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Achievements and Challenges in Legume Breeding for Pest and Disease Resistance

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ABSTRACT (max. 300 words):

Yield stability of legume crops is constrained by a number of pest and diseases. Major diseases are rusts, powdery and downy mildews, ascochyta blights, botrytis gray molds, anthracnoses, damping-off, root rots, collar rots, vascular wilts and white molds. Parasitic weeds, viruses, bacteria, nematodes and damages caused by chewing and sap-sucking insects add to this long list of constraints for legume production. Their incidence and relative importance together with current understanding of their interactions with the host plants are presented.

State of the art of current achievements and limitations for breeding for biotic stress resistance are listed and critically discussed. The recent development of large scale phenotyping, genome sequencing and analysis of gene, protein and metabolite expressions can be of great help to further decipher plant-pathogen interactions and identify key resistance components that may be introgressed into crop plants through breeding.

KEY WORDS: rust, powdery mildew, downy mildew, ascochyta blight, botrytis gray mold, anthracnose, damping-off, root rot, collar rot, vascular wilt, white mold, parasitic weed, virus, bacteria, nematode, insect pests, marker assisted selection, genetics, transcriptomics, proteomics, metabolomics,

I. CURRENT AND EXPECTED FUTURE IMPORTANCE OF MAJOR BIOTIC STRESSES ON LEGUME CROPS

Grain and forage legumes are among the most important crops worldwide just behind cereals for both animal and human consumptions (Graham and Vance, 2003). However, yield of most legumes is still relatively variable and low due to limited adaptability of available cultivars to a broad range of environmental conditions, and susceptibility to pests and diseases.

A. Biotrophic fungi

Foliar diseases caused by biotrophic pathogens, such as rusts, downy mildews and powdery mildews, are major limiting factors in legume production and the most important of these are present in all areas where legumes are cultivated (Sillero *et al.*, 2006).

Several rust species can infect grain and forage legumes, most of them belonging to the genus *Uromyces* but also to other genera such as *Phakopsora* or *Puccinia*. *Uromyces viciae-fabae* causes faba bean rust, being of importance in the Middle East, North Africa, Europe and China, where moderate to substantial yield losses can occur (Sillero *et al.*, 2010). *U. viciae-fabae* is considered to be the causal agent of the rusts of other legumes such as lentil, pea and vetch. However, although no *forma speciales* (ff.spp.) have formerly been acknowledged, it is clear that isolates of *U. viciae-fabae* are specialized with respect to their hosts (Emeran *et al.*, 2005, 2008; Barilli *et al.*, 2011). *U. viciae-fabae* has been reported to infect pea particularly in sub-tropical areas (Kushwaha *et al.*, 2006), while in cooler regions *U. pisi* is predominant (Barilli *et al.*, 2009b). *U. appendiculatus* infects common bean worldwide, but it is most prevalent in

humid tropical and subtropical areas (Miklas *et al.*, 2006). *U. ciceris-arietini* infects chickpea causing significant losses if infection occurs early in the growing season (Sillero *et al.*, 2012).

Soybean rust is caused by *Phakopsora pachyrhizi* and *P. meibomiae*. The most aggressive one is *P. pachyrhizi*, known as the Asian soybean rust, which is widespread in the eastern hemisphere from the former USSR to Japan, Australia, India and China, and has recently been introduced in Africa, Hawaii, South America and continental USA (Schneider *et al.*, 2005). *P. meibomiae*, the less virulent species, has only been found in limited areas of the western hemisphere. Groundnut rust (*Puccinia arachidis*) is an economically important disease, which is present in almost all areas of the world where the crop is grown (Subrahmanyam and McDonald, 1983).

Downy mildew is a disease caused by the pathogen *Peronospora viciae*, including ff.ssp. *pisi* and *fabae*, affecting pea and faba bean, respectively. Downy mildews are widespread diseases but are most frequent and severe in cool, maritime climates (Ahmed *et al.*, 2000; Biddle, 2001).

Early studies ascribed *Erysiphe poligony* as the causal agent of most powdery mildews infecting legumes. Several species are acknowledged today, although classification is still under revision in some instances, and unclear in others. The most important is *E. pisi*, for which ff.spp. *pisi*, *medicaginis* and *vicia-sativa* have been described infecting pea, alfalfa and vetches, respectively (Falloon and Viljanen-Rollinson, 2001). Recently *E. baeumleri* and *E. trifolii* has also been reported to cause powdery mildew on lentil and pea (Ondřej *et al.*, 2005; Attanayake *et al.*, 2009, 2010). Other species cause powdery mildew on other crops, such as *E. diffusa* on soybean, *Podosphaera phaseoli* on cowpea and *Leveillula taurica* on chickpea. The identity of the powdery mildew infecting common bean is still unclear, but it seems to be close to

E. diffusa (Almeida *et al.*, 2008; Trabanco *et al.*, 2012). Powdery mildew is also important in *Hedysarum coronarium*, being incited by a very specific mildew species still unnamed.

