent. The use of wheat genotypes that are resistant to WM is another way to control its attack. However, it has been recommended that the best control measure is to use predators that can eat the WM larvae so that it is unable to multiply further. The examples of predators include polyphagous which might control WM at the different vulnerable stages. The concept of host plant resistance is another way to control WM by developing such genotypes that can resist the development of WM. The host plant resistance includes resistance mechanism and genetics in which genotypes produce antitoxic substances lead to minimize WM infestation. These genotypes changes oviposition in the field and reduce the egg densities in the field resulting in lower numbers of WM. The research studies has depicted that these lines could control WM larvae from 58 to 100% (Lamb et al. 2000).The development of antibiosis is another way to control WM and resistance in spring wheat is linked with the production of phenolic compounds from seeds which might destroy the WM (Ding et al. 2000).In the same way, use of selection protocols and field methods like screening of wheat genotypes and cultural practices are the important ways to control WM. The modifications in the oviposition sites can also control WM to a considerable degree. Breeding wheat for resistance to insects is an easiest and cheapest mean to control WM

8. Hessian fly

The *Mayetiola destructor* called Hessian fly (HF) belongs to the species of fly and is destructive pest of cereal crops including wheat, barley and rye. It is native of Asia and transported to Europe and North America through straw. HF has two generations in a year but it can go to five. The dark coloured female lays 250-300 reddish eggs on plants during spring season. After 3-10 days larvae hatch from the eggs and they cannot survive in the open air therefore they move to the base of leaf sheath which is preferred feeding site. The larvae (maggots) crawl down to the crown of the plant during fall season. The meristemic activities in node causes wheat stem to elongate and maggots are usually found at the top of leaf nodes. The HF infestation will be found at the top because female prefer to lay eggs on new leaves which comes out from nodes. The maggots are reddish brown and as they feed and grow it changes colour become white and greenish white. The feeding of maggots is on stem and after scraping the stem it start sucking up the sap which comes out from the wound. The larvae remain feeding for fourteen to thirty days. The flaxseed is the shiny, protective case where maggot spent its last stage and it is built from insect skin and has resemblance with the seed of flax plant. The attack of larvae is so severe that plants are unable to bear grain. The HF comes out from the flaxseed structure when climatic conditions become favourable. The adults come out and start new generations and if climatic conditions are extreme (too hot or cold) it remained inside the flaxseed coat until climatic conditions become favourable. The presence of HF and there maggots at the same time is very uncommon particularly during heavy infestations. The complete life cycle from egg to adults requires 35 days if temperature is favourable.

The damage caused by HF maggots is mainly on vegetative growth which might reduce the activity of photosynthesizing machinery resulting to stunting growth. The maggots during feeding also inject toxic substances resulting to inhibition of plant growth. These toxins act as inhibitors to the plants and overall hormonal action of plants is disturbed resulting to poor growth. However, damage could be severe if timing and degree of infestation is perfectly matched with crop phenological stages. The single maggots can cause significant damage to wheat plants because toxins released during feeding interfere with wheat crop growth. Meanwhile, if the attack of larvae is at the single leaf stage, then it will be killed immediately. The attack at later stages causes destruction of first tillers and growth of the crop is delayed. The weakening and shortening of stems occur due to larval attack and it might break from the first or second node before the harvest of crop, resulting to head loss. The reduction in yield and quality of crop will be observed with severe mechanical losses to stem and head during heavy infestation.

The use of preventive rather than chemical control methods can control the population dynamics of the insect. These methods include biological and cultural approaches which are reliable and feasible for wheat crop growers. The growing of resistance cultivars by the use of biotechnology is the best way to control the damage caused by HF. The tissues of plants contain several types of carbohydrate binding proteins called lectins. These lectins have potential to build resistance in wheat against insects. The identification of genes which might produce this type of lectins will be the best way to control HF. The genes include Hfr-2 called as HF destructor which is expressed in the leaf sheaths of the resistance genotypes (Puthoff et al., 2005). Similarly, mannose binding lectins which serve as storage protein accumulate in the phloem sap and might act against HF. These lectins have anti-insect properties because they accumulate in the midgut of the insect and kill them immediately. The production of Wci-1 mRNAs and Hfr-1 in response to the attack of HF larvae is another defensive mechanism which is present in resistant varieties of wheat. The Hfr-1 gene is called a defender gene against HF and it can control crop from severe attack (Subramanyam et al., 2006). Meanwhile, there are a number of different sources of incorporation of resistance traits into wheat which might build a defensive mechanism in crop against HF. Antibiosis is the main mechanism of resistance associated with these genes and is expressed as the death of first larvae. The biochemical nature of antibiosis in wheat includes development of silica in sheaths and production of free amino acids, organic acids and sugars in plants. The development of resistance genotypes in wheat breeding programmes might improve the durability of resistance in wheat genotypes against HF. The breeding programmes include use of resistant genes or combination of different levels of resistance genes that might respond differentially to abiotic and biotic stresses. The use of genes which have potential to control HF is the best way to control population dynamics of HF larvae in wheat crop. The knowledge of molecular markers and QTL mapping associated with resistance genes incorporation in wheat is another landmark which might be used to control HF. The use of simulation genetic models might be used to check the development of single gene resistance in crop and it is an adequate way to control the HF.

