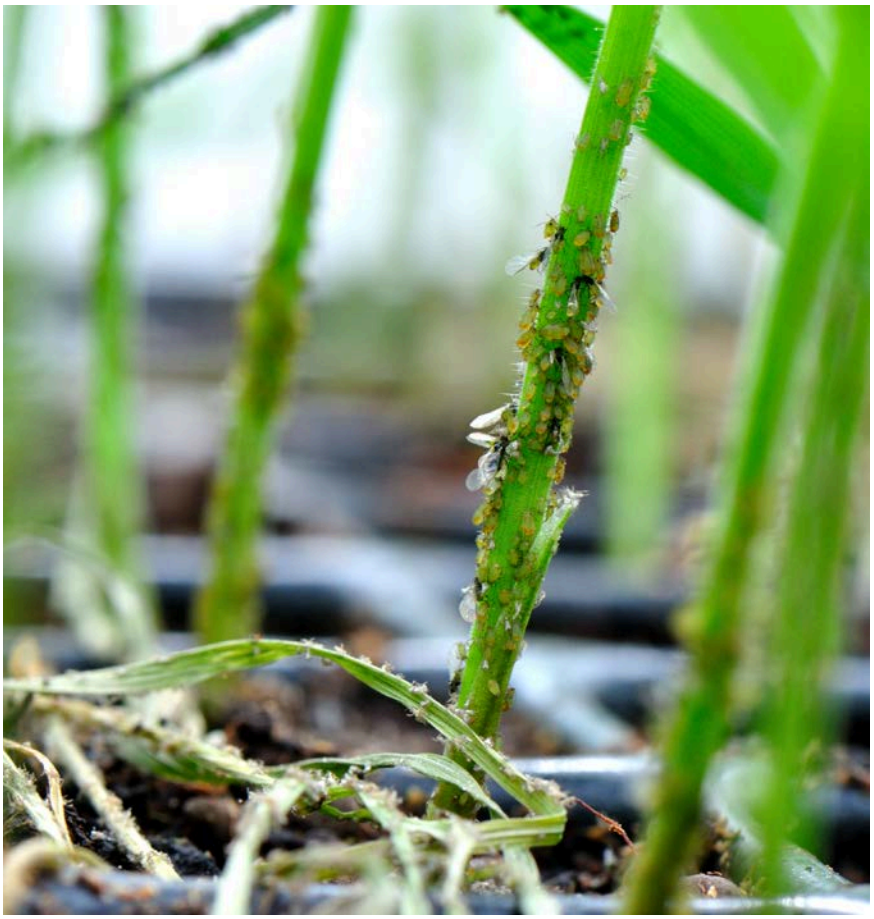
RESISTANCE TO APHIDS IN WHEAT

FROM A PLANT BREEDING PERSPECTIVE



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Introductory Paper at the Faculty of Landscape Planning, Horticulture and Agricultural Science 2012:6
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ABSTRACT

Wheat is one of the main staple foods in the world. Among the many constraints there are for wheat production, aphids cause severe damage by their feeding, and by virus transmission. The current document summarizes the available information regarding wheat resistance to aphids. The text is divided into four sections. The first section "**Plant resistance to insects: from a historical perspective**" serves as an introduction, where some important factors shaping Plant Resistance to insects as a discipline are presented. The section "**Categories of resistance and plant defenses to aphids**", presents the concepts of Plant Resistance to insects, with wheat-aphid examples. The methods to assess the categories of resistance are briefly discussed in this section as well. "**Genetic resources for resistance to aphids in wheat**", the third part of the document, is focused on the wheat relatives in which resistance to biotic stresses and to aphids in particular can be found. The aim of this section is to describe the resistance donors in relation to each of the main aphid species that attack wheat. Finally, some aspects on how to improve wheat for resistance to aphids are discussed in the fourth section, "**Considerations for wheat breeding**".

FOREWORD

Wheat is one of the main staple foods in the world; according to FAO (2010), more than 200 million of hectares are harvested annually and around 650 million tonnes are produced. This crop and some other cereals have accompanied human civilization since agriculture started. However, the continuing urbanization and population growth, the gradual intensification of agriculture, the reduction of genetic diversity of crops by means of the strong selection pressure put on them by plant breeding, vast cropping areas dedicated to monoculture, and some other factors, have generated several constraints not only to wheat cropping but for food production in general. Nowadays we are witnessing the consequences of the environmental manipulation that human beings have created on Earth, including many historical-social problems such as hunger and malnutrition, among others. Therefore, collaborative actions to ameliorate this situation must be taken. An important contribution is by increasing knowledge in science and discovering plant germplasm that can be used to increase food production.

Among the many constraints for wheat production, aphids cause severe damage by their feeding, up to 45 % of yield loss, and by virus transmission, up to 80 % when combined with aphid feeding. Incorporation of resistance by means of plant breeding is a strong tool to control aphids. However, breeding for resistance and deployment of aphid resistant wheat cultivars have mainly been restricted to two aphid species, *Schizaphis graminum* and *Diuraphis noxia*, even though the damages caused by other species such as *Rhopalosiphum padi* and *Sitobion avenae* are well documented.

For the reasons outlined above, the Ph. D. project entitled "Enhancing and deploying resistance to multiple aphid species in bread wheat" was started in fall 2010. It is conducted under the supervision of Prof. Inger Åhman in the Department of Plant Plant Breeding and Biotechnology at the Swedish University of Agricultural Sciences (SLU), in collaboration with the International Maize and Wheat Improvement Center (CIMMYT) in Mexico, and financed by the Monsanto's Beachell-Borlaug International Scholar Program. There are four major objectives in the project: 1) to find resistance sources to multiple aphids (*S. graminum, R. pad* and *S. avenae*) for their incorporation in wheat; 2) Unraveling the genetic basis of *S. graminum* and *R. padi* resistance in a `Synthetic Hexaploid Wheat' mapping population; 3) determining the utility of resistance genes in a yield loss assessment trial and 4) study tolerance as mechanism of resistance and induced resistance to aphids in wheat.

The current document is an effort to compile, in a summarized way, the available information regarding wheat resistance to aphids, ranging from the general categories of resistance, to the genetic resources and genes of resistance that have been found. The text is divided into four major sections. The first section "**Plant resistance to insects: from a historical perspective**" serves as an introduction, where some important factors shaping Plant Resistance to insects as a discipline are presented. "**Categories of resistance and plant defenses to aphids**", is to present the concepts of Plant Resistance to insects, with wheat-aphid examples. The methods to assess the categories of resistance are briefly discussed in this section as well. In "**Genetic resources for resistance to aphids in wheat**", the third part of the document is focused on the wheat relatives in which resistance to biotic stresses and to aphids in particular can be found. The aim of this section is to describe the resistance donors in relation to each of the main aphid species that attack wheat. Finally, some aspects on how to improve wheat for resistance to aphids are discussed in the fourth section, "**Considerations for wheat breeding**".

CONTENTS

1. PLANT RESISTANCE TO INSECTS: FROM A HISTORICAL PERSPECTIVE

Agriculture started about 10,000 years ago, not only in one place but independently in various regions of Asia, Africa and America where certain wild plants were domesticated, with time into locally adapted landraces. During this development, traditional farmers around the world improved their land races by plant selection from one generation to the next, thereby improving many plant traits, among them plant resistance (PR) to important pests and diseases. Probably, the earliest documented report of PR was published in the XIX century; Havens (1801) started simple experiments on the Hessian fly biology inspired by a farmer observation and reported that the insect damage differed between wheat cultivars; although this phenomenon was observed it was not yet called PR.

In the third decade of the 19th century, Lindley (1831) published a guide for garden management in which the apple variety Winter Majetin was described as defiant to the woolly apple aphid, at that time called white mealy insect.

Another classic example quoted by Howard (1930) and Brader (1987) is the grape *Phylloxera*, which devastated around 1.2 million hectares during the Franco-Prussian war in 1870. The first damaged fields were found in 1863 and 14 years later most of the grape wine fields had disappeared in Europe. It is believed that this insect was introduced to Europe in the second half of the 19th century, when botanists from England collected wild *Vitis* species in North America that they brought back to their home continent. The problem with this devastating pest was solved by grafting European *Vitis vinifera* varieties on aphid resistant American native *Vitis* species rootstocks, a solution that is still in place to fight this pest.

From all such prior observations made by farmers and scientists, PR became an important subject in applied entomology (Ortman & Peters, 1980). A review on the status of the knowledge of PR to insects was published by Snelling (1941). He listed 567 references of which only 37 were published between 1792 and 1920. Thus, 530 reports were published in the next 20 years, showing a great increase in resistance research. However, the first comprehensive review "Plant resistance to insects" discussing the principles of PR to insects was published in 1951, by Reginald H. Painter. Earlier, Painter (1941) pointed out that insect resistance is based on three mechanisms and that even though PR could be an important tool in crop production, it is not a cure-all method since it only helps in the control of specific insects in particular crops.

Despite the growing interest in resistance to insects during the 19th and 20th centuries, importance of PR as an insect control method was surpassed when new findings on the chemical control to fight insects arose during the post-World War II period. Chemical control showed spectacular results in insect combat and research strategies shifted rapidly from the insect-host interactions point of view to this new approach, which later created serious problems in the environment and caused insect resistance to insecticides as well. Insecticides such as DDT became very popular among farmers and also for urban use against pests. However, in the book "*The Silent Spring"* Carson (1962) alerted about the detrimental effects of pesticides in the environment and pointed out that pesticides should be used responsibly. This book was important for the start of the modern environmental movement.

Currently, chemical control is the most widely used method to fight biotic constraints for food production. However, genetic plant resistance can provide a more effective control in terms of costs and ease of handling, and with less or no negative ecological impacts. Therefore,

incorporation of durable and broad spectrum resistance to diseases and pests is one of the main objectives in plant breeding programs around the world.