B. Foliar Necrotrophic fungi

The major foliar necrotrophic fungal diseases in legumes are ascochyta blight, chocolate spot and anthracnose (Tivoli *et al.*, 2006). Ascochyta blight is incited by different pathogens in the various legumes. *Didymella rabiei* causes this disease in chickpea, *D. fabae* in faba bean and *D. lentis* in lentil (Hanounik, 1980; Nene and Reddy, 1987; McDonald and Peck, 2009). In pea, the disease is caused by a complex of fungal species that includes *Ascochyta pisi*, *Didymella pinodes*, and *Phoma medicaginis* var. *pinodella* (Tivoli *et al.*, 2006; Khan *et al.*, 2013). *Phoma koolunga* and *P. glomerata* have also been reported to be part of this complex in Australia (Davidson *et al.*, 2009; Tran *et al.*, 2013). Of the ascochyta complex in pea, *D. pinodes* is the most frequent and damaging (Tivoli and Banniza, 2007). *Phoma medicaginis* var. *medicaginis* and *P. medicaginis* var. *macrospora*, which differ in conidial size and septation, both contribute to spring black stem and leaf spot of alfalfa (Boerema *et al.*, 1993). *Medicago truncatula* has been also described as a host for *P. medicaginis* var. *pinodella* (Ellwood *et al.*, 2006).

Botrytis gray mold caused by *Botrytis cinerea* is a serious disease of chickpea and lentil (Davidson *et al.*, 2004; Pande *et al.*, 2005). Severe epidemics have occurred in Argentina, Australia, Bangladesh, India, Nepal and Pakistan. In faba bean, chocolate spot, caused by *B. fabae*, is also quite a destructive disease (Stoddard *et al.*, 2010).

Anthracnoses, caused by *Colletotrichum lupini* in lupin, *C. truncatum* in lentil and *C. trifolii* in alfalfa, are also important diseases (O'Neill and Saunders, 1994; Sweetingham *et al.*, 1998; Buchwaldt *et al.*, 2004, Mackie *et al.*, 2007), in particular in warm humid climates, where anthracnose stem lesions and crown rot are major limitations to lucerne persistence and productivity. On lentil, the pathogen has two distinct races (Buchwaldt *et al.*, 2004). Annual medics (Torregrosa *et al*., 2004) are also subjected to anthracnoses caused by *C. trifolii*.

C. Soil-borne, necrotrophic and hemibiotrophic fungi and oomycetes

Many soil-borne pathogens can affect legumes. Despite the high diversity of soil-borne pathogens, they can be classified in 3 main diseases, damping-off / root rot, vascular wilt and stem wilt (Infantino *et al.*, 2006). Impact of these diseases on legume crop production is highly dependent on environmental conditions. Damping-off, and root rots caused by *Aphanomyces euteiches*, *Rhizoctonia solani*, *Phytophthora* spp. and *Verticillium* spp. are favoured by humid and cooler climate as found in northern European and North American latitude (Kraft and Pfleger, 2001; Vandemark *et al.*, 2006; Chen *et al.*, 2011). In contrast, fusarium wilt and root rots caused by *Fusarium* spp. and *Macrophomina phaseolina* are generally favoured by warm and dry conditions (Chaudhary *et al.*, 2006; Afouda *et al.*, 2009). As a consequence, their incidence was reported to increase under water-stress and compacted soil conditions (Chaudhary *et al.*, 2006). The adverse impact of the soil-borne pathogens is expected to increase in near future due to climatic changes that are likely to change the repartition and increase the intensity of these diseases.

Damping-off is characterised by seedling wilting at emergence. This disease is caused by multiple fungal and oomycetes pathogens alone or in complex. The most frequent legume damping-off pathogens are *Pythium* spp. and *Rhizoctonia solani*, for which there is a number of legume-infecting anastomosis groups (Anderson *et al.*, 2013). Both can lead to up to 80% of plant death in the field (Denman *et al.*, 1995; Wang *et al.*, 2003). Damping-off can also be induced by *Fusarium* spp., *Macrophomina phaseolina* and *Phytophthora* spp. The main agent of damping-off by *Fusarium* species is *F. solani* (Etebu and Osborn, 2009) although *F. graminearum* has also been described as major damping-off agent of soybean (Ellis *et al.*, 2012).