The HF population dynamics could be controlled by modification in tillage practices and change in the cropping pattern which can destroy the life cycle of pest. The delayed planting is another way to control the HF. There are large numbers of different parasitoids which attack the HF and might be used to control its attack on crop. The use of chemicals to control HF is

not recommended. The best way to control HF is development of resistant genotypes which work like systemic insecticides. Similarly production of novel jacalin like lectin gene from wheat responds significantly to the infestation of HF larvae and could be used effectively in future breeding programmes. The wheat genotypes having higher levels of Hfr-1 at the larval feeding sites and only small amount of lectin at these sites will control the larvae.

Insect	Resistant Gene	Primer Sequences	Gene Origin	Gene Location	Affiliation
GreenBug	gb1	Not mapped	T. Turgidum/T. Durum		
	Gb2	ATATCTCAACCAACTTCACAAAGTC CATTGTTTAAAAAGAGGGGATATG	S. Cereale		Lu et al., 2010
	Gb3	5'- AGC GAG GAG GAT GCA TCT TAT T -3' 5'- GAC ATA CAC ATG ATG GAC ACG G-3'	T. Tauschii	7DL	Weng et al., 2002.
	Gb4	Not mapped	T. Tauschii		
	Gb5	Not mapped	T. Speltoides		
	Gb2/Gb6	TATACACCAACAAGTAGCGACAATA AAACAAACCTTCAGTATCTTCTCAC	S. Cereale		Lu et al., 2010
			5'-CTCACCGTCC-3'		Kharrat et al., 2012
			5'- GAGCCCTCCA-3'		Kharrat et al., 2012
		RAPD-PCR based Single Decamers	5'-TCACGTCCAC-3'		Kharrat et al., 2012
			5'- GGCTCATGTG-3'		Kharrat et al., 2012
		5'- AGTC-GTCCCC-3'		Kharrat et al., 2012	
Hessian Fly	H9	5'- GGA AGC GCG TCA GCA CTA GGC AAC -3' 5'- GGC TTC TAG GTG CTG CGG CTT TTG TC -3'	T. Aestivum	1AS	Kong et al., 2005
	H13	5'- CAA ATG CTA ATC CCC GCC -3' 5'- TGT AAA CAA GGT CGC AGG TG -3'	T. Aestivum	6D	Liu et al., 2005
	H25	5'- CTG CCT TCT CCA TGG TTT GT -3' 5'- AAT GGC CAA AGG TTA TGA AGG -3'	S. Cereale	4A	Sebesta et al., 1997
	H26/H32	5'- CCT AAC TGA GGT CCC ACC AA -3' 5'- GCA AAG GAC TTG ATG CCT GT -3'	T. Tauschii		Yu et al., 2010
	H31	5'- TCC TAC CTC CAT TCC CCT TT -3' 5'- TCA AAA TGA ATC GGA AGG GT -3'	T. Turgidum	5BS	Williams et al., 2010
Stem Saw Fly	Hdic	5'- GAC AGC ACC TTG CCC TTT G -3' 5'- CAT CGG CAA CAT GCT CAT C -3'	T. turgidum ssp. dicoccum	1AS	Liu et al., 2005
	<i>Qss.msub-3BL</i>	5'- GCA ATC TTT TTT CTG ACC ACG -3' 5'- ACG AGG CAA GAA CAC ACA TG -3'		3BL	Cook et al.,
	<i>Qss.msub-3BL</i>	5' GCAATCTTTTTTCTGACCACG 3' 5' ATGTGCATGTCGGACGC 3'	Durum wheat	3BL	

Insect	Resistant Gene	Primer Sequences	Gene Origin	Gene Location	Affiliation
	<i>Qss.msub-3BL</i>	5' GTTGTCCTATGAGAAGGAACG 3' 5' TTCTGCTGCTGTTTTCATTTAC 3'		3BL	
	Dn1	5' TCTGTAGGCTCTCTCCGACTG 3' 5' ACCTGATCAGATCCCACTCG 3'	T. Aestivum		Peng et al., 2007
	Dn2	5'- GAT CAA GAC TTT TGT ATC TCT C -3' 5'- GAT GTC CAA CAG TTA GCT TA -3'	T. Aestivum	7D/1B	Peng et al., 2007
	dn3	not mapped			
RWA	Dn4	5'-CTG TTC TTG CGT GGC ATT AA-3' 5'-AAT AAG GAC ACA ATT GGG ATG G-3'	T. Aestivum	1DS	Peng et al., 2007
	Dn5	5' TCTGTAGGCTCTCTCCGACTG 3' 5' ACCTGATCAGATCCCACTCG 3'	T. Aestivum	7DS	Peng et al., 2007
	Dn6	5' TCTGTAGGCTCTCTCCGACTG 3' 5' ACCTGATCAGATCCCACTCG 3'	T. Aestivum	7DS	Peng et al., 2007
	Dn7	Xscb241 RFLP marker		1RS	
	Dn8	5' TTCCTACTGTAAGGGCGTT 3' 5' CAGCCTTAGCCTTGCG 3'	T. Aestivum	7DS	Peng et al., 2007

Table 1. Resistant genes for different insects along with their primer sequences, origin, and location

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