Van der Plank (1963) developed the theoretical concepts of PR from the perspective of phytopathology, and classified it as vertical (race-specific) and horizontal (race-nonspecific). This conceptual frame work has been used to classify plant resistance mainly to diseases but it is also useful for insects. Race-specific resistance is based on a 'gene for gene interaction' between pathogens and plants. So-called major genes in plants, with large phenotypic effects, are involved in this kind of resistance. Complete resistance is usually but not always exhibited in this interaction and commonly plants respond to attack by hypersensitive reactions. Another major characteristic of this resistance is the regular boom-bust resistance/susceptibility cycles due to strong selection put on the pathogens. The deployment of cultivars with major resistance genes rapidly results in occurrence of other races of the pathogen that can overcome the resistance (McDonald & Linde, 2002).

The non-specific resistance is commonly more genetically complex and normally characterized as being incomplete (Poland *et al.*, 2009). This type of resistance is highly influenced by the environment. Genetic studies have revealed that "minor genes" with small and additive phenotypic effects are involved. It is also called quantitative resistance (QR) and does not exhibit the boom-bust cycles. These genes are theoretically effective against all races of the pathogen, even though the level of resistance may vary (McDonald & Linde, 2002). McDonald and Linde, (2002) suggested that QR is overcome by pathogens in a gradual process instead of being rapidly overcome, though there are no exhaustive studies on this. Non-specific defense mechanisms can provide broader and more robust resistance to fight pathogen evolution (Poland *et al.*, 2009).

Nowadays, new methods are rapidly being developed in plant genetics and mechanisms for resistance are being revealed at the molecular level. Many new resistance genes have been discovered and deployed successfully in some of the most important crops in the world, though in the majority of cases conferring disease rather than insect resistance. All this development has represented enormous steps forward for plant resistance to become a solid interdisciplinary activity that contributes in improving pest management and consequently food production in a sustainable way.

2. CATEGORIES OF RESISTANCE AND PLANT DEFENSES TO APHIDS

Plant resistance is defined as the genetically inherited traits in a plant of a population, or a race or variety of a certain species; resulting in less damage than in other (susceptible) individuals which lack these genetic characteristics. In this way, PR is conditioned by the presence of certain genes that express the presence or absence of certain chemical or morphological traits that interfere with the ability of an herbivore to utilize a plant, and the plant to tolerate the attack (Kennedy & Barbour, 1992; Smith, 2005).

Painter (1941) classified resistance to insects in the following three categories: Non-Preference, Antibiosis and Tolerance. However, 37 years after Painter´s classification, Kogan and Ortman (1978) proposed the term "Antixenosis" to substitute the insect-focused "Non-preference" concept so that all three terms refer to characteristics of the resistant plant.

2.1 Antixenosis

The term antixenosis is derived from the Greek word *xeno*, which means guest. Antixenosis can be considered as the first defensive line in plants against insect damage. It negatively affects the normal insect host finding and acceptance process. Consequently, the sensory systems of the insect pests are involved, i.e. olfaction, vision, gustation and thigmoreception (Smith, 2005).

2.1.1 Host finding process in aphids

The general host-location process in insects includes the following phases: 1) searching (orientation); 2) recognition (landing and probing) and 3) acceptance (feeding and reproduction).

Powell *et al*. (2006) defined a sequence of behaviors for the host selection process in aphids consisting of 6 stages: a) pre-alighting behavior; b) initial plant contact and assessment of surface cues before stylet insertion; c) probing epidermis; d) stylet pathway activity; e) sieve element puncture and salivation and f) phloem acceptance and sustained ingestion. Since aphids have low capabilities for directed flight, they can only be oriented upwind at low wind speeds; although they are able to remain airborne for long time and thus be transported considerable distances by air movements (Pettersson *et al.*, 2007). In aphids, host selection is mainly based on chemical cues (Powell & Hardie, 2001) but visual signals may also play a role (Doering & Chittka, 2007).

2.1.1.1 Pre-landing: Visual responses

Prokopy and Owens (1983) defined vision as the ability to perceive spatial patterns which are expressed as physical stimuli with certain spatiotemporal photo fluxes which differ in energy and frequency composition. Variation in brightness, hue and saturation can be registered by the optical receptors of insects. The spatial distribution of photo fluxes gives information on object shape, size, distance and motion. The vision process depends on the nature of the viewed surface, the optical background, the illuminant and viewer's angle and sensitivity (Prokopy & Owens, 1983).

The main visual system in insects is composed of two compound eyes connected to three visual ganglia in each optic lobe (lamina, medulla and lobula) of the brain (Bouzerdoum,

1993). The basic receptor units for light perception are located in the retina of the compound eye, and also in the ocelli (Doering & Chittka, 2007). Visual cues during host searching and location result from the spectral quality of light, and dimensions and shape of plants (Smith, 2005).

Electroretinograms made in the green peach aphid (*Myzus persicae* (Sulz.)) revealed three spectral types of photoreceptors (Kirchner *et al.*, 2005): green (530 nm), UV (320-330 nm) and blue (440-480 nm). No receptor for red was found in this species. The maximum peak of sensitivity was found at 530 nm.

It is well known, that aphids land preferentially on yellow colored surfaces (Pettersson *et al.*, 2007). However, not all the aphid species are attracted by yellow color to the same degree. For example the bird cherry-oat aphid (BCOA), *Rhopalosiphum padi* (L.), shows a higher response to green than yellow color, whereas the English grain aphid (EGA), *Sitobion avenae* (Fabricius), the greenbug (GB), *Schizaphis graminum* (Rondani), and the corn leaf aphid, *Rhopalosiphum maidis* (Fitch), prefer yellow rather than green (Kieckhefer *et al.*, 1976). Ahman *et al*. (1985) concluded that one important factor for landing rate by the alates of BCOA is the size of green area, possibly explaining why aphid landing rates increase as plant density increases in the field.

Apart from the visual stimuli, volatile chemicals are perceived during orientation of an insect, something that has been more emphasized in research lately (Smith, 2005).

2.1.1.2 Prelanding: Olfactory responses

Chemoreception includes olfaction and gustation, involved in the most important behaviors for host discrimination by insects (Gillot, 2005). Olfaction, the ability to perceive odors, is considered a corner stone in the insect host finding processes. Olfaction can be defined as chemostimulation by substances in low concentrations and volatile at physiological temperatures (Gillot, 2005).

The primary olfactory structures of insects are located in the antennae (Gillot, 2005). The primary rhinaria are chemosensillae that play an important role in odor detection by aphids (Pettersson *et al.*, 2007). These organs are located at the last two antennal segments.
All plants release volatiles and these may act as repellents or attractants to insects. Since blends of volatiles may differ in number of components as well as in their concentrations, these are complex plant cues for herbivorous insects. Even though there are many volatiles that are common to all plants there are also those that are specific to certain plant genera that herbivorous insects are able to exploit (Bruce *et al.*, 2005).

Some volatiles are mainly released by plants upon damage, for example methyl salicylate. This compound was found to be repellent to the BCOA, EGA, the rose grain aphid (RGA), *Metopolophium dirhodum* (Walker), and to the black bean aphid (*Aphis fabae* Scopoli) (Hardie *et al*., 1994; Pettersson *et al*., 1994; Pickett & Glinwood, 2007). Another indirect effect of methyl salicylate on aphids is as a promoter of plant-induced resistance (Pickett & Glinwood, 2007).

The plant volatile cis-jasmone, is known to have a role in plant resistance as well, and can be released by plants when an insect damage occurs (Birkett *et al.*, 2000). By spraying this compound on wheat plants at seedling stage, Bruce *et al.*, (2003) and Birkett *et al*., (2000)

showed that jasmone may act as a repellent to the BCOA, the EGA and the RGA; besides it had a negative effect on the aphid growth. Similar results with jasmonic acid on cereal aphids and thrips were reported by El-Wakeil *et al.* (2010). Interestingly, cis-Jasmone, has been found to be an attractant to some natural enemies of pests such as ladybird beetles and parasitoids (Birkett *et al.*, 2000).

2.1.1.3 Post-landing responses

Once insects have alighted on plants, their behavior is further influenced by plant morphology and chemistry (Pettersson *et al*., 2007). Insects are sensitive to a wide range of physical stimuli, such as air movement, sound, and physical contact (Gillot, 2005). Tactile stimuli are perceived by mechanosensillae typically found in great density in specialized hairs on tarsae, mouth parts, antennae and the ovipositor, but can be found in all parts of the body (Gillot, 2005). Plant morphological characteristics may affect host acceptance behaviors of insects, such as feeding and reproduction (Smith, 2005).

For example, Roberts and Foster (1983) reported differences in host acceptance by the BCOA, when aphid behavior was compared on the antixenotic wheat cultivars Downy (pubescent-leaved) and the susceptible Abe (glabrous-leaved). However, leaf pubescence is not always directly related to wheat resistance to BCOA. Papp and Mesterhazy (1993, 1996) studied the resistance in 26 winter wheat genotypes and indicated that leaf pubescence of resistant cultivars did not influence the infestation rate by the aphids in their experiments, which suggest a cause of resistance other than hairiness.

According to Guillot (2005) sensilla for tasting and smelling are structurally similar and there is no physiological neither morphological basis to separate them as different sensory systems. Upon direct contact insects may taste plants with chemosensory sensilla styloconica present on maxillary palpi, and labral gustatory receptors (Smith, 2005). But such organs can also be found in antennae, tarsae, and ovipositor depending on the taxonomic order of the insects (Guillot, 2005). In aphids, labrum surrounds the mandibular stylet (outer stylets) and maxillary stylet (inner stylets), forming the salivary and food canals. Each mandible has two dendrites that probably function as propioceptors, which allow aphids to monitor stylet position and movement during plant penetration (Powell *et al.*, 2006).

After landing, the most important factor for aphid decision to reject or accept a plant as host is information received at stylet insertion (Powell *et al.*, 2006). It is believed that aphids suck up small sap samples that are rapidly transported to the pharyngeal taste organ at probing. Plant penetration can be divided into three phases: 1) pathway phase, regarded as the phase where brief cell punctures occur; 2) xylem phase, considered as drinking to relieve water stress; and 3) phloem phase, which is where the main feeding takes place, but always preceded by sieve element salivation to suppress phloem wound response (Pettersson *et al*., 2007). It is at the phloem level where the final decision to accept or reject a plant is made (Pettersson *et al*., 2007).

A number of papers have reported significant differences in feeding behavior of GB when compared on resistant, induced resistant, and susceptible lines (*e.g.* Goussain *et al*., 2005; Pereira *et al*., 2010; Ryan *et al*., 1987).