When infection with these fungal species occurs at later stages, the disease is expressed as root rot. Among the different root rot pathogens, *Fusarium solani* and related species are a major threat to most legumes including common bean, pea, chickpea and lentil (Schneider *et al.*, 2001; Hamwieh *et al.*, 2005). *Fusarium virguliforme* (formerly *F. solani* f.sp. *glycine*) and associated *Fusarium* spp. are also responsible for the sudden death syndrome, one of the major constraints of soybean growth in most growing areas (Wrather and Koenning, 2009). Aphanomyces root rot caused by the oomycete *A. euteiches* is also an important legume threat and is one of the major constraints of pea production in North America and Europe (Gaulin *et al.*, 2007). Like most soil-borne pathogens, *A. euteiches* has a very wide host range and it is able to cause disease to many legume crops including common bean, vetch, lentil, faba bean, alfalfa and red clover (Levenfors *et al.*, 2003). *Macrophomina phaseolina*, responsible for charcoal rot disease, is another important root rot inducing pathogen. It has been reported in many legume growing areas from Northern European countries to New Zealand as an important pathogen of common bean, soybean, mungbean, peanut, cowpea, faba bean and chickpea (Abawi and Pastor-Corrales, 1989; Afouda *et al.*, 2009;

Devi *et al.*, 2011; Gupta *et al.*, 2012). For instance it was reported to rank within the top 10 major yield limiting diseases of soybean in USA (Wrather and Koenning, 2009). Brown root rot of alfalfa and other perennial legumes, caused by *Phoma sclerotioides* (synonym *Plenodomus meliloti*), develops as plants emerge from winter dormancy. Lesions that girdle the taproot at or near the crown lead to plant mortality (Wunsch and Bergstrom, 2011).

Vascular wilt in chickpea, pea, lentil, common bean and alfalfa is mainly caused by the ascomycete *Fusarium oxysporum* ff.spp. *ciceri*, *pisi*, *lentis*, *phaseoli* and *medicaginis*, respectively. Fusarium wilt is a major constraint in the production of pulse crops such as chickpea (Navas-Cortes *et al.*, 2000; Nene and Reddy, 1987), pea (Kraft, 1994) and lentil (Bayaa *et al.*, 1997) in most growing areas. It can also cause yield reductions in common bean (Abawi and Pastor-Corrales, 1989), soybean (Zhang *et al.*, 2009), lupin (Abd El-Rahman *et al.*, 2012), cowpea (Pottorff *et al.*, 2012), pigeonpea (Marley and Hillocks, 1996), alfalfa (Antonopoulos and Elena, 2008), birdsfoot trefoil (*Lotus corniculatus*) (Wunsch *et al.*, 2009) and red clover (Venuto *et al.*, 1995). *F. oxysporum* f.sp. *medicaginis* can also infect the model legume *M. truncatula* (Ramirez-Suero *et al.*, 2010). Apart from fusarium wilt, vascular wilt disease can also be induced by *Verticillium dalhiae* and *V. albo-atrum* that provoke similar wilt symptoms. In temperate regions, *V. albo-atrum* is a major pathogen of alfalfa that can reduce alfalfa production by up to 50% (Vandemark *et al.*, 2006). Besides this specific case, verticillium wilt has also been described in pea (Isaac and Rogers, 1974), faba bean (Berbegal and Armengol, 2009), soybean (Tachibana, 1971; Wiles, 1968), clover (Milton and Isaac, 1976), cowpea (Wiles, 1968) and *M. truncatula* (Ben *et al.*, 2013b; Negahi *et al.*, 2013b).

White mold or sclerotinia stem rot of legumes is caused by three ascomycete species of *Sclerotinia*. *Sclerotinia sclerotiorum,* one of the most ubiquitous plant pathogens, causes white mold on more than 400 plant species including all legume crops (Boland and Hall, 1994). *S. sclerotiorum* causes white mold of common bean, and of nonlegume crops such as canola and sunflower (Boland and Hall, 1994). *S. sclerotiorum* has also been reported to cause mild to moderate damage on pea mainly in humid and high temperature conditions (Huang and Kokko, 1992). *Sclerotinia trifoliorum* causes stem rot or white mold of mostly cool season legumes like alfalfa, clover, and chickpea (Kohn, 1979; Njambere *et al.*, 2008). *Sclerotinia minor* caused white mold of peanuts and chickpea, and also of non-legume crop lettuce (Kohn, 1979; Matheron and Porchas, 2000). The three species of *Sclerotinia* were reported to cause white mold on chickpea. The species of *Sclerotinia* are all characterized by producing melanised vegetative structured sclerotia, which enable the species to survive in soil for up to eight years (Njambere *et al.*, 2008).