2.1.2 Measurement of Antixenosis

Antixenosis tests are essentially based on measuring the attractiveness of a plant genotype to insects. In other words it measures the differential response by the insects to one plant genotype relative to another. It can be expressed as the number of individuals, or the amount of feeding or reproduction, per plant or plant part.

The most common type of antixenosis test with aphids is the free-choice test, in which each entry from a set of plant genotypes is equidistantly planted in a circular pattern, near the edge of a pot. Then aphids, either apterous or alate, are released in the center of the circle and counted on the plants 24 and 48 hours later (*e. g.* Flinn *et al*., 2001; Hesler *et al*., 1999; Hesler, 2005; Lage *et al*. 2003; Webster *et al*., 1994). However, Webster and Inayatullah (1988) indicated that plants sown in flats in a complete randomized design provides more accuracy on the antixenotic effects of plants compared with the circular arrangement. A variant of this free-choice test is that by Webster *et al*. (1994), in which leaf sections from different plant genotypes are placed in glass vials with distilled water and held in a testing platform. In the same way as previously mentioned, leaves of different genotypes are positioned in a circular pattern and exposed to aphids and counting is done 48 hours after aphid release. However, when this antixenosis test variant was conducted with GB, it showed contrasting results compared with the test using intact plants, but consistent results for the yellow sugar cane aphid (*Sipha flava* Forbes) (Webster *et al.*, 1994). In antixenosis tests, light orientation must be managed properly, since aphids are attracted to light sources, possibly giving false resistance/susceptibility results.

Antixenosis is considered by some authors as an important component of resistance, since it may reduce initial infestation levels (Webster & Inayatullah, 1988). However, in current agricultural practice where monoculture predominates, antixenosis might be a poor defense for plants since insects deprived of their preferred host may eventually accept a less preferred one.

2.2 Antibiosis

Antibiosis is defined as the resistance mechanism which directly and negatively affects the physiology of an insect. Antibiotic effects on insects can vary widely, and may result from morphological and chemical plant factors (Smith, 2005). This mechanism of resistance may lead to higher mortality rates, reduced body size and weight, prolonged periods of development and/or reduced fecundity of insects (Smith, 2005).

This type of resistance has been found in several plant species in relation to several insect species. For example in wheat and its relatives, antibiosis has been reported to the GB (Flinn *et al*., 2001; Lage *et al*., 2003; Smith & Starkey, 2003; Webster *et al*., 1994; Webster & Porter, 2000), the RWA (Smith *et al*. 1991, Hein 1992, Hawley *et al*. 2003), the BCOA (Hesler *et al*., 1999; Hesler , 2005) and the EGA (Lowe, 1984).

The main antibiotic defense traits are plant allelochemicals, which are non-nutritional chemicals produced by plants that affect the biology or behavior of another species. Allelochemicals can be constitutive or induced. Such compounds can be active in low concentrations (*e.g.* the hydroxamic acids DIMBOA, DIBOA), or active in a more quantitative manner (*e.g.*, apymasin, chlorogenic acid and maysin) (Smith, 2005).

Givovich *et al*. (1994) studied the correlation between concentration of hydroxamic acids and performance of BCOA on wheat and found DIMBOA-glucoside in wheat seedlings to be negatively correlated with the relative growth rate of the aphid. Similar results were found by Givovich & Niemeyer (1996) when exposing RWA to wheat plants expressing different concentrations of hydroxamic acids. Other studies conducted by Ni & Quisenberry (2000) showed that the genes *Dn5* and *Dn1* conferring antibiosis to RWA might be related to concentrations of secondary metabolites. However, *Dn5* was related to high concentrations of DIMBOA while *Dn1* was not. It is hypothesized that a gene for DIMBOA synthesis is up-regulated by *Dn5*, since the locus responsible for this and the resistance gene *Dn5* are located in different homologous chromosome groups.

Nutritional status of plants can produce antibiotic effects on insects as well (Smith, 2005). One study conducted by Cipiela & Sempruch (1999) showed that concentrations of L-DOPA (synthesized from tyrosine) and ornithine (originated from the enzyme arginase; arginine plays an important role in the urea cycle) are negatively correlated to the intrinsic rate of increase of the EGA when the substances were measured in winter wheat.

On the other hand, aphids are well known because of their ability to change plant chemistry of their hosts and in some cases, enhance nutritional levels of susceptible plants, as shown by Telang *et al*. (1999). When nymphs of RWA infested the susceptible wheat Arapahoe, results showed an increase in essential amino acids compared with non-infested plants. Castro *et al*. (2007) found four wheat substitution lines with significant increases in protein content when infested with GB. This germplasm was previously characterized by its antibiotic effects on GB and consisted of a set of intervarietal chromosome substitution lines in the background of var. Chinese Spring and one synthetic hexaploid wheat derived from *Aegilops tauschii*.

Although chemicals are the most common causes for antibiotic effects, plant structures like trichomes may also directly affect the physiology of insects in a negative way. For example, in the cereal leaf beetle (*Oulema melanopus* (L.)) (Papp & Mesterhazy, 1992, 1993), where eggs become punctured and desiccated by high densities of wheat-leaf trichomes, something which also may cause the death of larvae due to damage in their alimentary canal as they feed on densely hairy leaves (Wellso, 1973, 1979).

In induced resistance, salicylate and jasmonate play a significant role for defense-signaling, as shown for instance in wheat induced by the EGA (Zhao *et al.*, 2009). However, defense pathways are not totally understood, even though many candidate resistance genes have been identified upon up-regulation by aphid feeding (Smith & Boyko, 2007; Delp *et al*. 2009). Smith & Boyko (2007) suggested that two different processes are involved in the activation of plant defenses to aphids; 1) a process mediated by gene for gene recognition which activates plant specific defenses to aphid feeding, triggered by aphid elicitors; 2) plant recognition of aphid damage, which activate general (basal) stress responses, and consequently provoke more general changes in plant chemistry. The latter process involves signaling-pathways of plants that are common to resistant and susceptible plants, whereas the former is specific for aphid resistant plants (Smith & Boyko, 2007).

2.2.1 Measurement of Antibiosis

Antibiotic effects are reflected in the insect's physiology. Procedures for identifying such effects are more laborious than antixenosis tests since they must give information about developmental, reproduction and/or mortality rates of the insects. One way is to build life

tables which include data about insect longevity, mortality and offspring produced per female per time unit on a certain plant genotype. From this can be calculated the intrinsic rate of increase (*rm*). Theoretically, *rm* can take on values from -1 to 1, which in practical terms means that, for example, a *rm*=0.145 insect population will increase 14.5% from one time unit to the next (*e.g.* from one day to the next one), under the conditions that the experiment is conducted (Krebs, 2009). Although *rm* was developed first for demographic estimations, it can be used as a measure of antibiosis because the lower the value, the higher is the resistance.

However this method is time consuming and alternative procedures have been proposed for aphid screening. Since female aphid weight is highly correlated with number of offspring (Dewar, 1977), other researchers have proposed the Mean Relative Growth Rate (MRGR) as a parameter of resistance (Leather & Dixon, 1984), even though the method was first developed to analyze plant growth by Blackman (1919) and later revisited by Fisher (1921) and Radford (1967). MRGR is calculated in a logarithmic scale, where final aphid weight is subtracted from the initial aphid weight and divided by the time that the experiment lasts.

MRGR of the BCOA was shown to be highly correlated with the *rm* parameter and fecundity when compared in various grasses (Leather & Dixon, 1984). Additionally, it is considered a useful tool since it can be applied without any assumption on the form of the growth curve (linear, exponential, *etc*.) and can be used to compare data from different experiments and/or treatments of the same experiment (Radford, 1967).

2.3 Tolerance

Many authors, mainly ecologists, classify tolerance as a mechanism different from resistance (*e.g.* Leimu & Koricheva, 2006; Mauricio *et al*., 1997; Stowe *et al*., 2000; Strauss and Agrawal, 1999; Tiffin, 2000). The argument behind this is that tolerance does not involve plant-insect interactions *per se* and the evolutionary mechanisms are different, since by "resistance" insects experience a selective impact whereas through tolerance they do not (Stowe *et al.*, 2000).

Contrary to this concept of tolerance that is seen from the insect's perspective, the evolution and genetics of tolerance do not exclude the other mechanisms of resistance, since in theory, natural selection could simultaneously favor antixenosis, antibiosis and tolerance (Rosenthal & Kotanen, 1994). Even though there is no selection pressure by plants on insects by means of tolerance, plants are selected by certain herbivores and the ones that endure insect damage will successfully reproduce.

Tolerance is undoubtedly a complex mechanism of resistance among plants, in general terms it is defined as the ability of plants to withstand or recover from an insect attack equal to the attack caused in a susceptible genotype and it is determined by the genetic characteristics that enable plants to continue growing, recover or add new growth after and/or during insect damage (Smith, 2005). Tolerant plants tend to produce more biomass than susceptible ones; therefore, plant traits involved in biomass production are related to this resistance mechanism

(Smith, 2005). Traits such as chlorophyll content can differ according to the aphid species, due to the aphids ability to modify plant chemistry as shown below. However, Rosenthal and Kotanen (1994) pointed out that compensation, seen as re-growth, is only one among several plant responses; such as storage capacity, photosynthetic rate, allocation patterns and nutrient uptake; that vary according to extrinsic (environment, type of herbivory, spatial distribution) and intrinsic (plant genetics) factors.

The ability of plants to tolerate insect damage has been widely reported, and it is known to be frequently interacting with the other mechanisms of resistance. For example in wheat and its relatives, tolerance to GB, the BCOA, the EGA and RWA has been reported by several authors (Boina *et al*., 2005; Zhu *et al*., 2004; Flinn *et al*., 2001; Hesler, 2005; Hesler *et al*., 1999; Lage *et al*., 2003; Lage *et al*., 2004; Ma *et al*., 1998; Smith & Starkey , 2003; Zhu *et al*., 2005).

Tests for physiological responses in susceptible plants and plants with RWA resistance gene *Dn*1 (confers antibiosis) and *Dn2* (tolerance) have shown that plants carrying the tolerance gene *Dn2* had less chlorophyll losses compared with *Dn1* and susceptible plants (Heng-Moss *et al*., 2003). Other physiological observations of infested and non-infested plants with RWA and BCOA, showed a slower decline of the photosynthetic capacity in plants infested with the latter compared with RWA-infested plants (Franzen *et al*., 2008). Even though the BCOA normally does not cause any visible symptoms in plants, it does affect gas-exchange and chlorophyll fluorescence (Frazen *et al*., 2008).

Boyko *et al*. (2006) suggested that the molecular basis for tolerance to the RWA in plants carrying the *Dnx* gene involves the up-regulation of transcription sequences similar to those that regulate photosynthesis, photorespiration, protein synthesis, antioxidant production and detoxification. Ni *et al.* (2002) showed that non-damaged leaf areas of plants infested with RWA increased their concentrations of chlorophylls, while the damaged areas showed opposite trends and a higher activity of chlorophyll degradation enzymes. Thus plants may compensate the loss of photosynthetic capacity by increasing metabolic activity in non-damaged areas. Smith *et al*. (2010) found that the resistance based on *Dnx* resistance gene to RWA leads to the up-regulation of more than 180 genes related to signaling and plant defenses. However, this pattern in plants infested with RWA is probably not the same compared with that caused by the BCOA, since this aphid does not cause any visual leaf symptoms, and according to Ni *et al*. (2002) does not cause the content of chlorophyll to decrease significantly in damaged areas, whereas the concentration of carotenoids is reduced. Even though the efficiency of photosystem II is affectected negatively as found by Frazen *et al*., (2008).

2.3.1 Measurement of Tolerance

Since tolerance is related to plant responses to insect damage, its measurement greatly depends on the aphid species that is being evaluated. Whereas in the case of RWA and GB it is possible to measure tolerance by estimating chlorophyll loss (Lage *et al*., 2003; Lage *et al*., 2004; Sotelo *et al*., 2009), in the case of the BCOA and the EGA it is not possible to utilize such criteria since these aphid species do not cause significant chlorophyll losses and no visible symptoms in the plants are shown. Therefore plant growth and biomass measurements are required (Dunn *et al*., 2007; Hesler, 2005; Hesler *et al*., 1999).

Dunn *et al*. (2007) proposed a 14 day exposure method to screen relatively large germplasm collections for resistance to the BCOA by measuring shoot and root biomass in 3 week-old winter wheat seedlings. Aphid infestation is done one week after germination by placing infested leaves directly from the aphid rearing to have an average density of 10-15 aphids per test plant. After two weeks, aphids are removed and shoots and roots are dried for 48 h at 65 °C and then weighed. The experiment is repeated twice, each with five replicates of infested and un-infested plants per genotype. Statistical analysis is made by comparing infested versus non-infested plants of the same genotype.

Lage *et al*. (2003) measured tolerance to the GB by quantifying biomass and chlorophyll losses. Biomass quantification was made by confining one potted seedling per cylindrical cage (40-50 cm x 10 cm). One week after germination 20 aphids were added daily to the cylindrical cages until the susceptible check was near death. Biomass of a certain plant was compared with a non-infested plant with similar initial height, with nine replicates. Chlorophyll measurements were made by using a portable device also known as SPAD meter which measures red and infrared transmittance to calculate a value that corresponds with the chlorophyll content. Recordings were made at the feeding site of 30 aphids confined in a clip-cage, five infested were compared with five un-infested plants. Plant tissue was exposed to aphids for four days.

3. GENETIC RESOURCES FOR RESISTANCE TO APHIDS IN WHEAT

Bread wheat (*Triticum aestivum* L.) is an allohexaploid organism (2n=6x=42), composed of three genomes (A, B and D), that arose 8, 000 years ago from the hybridization of *Triticum turgidum* L. (AABB) and *Aegilops tauschii* Coss. (DD) (Faris *et al.*, 2002). Whereas the origin of A *(Triticum monococcum* L.) and D (*Ae. tauschii*) genomes are known, the origin of the B genome remains uncertain, though it is believed that it originates from *Aegilops speltoides* Tausch (Faris *et al.*, 2002). Even though wheat is a hexaploid organism, it shows a diploid-like behavior (Naranjo *et al.*, 1987).

However, the polyploid nature of hexaploid wheat enables it to buffer and tolerate numerous changes in its genome, allowing introgression of genetic variation from related species. This possibility of developing a series of various useful genetic stocks for research purposes was pioneered by E. R. Sears since early 1950s (Faris *et al.*, 2002). From an applied point of view, resistance to several biotic constraints to wheat has been successfully deployed in wheat by means of interspecific and intergeneric hybridization; some of the transferred genes are shown in Table 1.

The methods used to introduce such genetic variation into wheat are highly dependent on the evolutionary distance between the species (Friebe *et al.*, 1996). Transferring genetic variation from species belonging to the primary gene pool of wheat can be achieved by direct hybridization, homologous recombination, backcrossing and selection; species enclosed in this group are the hexaploid landraces, the cultivated tetraploid (*T. turgidum*), the wild emmer wheat *(T. dicoccoides*), the diploids *T. monococcum* and *Ae. tauschii*. From the secondary gene pool (*e. g.* polyploid *Aegilops* species, *Secale* species, *Agropyron elongatum, A. intermedium* and *A. trichophorum*) homologous recombination is possible if loci of interest are placed in homologous chromosomes, whereas for the species belonging to the tertiary gene pool (*e. g. Elymus* species), gene transfer cannot be achieved by homologous recombination but by exploiting the centric breakage-fusion of univalents, induced homoeology and radiation treatment to induce chromosome breaks (Friebe *et al*., 1996).

Chromosome pairing between wheat and rye (*Secale cereale* L.) makes it possible to transfer desirable agronomic traits from the latter into wheat. According to Naranjo *et al*. (1987) wheat chromosomes in groups 1, 2, 3, 5 and 6 are homoeologous to 1R, 2R, 3R, 5R and 6R chromosomes from rye, respectively, whereas 4R and 7R show partial reciprocal homoeology to groups 4 and 7 of wheat. This has been successfully exploited and hundreds of wheat cultivars have been produced carrying wheat-rye translocations 1BL.1RS and 1AL.1RS (L=chromosome's long arm; S=chromosome's short arm), and the chromosome substitution 1R(1B) (Rabinovich, 1998). Chromosome 1R from rye has been widely used in wheat because it carries loci for improving yield potential, wide adaptation and resistance to GB, powdery mildew, stem rust, leaf rust and yellow rust (Friebe *et al*., 1996; Kim *et al.*, 2004; Lu *et al*., 2010; Mater *et al*., 2004; Rabinovich, 1998; Villareal *et al*., 1996;).

Other genetic sources for broadening wheat diversity is provided by the *Aegilops* genus, which consists of eleven diploid, ten tetraploid and two hexaploid species, carrying diverse genomes: D, S, U, C, N and M (Schneider *et al.*, 2008). Some *Aegilops* species provide resistance to abiotic and biotic factors, which can be transferred into wheat using conventional crossing and recombination methods (Schneider *et al.*, 2008).

As an example of the utility of such genetic resources for aphid resistance, Smith *et al*. (2004b) evaluated 21 accessions from six species of *Aegilops* and one accession of *T. araraticum* that were previously identified to be resistant to BCOA and found antibiotic effects on EGA and RWA in *Ae. neglecta* accession 8052, so this accession is reported to be resistant to three aphid species. Both tolerance and antibiosis to BCOA was found in *T. araraticum*, accession 168 (Smith *et al.*, 2004b). Migui & Lamb (2003) evaluated resistance to BCOA, EGA and GB in 19 species related to wheat, and found that the ploidy level plays an important role in resistance to aphids; the species with low ploidy level were more frequently resistant. However, no single accession carried resistance to all three aphid species, but either to the combination of GB and BCOA or GB and EGA (Migui & Lamb, 2003). When considering all three resistance mechanisms, species such as *T. boeoticum*, *Ae. tauschii* and *T. araraticum* had the higher levels of antibiosis to BCOA, whereas *Ae. tauschii* and *T. turgidum* had the higher levels of overall resistance to GB, while *T. araraticum* and *T. dicoccoides* presented the higher levels of overall resistance to EGA (Migui & Lamb, 2003).

Table 1. Examples of introgressed genes from related species to wheat conferring resistance to diseases and insects.

Germplasm	Species	Gene	Trait
Transfer (T47)	*Aegilops umbellulata*	*Lr9*	Leaf Rust
2A/2M#4/2	*Ae. speltoides*	*Lr28*	Leaf Rust
C82.2	*Ae. speltoides*	*Sr32*	Stem rust
CI17884	*Ae. speltoides*	*Gb5*	Greenbug
R1A	*Ae. longissima*	*Pm13*	Powdery mildew
Compair	*Ae. comosa*	*Yr8/Sr34*	Yellow and stem rust
C747	*Triticum timopheevii*	*Sr36/Pm6*	Stem rust and Powdery mildew
Line W	*T. timopheevii*	*Sr37*	Stem rust
Tatcher	*T. timopheevii*	*Lr18*	Leaf rust
RL6087	*T. timopheevii*	*Sr40*	Stem rust
Agatha	*Agropyron elongatum*	*Lr19/Sr25*	Leaf and Stem rust
7Ag#11	*A. elongatum*	*Lr29*	Leaf Rust
Agent	*A. elongatum*	*Sr24/Lr24*	Stem and Leaf rust
K2046	*A. elongatum*	*Sr26*	Stem rust
T4	*A. elongatum*	*Lr38*	Leaf rust
Amigo	*Secale cereale*	*Gb2/Pm17*	Greenbug and Powdery mildew
GRS.1201	*S. cereale*	*Gb6*	Greenbug
WRT238	*S. cereale*	*Sr27*	Stem rust
WGRC28	*S. cereale*	*Pm20*	Powdery mildew
KS85HF011	*S. cereale*	*H21*	Hessian fly
88HF16	*S. cereale*	*H25*	Hessian fly

Modified from: Friebe *et al.*, 1996.

Interspecific crosses between tetraploid wheats and the goat grass (*Ae. tauschii*) for producing synthetic hexaploid wheats have been widely used to introduce new genetic variation into common wheat. The relatedness between the two species facilitates successful crossings and after chromosome doubling the synthetics can be field tested (Figure 1) (Mujeeb-Kazi, 1995). To successfully exploit the benefits of the interspecific crosses it is important to consider the following aspects (Mujeeb-Kazi & Wang, 1995): 1) The genome constitution of the donor species; 2) the genomic relationship between donor and recipient species; 3) chromosomal location of the loci of interest; 4) whether the gene(s) of interest can be expressed in the recipient species; and 5) whether gene transfer has any negative effect on the recipient species.

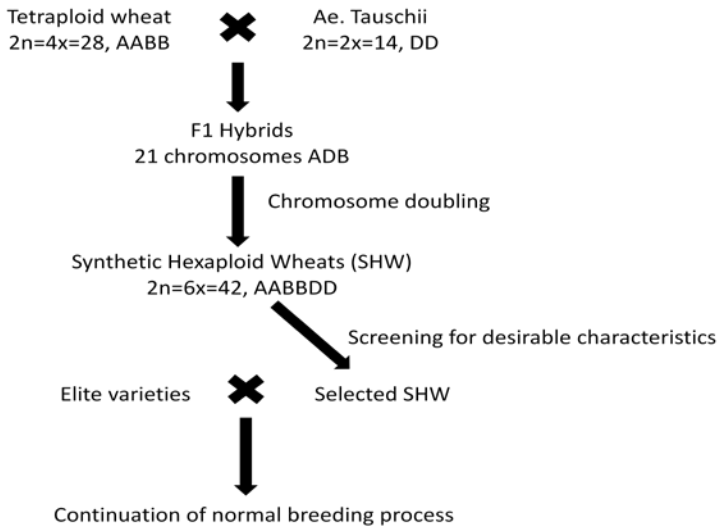

Figure 1. Development of Synthetic Hexaploid Wheats (SHW)**.** Modified from: Mujeeb-Kazi, 1995

3.1 The Greenbug (*Schizaphis graminum*)

The greenbug (GB) is a paleartic insect, probably originated from the Middle East or Central Asia. It is widely distributed in Asia, southern Europe, Africa and North and South America (Blackman & Eastop, 2007). The apterous individuals are light-green with dark-tipped siphunculi, and typically with a green longitudinal stripe on their abdomen (Stoetzel, 1987). GB feeds on species of several genera of *Poaceae*, such as: *Agropyron, Avena, Bromus, Dactylis, Eleusine, Festuca, Hordeum, Lolium, Oryza, Panicum, Poa, Sorghum, Triticum,* and *Zea* (Blackman and Estop, 2007). It is capable of transmitting Barley Yellow Dwarf Virus (BYDV) especially the SGV strain (Gray & Gildow, 2003).

Economic losses due to GB damage have been estimated to be up to $100 million per year in wheat, only in the southern Plains of the United States (Webster & Kenkel, 1999). GB can reduce grain weight by 35-40% at the low density of 15 aphids per plant in winter wheat (Kieckhefer & Gellner, 1992). Additionally, chemical control of GB can present a big challenge since populations that are resistant to organophosphorous insecticides have been identified (Peters *et al*., 1975; Teetes *et al*., 1975; Gao & Zhu, 2000).

In North America, this aphid has been a serious pest since 1880's, but it was not until 1950's that the first resistant wheat cultivars started to be developed, after the durum wheat 'DS 28A' was identified as a resistance source (Porter *et al.*, 1997). However, later, in 1961 a GB population was identified for its ability to damage DS 28A wheat. Such an aphid population was named biotype 'B' (Porter *et al*., 1997). Successively, new GB populations were identified to cause differential damage to wheat genotypes. Currently, there are eleven biotypes designated from letter A to K (Berzonsky *et al*., 2003; Porter *et al*., 1997). Since these different GB populations were designated according to their capability to injure plant genotypes with certain resistance genes, the 'biotype' concept is related to a phenotypic expression that does not totally reflect aphid genetic diversity, but it is still useful for plant breeders (Blackman & Eastop, 2007).

Weng *et al*. (2010) studied the genetic diversity based on SSR markers in GB biotypes E, G, H, I and K and other isolates collected on various hosts and found that biotypes E, I and K are genetically related. Additionally I and K biotypes were clustered in a subgroup different from biotype E, whereas biotype H is genetically distant from all of the other biotypes. Host association may have a significant role in this genetic differentiation, since biotypes I and K were first found in sorghum and biotype E was identified in wheat, whereas biotype G has been collected mainly on *Agropyron* species and biotype H on *Ae. cylindrica* and *A. intermedium* (Burd & Porter, 2006 ; Weng *et al*., 2010). By crossing different GB clones virulent to genes *Gb2* and *Gb3*, Puterka & Peters (1989) showed that the virulence is genetically ruled and that several genes with epistatic interactions could be involved, although the virulence behaved in a 'gene for gene' fashion between aphid and plant.

Contrary to the common thought that the evolution of GB biotypes resulted from the deployment of resistant cultivars, Porter *et al*. (1997) demonstrated, through a historical revision, that GB biotypes were already present in nature before resistant cultivars were widely released. In fact, another study conducted by Burd & Porter (2006) showed that biotype diversity of GB is higher than expected; in their samplings 16 populations expressed a unique response to the known resistance genes. These biotypes were identified in Kansas, Nebraska, Oklahoma and Texas in the USA, however biotypes E and I were the most widely distributed (Burd & Porter, 2006). These results support the conclusions drawn by Porter *et al*. (1997).

Because of the symptoms caused by GB (Figure 2) it is possible to perform massive screenings, allowing the identification of resistant germplasm in short time spans (10-14 days). Protocols consist of: sowing completely randomized row or hill plots of eight to ten seeds in flats; three days after emergence plants are infested by placing infested leaves on the plots with an average density of four to five aphids per plant; scores of symptoms in percent of chlorosis are taken 10-14 days after infestation, or using a 0-9 damage scale where 0=No damage and 9=dead (Berzonsky *et al*., 2003).

There are 14 genes reported to cause resistance to GB in wheat or wheat relatives, originating from various resistance sources, mostly from *Ae. tauschii* (Table 2).

Genes *Gba*, *Gbb*, Gbc, Gbd and *Gbx1* are located in the same region of chromosome 7D, and could be either allelic or linked to Gb3, but further allelism tests are needed (Zhu *et al.*, 2005). All except *Gbx1* are linked to the *Xgwm671* SSR marker, which also suggest that these loci are either allelic or linked (Zhu *et al.*, 2005). SSR markers *Xbcd98* and *Xwmc157* are tightly linked to *Gby* and *Gbz*, respectively, and correspondingly located in chromosomes 7A and 7D, they can be useful for wheat breeding programs to develop resistant cultivars to GB by marker assisted selection (Boyko *et al*., 2004; Zhu *et al.*, 2004).

Table 2. Greenbug resistance genes, origin, chromosome location, linked markers and virulence response to biotypes.

Gene	Germplasm	Species origin	Chromosome	Markers	GB biotype resistance[14]
Gb1[1]	DS 28A	*Triticum durum*	Not mapped		A, F, J
Gb2[2, 3]	Amigo; TAM107 and TAM200	*Secale cereale*	1AL.1RS	*XIA294*	B, C, J
Gb3[4, 5]	Largo	*Ae. tauschii*	7DL	*Xgwm037; Xwmc634*	C, E, H, I, J, K
Gb4[6, 7]	CI 17959	*Ae. tauschii*	7DL	Allelic or closely linked to Gb3	C, E, I, J, K
Gb5[7, 8]	CI 17882; CI 17884 and CI 17885	*Ae. speltoides*	7S(7A)		C, E, I, J, K
Gb6[3, 9]	GRS1201	*S. cereale*	1AL.1RS	*XIA294*	B, C, E, G, I, J, K
Gb7/Gbx2[5, 10]	W7984	*Ae. tauschii*	7DL	*Xwg420; Xwmc671*	C,E, I, K
Gba[11]	CETA/*Ae. tauschii* Wx1027	*Ae. tauschii*	7DL	*Xwmc671; Xbarc53*	I*
Gbb[11]	CROC 1/*Ae. tauschii* Wx224	*Ae. tauschii*	7DL	*Xwmc671; Xbarc53*	I*
Gbc[11]	68111/Rugby//Ward// *Ae. tauschii* TA2477	*Ae. tauschii*	7DL	*Xgwm671; Xgdm150*	I*
Gbd[11]	Altar 84/*Ae. tauschii* TA2841	*Ae. tauschii*	7DL	*Xgwm671; Xwmc157*	I*
Gbx1[11]	Wichita/TA1695//2* Wichita	*Ae. tauschii*	7DL	*Xwmc157; Xgdm150*	I*
Gby[12]	Sando's 4040	*T. aestivum*	7A	*Xpsr119*; *Xpr1B*; *Xbcd98* are 99.77% correlated to Gby	I*
Gbz[13]	KSU97-85-3	*Ae. tauschii*	7DL	*Xwmc671; Xbarc53. Xwmc157* is completely linked to Gbz	I*

Source: [1]Curtis *et al*. (1960); [2]Sebesta & Wood (1978); [3]Lu *et al*. (2010); [4]Joppa & Williams (1982); [5]Weng *et al*. (2005); [6]Martin *et al*. (1982); [7]McIntosh *et al*. (2010); [8]Tyler *et al*. (1985); [9]Porter *et al*. (1991); [10]Weng & Lazar (2002); [11]Zhu *et al*. (2005); [12]Boyko *et al*. (2004); [13]Zhu *et al*. (2004); [14]Burd & Porter (2006). * No data available on other GB biotypes.

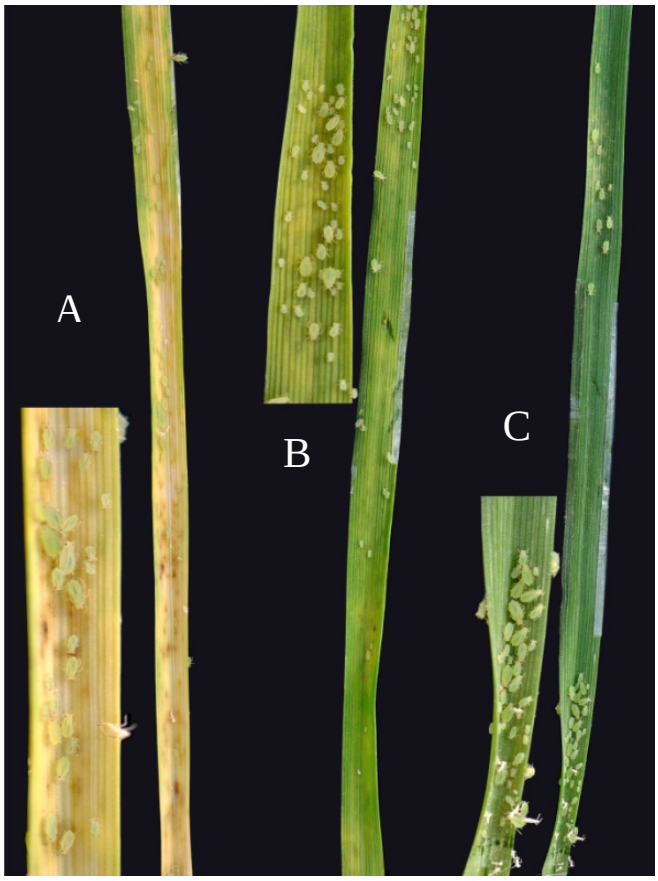

Figure 2. Greenbug biotype E on wheat leaves 15 days after infestation. A) Feeding damage on a susceptible leaf; B) Intermediate resistance reaction to GB feeding; B) Resistant leaf of Largo carrying *Gb3* resistance gene. Photo by Crespo H. L. A.

3.2 The Bird Cherry-Oat Aphid (*Rhopalosiphum padi*)

The apterous individuals of the bird cherry oat aphid (BCOA) are pear shaped, varying from yellowish-green color to dark olive or greenish-black with reddish-orange marks on the distal part of the abdomen (Figure 3). Siphunculi are swollen, constricted near to the flange (Blackman & Eastop, 2007; Stoetzel, 1987). BCOA is vector of BYDV, particularly strain PAV and strain RPV of cereal yellow dwarf virus (Gray & Gildow, 2003).

According to Blackman & Eastop (2007) the origin of this aphid is difficult to trace because it is currently distributed worldwide and its sexual phase takes part on various *Prunus* species; in Europe it overwinters on *P. padus*, and in North America on *P. virginiana*. Based on phylogenetic studies using SCAR markers on nuclear DNA, mitochondrial DNA (cyt.b) markers, and tracking life history of aphids, it has been shown that there are two lineages differing in their life cycle (Delmotte *et al.*, 2003; Simon *et al*., 1991; Simon *et al*., 1996): 1) holocyclic, with the sexual phase on the primary host (*P. padus*) and a parthenogenetic phase during summer in *Poaceae* species; 2) anholocyclic, with only the parthenogenetic phase on grasses; this occurs in places where the winter is mild.

Even though the economic losses caused by this aphid in the absence of virus are not reported, it can significantly reduce yield by 31% (Voss *et al.*, 1997) and up to 62% (Riedell *et al.*, 2003) when damage is combined with BYDV infection.

Rye is proven to be a valuable source of resistance to BCOA in wheat. It has been found that rye-derived wheat lines and triticales may express all three categories of resistance (Hesler, 2005; Hesler & Tharp, 2005; Hesler *et al*., 2007). Additionally, Tremblay *et al*. (1989) tested antibiosis and antixenosis of *A. elongatum*, *A. intermedium*, *A. repens* and *Elymus angustus* and its wheat hybrids by doing pairwise comparisons. Antibiotic effects were found in the parental grasses and the hybrids, but antixenosis was not expressed in such germplasm (Tremblay *et al.*, 1989). Dunn *et al*. (2011) estimated tolerance responses to the BCOA in 4,056 wheat accessions from the USDA-ARS National Small Grain Collection. They found that only 92 entries exhibited similar shoot and root growth with and without aphids, and 17 showed an even higher shoot and root growth compared with the non-infested counterpart. However, so far no resistance genes to BCOA have been properly identified or introgressed into elite wheat cultivars (Porter *et al.*, 2009).

Figure 3. Bird cherry-oat aphid infesting wheat spikes. Photo by Crespo H. L. A.

3.3 The English Grain Aphid (*Sitobion avenae*)

S. avenae (the English Grain Aphid; EGA) is a yellow-green or reddish brown aphid, small to medium sized and broadly elongated (1.9-3.5 mm). It has a pale cauda and typically black knees and cornicles, the latter twice as long as the cauda (Blackman & Eastop, 2007; Stoetzel, 1987) (Figure 4). The EGA aphid is a vector of BYDV, particularly the strains MAV and PAV (Blackman & Eastop, 2007).

The origin of the EGA is probably European, and it is currently present in Europe, northern and southern Africa, eastern India and Nepal and North and South America (Blackman & Eastop, 2007). This aphid species has no host alternation. It overwinters on Poaceae species where the sexual cycle occurs, even though aphids can continue reproducing

parthenogenetically all the year. Four lineages differing in their strategy of reproduction have been identified in EGA (Newton & Dixon, 1988; Dedryver *et al.*, 1998): 1) a lineage that exhibits only parthenogenesis, unable to produce sexual morphs; 2) a clone that produces males and parthenogenetic females; 3) a cyclic parthenogenetic lineage capable of producing both sexes; and 4) a lineage derived from the last group, and classified as an intermediate clone which partly turn into sexual morphs after a certain time span (two months).

According to Voss *et al*. (1997) yield losses in spring wheat caused by EGA are most significant at booting stage, reducing yield up to 21% at a density of 300 aphid-days. However, Kieckhefer & Kantack (1980) found that the most significant yield losses caused by this aphid occur at the seedling stage. Yield is reduced at boot stage, but this reduction is less compared to the one at seedling stage, probably explained by older plants being more able to compensate for the damage than seedlings, since the same population density and infection time period was applied for testing both plant stages. Normally populations of EGA have their highest reproductive rate at heading stage (Watt, 1979). In both studies (Kieckhefer & Kantack, 1980; Voss *et al*., 1997) it is concluded that the damage caused by the EGA is less deleterious than that of GB and BCOA when tested at the same density.

So far only one resistance gene to the EGA has been mapped, in the durum wheat line C273. This gene (*RA-1*) is located in the 6AL chromosome and it is reported to be linked to SSR markers Xwmc179, Xwmc553 and Xwmc201 (Liu *et al.*, 2011). Variation in resistance has been found in the wheat relatives *T. monococcum*, *T. boeticum*, *T. araraticum*, *T. dicoccoides* and *T. urartu* (Migui & Lamb, 2003; Migui & Lamb, 2004; Di Pietro *et al*., 1998), and in common wheat as well (Havlickova, 1993). Although no biotypic composition has been reported as in the case of GB or RWA, there is variation in virulence identified between different aphid clones to certain wheat cultivars and *T. monococcum* accessions (Caillaud *et al*., 1995; Lowe 1981; Xu *et al*., 2011).

Figure 4. English grain aphids infesting oat plants under rearing conditions. Photo by Crespo H. L. A.

3.4 The Rose Grain Aphid (*Metopolophium dirhodum*)

Apterae of *M. dirhodum* (RGA) are yellow-green with a darker green stripe along the dorsum; sized small to medium; cornicles not reticulated, long, pale and tip sometimes dark (Stoetzel, 1987). The RGA is a holocyclic species which overwinters on its primary hosts which are species of *Rosa*. Its secondary hosts are species of Poaceae, Cyperaceae and Juncaceae (Weber, 1985). This aphid is also a vector of the BYDV, and can efficiently transmit strains PAV and MAV.

Watt and Wratten (1984) studied the level of damage by the RGA at booting and flowering stages in winter wheat and found that it causes yield losses up to 15% when the aphids feed at the early plant stage. However, few studies have addressed wheat resistance to the RGA. Variation in resistance has been found in common wheat (Havlickova, 1997, 2001; Lamb and McKay, 1995).

3.5 The Russian Wheat Aphid (*Diuraphis noxia*)

Wingless individuals of *D. noxia* (RWA) are yellow-green or gray-green, small, convex and elongated; cornicles are pale, short, truncate and about as long as wide. It has an elongated cauda with a supra caudal process on the dorsum of the eight abdominal tergite (Stoetzel, 1987). RWA injects a toxin into plants while feeding, causing a characteristic leaf rolling, at the same time creating a protected site for the colony. When the ears are infested, these become bent (Blackman & Eastop, 2007). Leaves get white, purple and yellow streaks (Berzonsky *et al.*, 2003). Damsteegt *et al*., (1992) reported RWA to be an inefficient vector of BYDV. On the other hand, Mowry (1994) found that plant x virus interactions may or may not affect aphid performance negatively, depending on plant genotype.

RWA occurs both as holocyclic and anholocyclic. It only feeds on species of Poaceae, predominantly on wheat and barley, but it can be found in rice, rye and oats as well (Blackman & Eastop, 2007; Stoetzel, 1987). It is widely distributed as a pest in East Asia, South Africa and North and South America, but not in Australia so far. It started to become a serious introduced pest in the mentioned regions by the end of 1970's to mid 1980's. This species is also present in south and central Europe, north Africa and the Middle East; however it is not considered a serious pests in these regions (Berzonsky *et al*., 2003; Blackman & Eastop, 2007).

RWA is believed to originate from Central Asia, between Caucasus Mountains and the Tian Shan (Berzonsky *et al*., 2003), which is confirmed by molecular genetic studies on the genetic variation in several collections around the world by using RAPD and allozyme markers (Puterka *et al.*, 1993). The least variation was found in countries where RWA was recently introduced, such as in USA, Mexico, South Africa, Turkey and France, whereas the major part of variation was found in populations originated from the Middle East and southern Russia.

RWA can cause up to 40% yield losses in winter wheat at an initial density of 15 aphids per plant during 30 days exposure at seedling stage (Kieckhefer & Gellner, 1992). Additionally, Randolph *et al.* (2003) compared yield response in the susceptible wheat TAM107 and the resistant variety RWA E1 carrying the *Dn4* resistance gene and found that yield reduction in the resistant wheat is only 1% even at high infestation level. Yield losses in the susceptible

variety was directly related to aphid density, whereas yield in the resistant variety tended to be constant along the experiment (Randolph *et al.*, 2003).

Test protocols to assess potentially resistant germplasm to RWA are very similar to the ones used for GB evaluations. The difference consists in the types of scores taken since with RWA, leaf rolling and stunting are the main symptoms, along with chlorosis, which is also considered (Berzonsky *et al.*, 2003). Currently, there are 11 genes reported to confer resistance to RWA, designated from *Dn1* to *Dn9* plus *Dnx* and *Dn1881*, all of them single dominant genes except for *Dn3* which is recessive. Most of them are located in the D genome from common wheat, one in the B genome and another one in 1RS from rye (Table 3).

Liu *et al*., (2001) showed that *Dn1*, *Dn2* and *Dn5* resistance genes are either allelic or tightly linked; additionally, their result showed that these three genes are linked to the *Dnx* gene. All these genes are linked to the same SSR marker Xgwm111 and contrary to what was previously reported (Ma *et al.*, 1998) *Dn1*, *Dn2* and *Dn5* are located in 7DS and not in 7DL (Liu *et al.*, 2001).

Unlike the development of GB biotypes, it is believed that the occurrence of new genetic variation in RWA with the ability to harm wheat is due to the deployment of resistant cultivars (Weiland *et al.*, 2008). Between 1986, when RWA was first found in USA, and 2003, only one biotype occurred, but later a new biotype designated RWA-2 was identified (Haley *et al.*, 2004). Only the *Dn7* gene from rye is effective to this aphid strain (Haley *et al.*, 2004). In 2006, three new RWA biotypes were identified, RWA-3, RWA-4 and RWA-5, of which RWA-3 is virulent to all known resistance sources, including *Dn7* (Burd *et al.*, 2006). Weiland *et al*. (2008) identified three more biotypes in Colorado State, RWA-6, RWA-7 and RWA-8, to which *Dn7* gene and the wheat genotypes Stars 02RWA2414-11, CO03765 and CI2410 are resistant. Several intermediate effects where observed by the other resistance genes. Smith *et al*. (2004a) found that RWA populations originated from Chile, Czech Republic and Ethiopia were virulent to *Dn4*, which is the resistance gene most widely deployed in wheat cultivars, however, *Dn6* so far persists to be effective.

Table 3. Russian wheat aphid resistance genes, origin, chromosome location, linked markers and byotipic response.

Gene	Germplasm	Species origin	Chromosome	Markers	RWA biotype resistance[8, 9, 10]
Dn1[1]	PI 137739	*T. aestivum*	7DS	*Xgwm111*	1
Dn2[1, 5]	PI 262660	*T. aestivum*	7DS	*Xgwm111*	1
Dn3[2]	SQ24	*Ae. tauschii*		*Not mapped*	1
Dn4[3, 5]	PI 372129	*T. aestivum*	1DL	*Xgwm106; Xgwm337*	1, 4, 5 and 6
Dn5[1]	PI 294994	*T. aestivum*	7DS	*Xgwm111*	1, 5 and 8
Dn6[3]	PI 243781	*T. aestivum*	7DS	*Xgwm111; Xgwm44*	1, 4, 5, 6, 7 and 8
Dn7[4, 6]	Turkey 77	*S. cereale*	1RS	*XHor2; Xscb241*	1, 2, 5, 6, 7 and 8
Dn8[1]	PI 294994	*T. aestivum*	7DS	*Xgwm635*	1
Dn9[1]	PI 294994	*T. aestivum*	1DL	*Xgwm642*	1
Dnx[1]	PI 220127	*T. aestivum*	7DS	*Xgwm111*	1, 6, 7 and 8
Dn1881[7]	1881	*T. turgidum*	7BS	*Xgwm46; Xgwm333*	1

Source: [1]Liu *et al*., (2001); [2]Nkongolo *et al*., (1991); [3]Liu *et al*., (2002); [4]Lapitan *et al*., (2007); [5]Ma *et al*., (1998); [6]Marais *et al*., (1994); [7]Navabi (2004); [8]Burd *et al*., (2006); [9]Haley *et al*., (2004); [10]Weiland *et al*., (2008).

4. CONSIDERATIONS FOR WHEAT BREEDING

Resistance deployed into elite genotypes is reckoned to be the most economically and ecologically sound strategy to fight biotic constraints to crop production, and it is considered to be the base of an integrated pest management. However, there are several issues that must be considered to successfully transfer resistance to aphids into elite wheat lines.

The decision of what germplasm to screen is important. Wheat wild relatives (WWR) and landraces are potential sources of resistance to aphids as shown previously. Probability of success in finding resistance sources could be increased if the germplasm to be evaluated is selected from the aphids' centre of origin/diversity and/or where the WWR and the landraces have historically co-evolved with the aphids. But other potential sources should not be dismissed, such as chromosome engineered wheat.

4.1. How to identify the resistance donors?

A key point is how to accurately identify resistant germplasm using the proper protocols or screening methods adapted to the biology and behavior of aphids. There are developed protocols to rapidly identify plant genotypes potentially resistant to the RWA and the GB in relatively large collections of germplasm. However, for the BCOA, the RGA and the EGA the absence of visual plant symptoms limits the number of genotypes that can be evaluated in single experiments. Thus, identification of resistant germplasm in large collections is challenging. The proposed method by Dunn *et al*., (2007) allows screening large amounts of germplasm for tolerance to BCOA. But because of differences in microenvironment conditions and seed quality among other factors, quantifying biomass in infested vs non-infested plants might create large experimental variation since not all plants of the same genotype would grow and develop exactly at the same rate. Although this issue can be ameliorated by increasing the number of replications in the experiments, and of course, pairing plants of about the same size.

Since it is known that chlorophyll fluorescence is reduced when BCOA feeds on wheat plants (Franzen *et al.*, 2008), this could be another option for evaluating resistance to these aphid species that do not produce visual damages. If chlorophyll fluorescence is found to be correlated to plant growth in the presence of feeding aphids, it can be useful as an indirect measure of tolerance. Chlorophyll fluorescence provides information about the efficiency of photosystem II (PSII) and consequently the overall photosynthesis rate in an almost instantaneous manner. It is easy to measure and relatively easy to interpret and could perhaps be adapted to screen large amounts of germplasm. Nonetheless, experiments should be well designed because PSII is considered to be the most vulnerable photosynthetic system to light stress and under field conditions responses could be inconsistent (Maxwell & Johnson, 2000).

Methods to measure antibiosis and antixenosis are well developed and several protocols are reported in the literature, as mentioned before throughout Section 2. However, such methods are laborious and difficult to implement in large plant collections or segregating populations, especially under field conditions.

4.2. The desired type of resistance

It is common to find all three categories of resistance interacting in a single plant genotype. When no-choice tests are carried out, it is often difficult to distinguish if reduced performance of aphids is due to antibiotic or antixenotic effects. This is important to consider, since as discussed before, antixenosis could be a poor defense for plants when monoculture systems predominate. Similarly, when measuring tolerance it can be difficult to distinguish if high plant performance is due to tolerance itself, or because aphid damage is reduced due to the plants are expressing one or both of the other two categories of resistance. However, there are reported genes that exclusively or predominantly provide one of the three categories of resistance to GB and RWA.

Since antibiosis causes high selection pressure on aphid populations the risk of promoting other virulent biotypes can be high, whereas tolerant germplasm does not put selection pressure on the insects and therefore the risk of favoring new virulent biotypes is minimum.

From the perspective of management of resistance, tolerance could be the most desirable category to deploy. However, besides being a complex trait and difficult to breed for, in farmers' fields, exclusively tolerant varieties will be infested by aphids as if they were susceptible but without significantly affecting production of biomass or seed yield. This could lead to a continued use of insecticides by farmers without exploiting the advantages of tolerant germplasm. One disadvantage of exclusively and/or predominately tolerant varieties could be that virus spreading will not diminish since aphid behavior or physiology is not affected; whereas antibiotic germplasm can reduce virus spreading by negatively affecting aphid performance (Tanguy & Dedryver, 2009). Unlike tolerant plants, antibiotic genotypes might provide the most spectacular resistance effects in the field by significantly reducing aphid populations. A combination of both mechanisms could be more advantageous than the deployment of a single one.

4.3. Breeding for multiple resistance to aphids

Another consideration for wheat breeding is the genetic diversity of aphids; it is well known that virulence patterns can vary in different geographic regions as demonstrated in RWA by Haley *et al*., (2004), Weiland *et al*. (2008) and Smith *et al*. (2004a). In the case of GB in the US, it is known that there are several unique biotypes which differ in virulence to the known resistance sources, and these biotypes were present in nature before resistant cultivars were grown (Porter *et al.*, 1997). This makes it necessary to consider the target region for which wheat is bred and to have information on the virulence patterns and dynamics of aphid populations in such geographical regions.

Discovery of broad resistance is ideally the best, but could be difficult to achieve. Currently most of the resistance genes that have been reported interact with aphids in a gene for gene fashion. Combining resistance genes would be a suitable option in the absence of resistance genes with broad effects. Porter *et al*. (2000), made crosses with parents carrying *Gb2*, *Gb3* and *Gb6* resistance genes to GB, and evaluated the F1 populations against biotypes E, F, G, H and I. It was shown that by pyramiding *Gb2* and *Gb3* resistance genes to GB no additional resistance levels are conferred, whereas the virulence pattern is reduced when *Gb3* and *Gb6* genes are combined, but pyramided genes had no stronger effects on aphid performance compared to the parents carrying the single genes (Porter *et al.*, 2000). A careful selection of genes to be combined is crucial.

The fact that the "biotype" concept expresses only a segment of the full aphid genetic diversity should not be overlooked, but for practical purposes evaluations of the known resistance sources to aphids in the different geographic regions where aphids are problematic could be a start and it would provide a general panorama of the "biotypic" composition. Whether the results show that populations respond similarly or differently to resistance sources, it is valuable information for resistance breeding. This necessarily involves collaborative efforts between institutions and a continuous flow of information.

Several pests commonly occur in the same geographic region, for example BCOA which is distributed worldwide and GB that is well distributed in southern Europe and North and South America. Thus it is common to find two or more aphid species in the same field and sometimes even on the same plant. However insects compete for resources and usually one species predominates over others. Therefore, constantly growing resistant varieties to a single species may lead to the predominance of the species that was not previously problematic. Finding genetic resources resistant to multiple species is the most desirable solution. As mentioned in previous sections, resistance to two or three aphid species have been found in wild relatives of wheat. Unraveling the genetic basis of such resistance sources is important, since the number of genes and their interaction are important aspects for plant breeding procedures.

One of the challenges for big breeding programs is that protocols to evaluate aphid resistance are difficult to implement on a large scale. Field selection represents a particularly difficult task in those species that damage wheat in the absence of visual symptoms, but for species such as the GB and the RWA, field selection can be reliably done as long as a homogeneous infestation of insects is present in the field, something that can be difficult to achieve regardless of natural or artificial infestations. Another problem with the EGA is that in some cases there is no correlation between seedling and adult plant resistance (Migui & Lamb, 2004). This becomes more significant if we consider that those aphids are more abundant and thus more deleterious at later stages (Voss *et al.*, 1997). Thus if phenotypic selection is carried out, it needs to be done at late plant stages (ear emergence and flowering). This is an additional complication, since it requires plants to be maintained free of non-target pests that may otherwise interfere with selection.

The combination of the selected bulk and single backcrossing approaches for wheat breeding has showed to be highly efficient in developing high yielding germplasm with resistance to diseases, especially when traits are inherited in a quantitative manner (Singh & Trethowan, 2007). In computer simulations Wang *et al.* (2009) showed that the use of this method is particularly advantageous when polygenic inheritance rules the traits of interest, conventional phenotypic selection is carried out and when the donor parents have some favorable alleles for agronomic performance.

As an option, molecular markers can greatly facilitate plant selection during the breeding process. In that sense, discovery of resistance sources and gene mapping of those, are important to consider as a fundamental part of selection strategies. However, if marker based selection is implemented in early segregating populations, it will require an efficient sampling, DNA extraction, a platform for genotyping and a rapid information flow system. This will still reduce the number of plants possible to asses and therefore will impact negatively on the advantages of the selected bulk breeding approach under phenotypic selection.

One way of incorporating aphid resistance as a trait to select for in wheat breeding via marker selection in the context of the selected bulk approach and without sacrificing the population size, could be by screening single plants for markers linked to the trait in the latest breeding generation, BC1F5/F6, or doing so when advanced lines are obtained (Figure 5). An additional advantage of implementing option A as shown in Figure 5 is that the initial advanced lines derived from the directed crosses for aphid resistance can as well be phenotyped, whereas phenotyping is more complicated in the segregating populations, especially for BCOA, EGA and RGA. This approach can be particularly useful if those symptomless aphid species are the breeding objective.

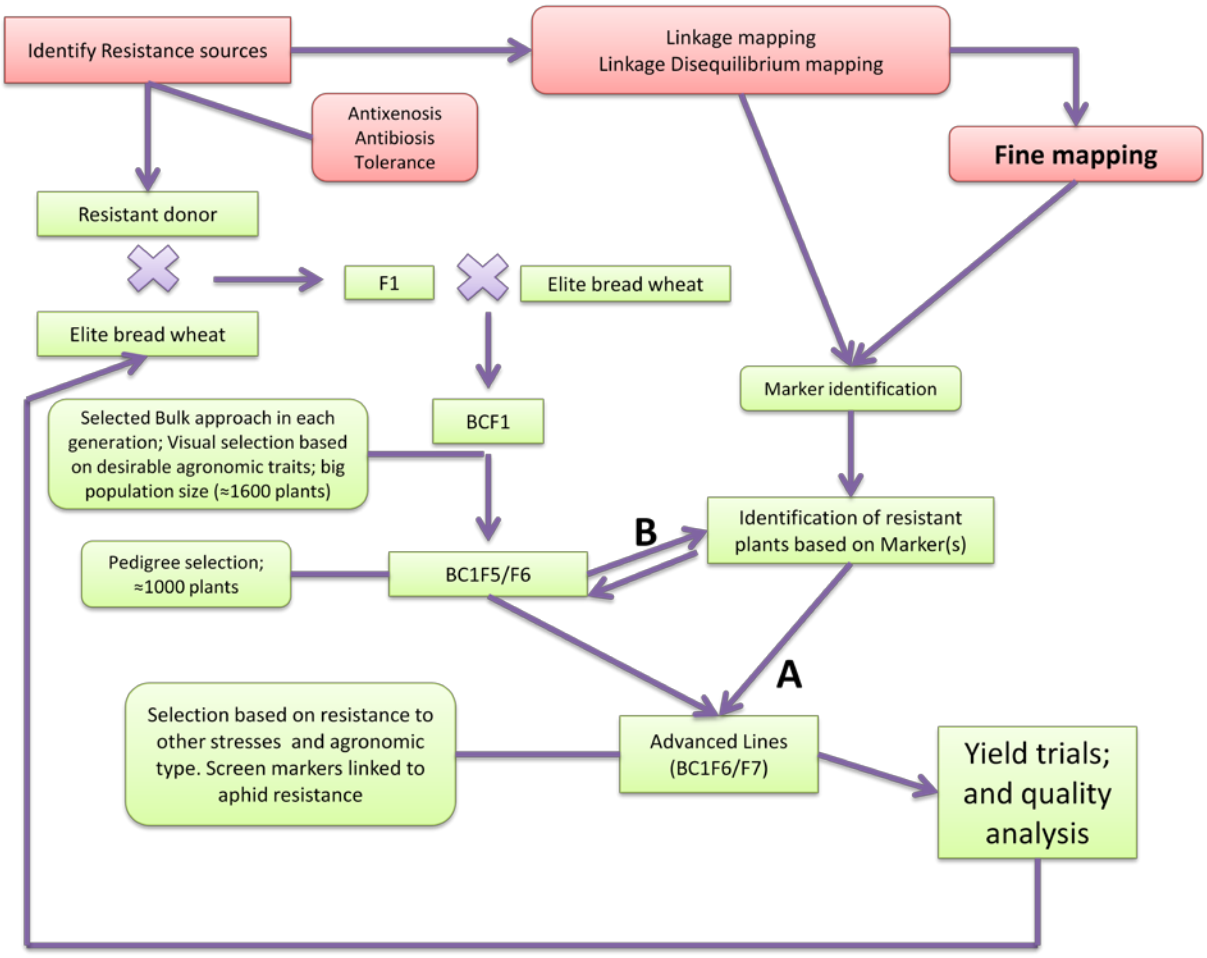

Figure 5. Incorporation of aphid resistance breeding in the selected bulk-single backcrossing approach as a trait to select for in wheat breeding by using molecular markers. **A)** Marker screening at late generations during pedigree selection; **B)** Marker analysis of the advanced lines at the end of the selection process.

A drawback following this strategy is the risk of not selecting all the plants carrying the resistance. Because, depending on the aphid abundance in the field, the resistance genes would not always contribute to fitness of plants. For instance, if a single gene was ruling the resistance, the allele frequency in the population will be 0.5 in the F2, and as homozygosis is approached and heterozygosis is reduced through generations, this allele frequency would not change unless resistance is linked to other traits, thus if for example 60 advanced lines are selected from a cross, there is the chance that half of them do not carry the resistance gene; if the number of resistance genes is higher, and if there is no linkage, the probability of having

two or more genes in a single advanced line becomes lower since the chances for them to co-segregate would be an independent event. Therefore the importance of having big population sizes and the possibility of marker assisted selection.

However, in the context of the selected bulk-single backcrossing scheme, when markers are not available or possible to use for logistic or economic reasons, dissecting the resistance mechanism of the donors is fundamental, since the selection method would be exclusively based on the phenotype and it should be adequate for the mechanism that is being transferred from the donor parent. If this was the case, phenotypic selection can hardly be implemented in the segregating population for symptomless aphid species, and it would perhaps be more efficient to do so with the advanced lines at the end of the breeding process. Therefore, phenotypic selection ideally needs to be not only precise but also simple, rapid and cheap. Thus, efforts to develop such screening methods must be made. For species such as RWA and GB it might be enough to ensure homogeneous distribution of the aphids by careful artificial infestations.

Some of the general considerations for wheat breeding involving quantitative traits suggested by Singh & Thretowan (2007) are the following:

- Careful selection of parents that will be used for crossing. Some genotypes have better combining ability and inherit more easily their characteristics to the offspring
- Single backcrossing approach favors retentions of most of the desired additive genes, and allows incorporation and selection of useful small effect genes from the donor parents
- Crossing parents carrying different sets of additive genes
- Develop large populations of segregating material to increase the probability of selecting good combinations
- Analyze obtained lines with molecular tools, if available, to confirm the presence desired genes

In the case of diseases, when broad resistance is not present in single plant genotypes, Singh & Thretowan, (2007) suggested intercrossing the resistance sources before crossing them with the elite material, and by having large segregating populations and utilizing flanking markers in early generations it is possible to combine different resistance genes in single genotypes. This strategy could be carried out if multiple resistance to aphids is not found in single wheat genotypes.

4.4. Concluding remarks

If aphid resistance is exclusively targeted, many of the previous considerations would not be as crucial as they were discussed and breeding would be more feasible and relatively easier to handle with small population sizes. However, this is usually not the case and aphid resistance is considered as only one among several desired characteristics for its incorporation into cultivated wheat, such as higher potential yield, adaptability to the conditions were plants will be grown, end-use quality, and tolerance/resistance to other abiotic and biotic stresses (heat, drought, diseases etc.). Hence, ways to easily implement aphid resistance selection (phenotypic or marker assisted selection) in wheat breeding programs are necessary, without sacrificing efficiency of breeding for other traits. In that sense phenotyping may be a bottle neck in germplasm enhancement for resistance to aphids. Even though selection methods for

aphid resistance breeding can be a challenging issue, they can also be well fitted into the current wheat breeding methods and take advantage of the new breeding technologies such as marker assisted selection or genomic selection.

There is a large variation of resistance traits in wild relatives of wheat and wheat landraces that can successfully be exploited by wheat breeding programs. However, the pre-breeding process is a crucial step in which efforts must be made before transferring resistance from less adapted germplasm, for example when using SHWs or wheat-alien translocations.

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