Collar rot of legume crops is caused by the Basidomycete *Sclerotium rolfsii*. Infection by this pathogen is usually restricted at the base of the plant in contact with the soil. It is a common and important pathogen of legumes, crucifers and cucurbits and its common legume hosts include chickpea, common bean, lentil and peanuts. It produces two kinds of hyphae, cord-like strands that extend into the soil and spreading from plant to plant, and irregular slender hyphae that are involved in plant infection. It also produces small sclerotia that can help the fungus survive in the soil. The disease is favoured by high moisture and warm temperature (>25 ºC). It may also cause stem or pod rot in several legumes under cool or warmer weather, respectively (Kolkman and Kelly, 2003).

D. Parasitic weeds

Legume crops can be damaged by a number of broomrape (*Orobanche* and *Phelipanche*) species. *Orobanche crenata* is widely distributed in the Mediterranean basin and Middle East, and it is a major constraint for most grain and forage legumes. *Orobanche minor* is widely distributed, but of economic importance only on clover for seed production. *Phelipanche aegyptiaca* (syn. *Orobanche aegyptiaca*) is not only of impotance on legumes but also on many vegetable crops in the Middle East and Asia. *Orobanche foetida* is widely distributed infecting wild legumes in Western Mediterranean area, but has been only been reported as a problem to faba bean in areas of Tunisia and more recently to common vetch in Morocco (Parker, 2009; Rubiales and Fernández-Aparicio, 2012).

Striga gesnerioides and *Alectra vogelii* cause considerable yield reduction of grain legume crops, particularly cowpea, throughout semi-arid areas of sub-Saharan Africa. Dodders (*Cuscuta* spp.) can be also damaging on some legumes in certain regions (Riches *et al*., 1992; Parker, 2009; Rubiales *et al.*, 2006).

E. Viruses

Viruses are obligate, subcellular agents that reside and multiply inside living cells, move in plants, and frequently cause diseases. Viruses may cause different symptoms on different host species or host cultivars, while different viruses may cause the same or similar symptoms on the same host plants. Thus accurate identification of viruses requires immunological and/or molecular techniques. Viruses enter plants through vectors or infected seed, and are spread from plant to plant by insect vectors. Thus for virus diseases, the host plant intimately interacts with both the pathogenic

viruses and their insect vectors, consequently, resistance mechanisms could be directed towards the virus, the vector or both. The most important vectors of legume viruses are aphids, particularly pea aphid, cowpea aphid, green peach aphid and soybean aphid, and leaf beetles.

On soybean, bean pod mottle virus and soybean mosaic virus frequently cause economical damages. Bean leaf beetle transmits bean pod mottle virus, while aphids and infected seeds transmit soybean mosaic virus. When infection or co-infection by both viruses occurs early in the season, seed yield and quality may be significantly reduced. Virus infection also predisposes soybean to seed infection by the fungal pathogen *Phomopsis*. Other important soybean viruses include alfalfa mosaic virus transmitted by aphids from forage legumes and tobacco streak virus which can be seed borne and transmitted by thrips.

There are 31 viruses reported that infect peanuts. Fortunately only a few of them are economically important (Nigam *et al.*, 2012). The most important virus disease of peanut varies based on the production region. In Africa, the groundnut rosette disease is the most important, while in the US it is tomato spotted wilt virus. Peanut bud necrosis disease in south Asia, peanut stripe virus disease in East and Southeast Asia, and cucumber mosaic virus disease in Argentina and China are also economically important (Nigam *et al.*, 2012).

On cool season legumes, the most important virus diseases include alfalfa mosaic virus, cucumber mosaic virus, pea enation mosaic virus, bean leaf roll virus, pea streak virus and pea seedborne mosaic virus. Because aphids transmit the viruses, epidemics of virus diseases always coincide with aphid epidemics. Thus forecasting of potential virus diseases relies on monitoring aphid populations. Among cool season

legumes, chickpea is unique in its reaction to virus diseases. Chickpea plants in general are not a good host to aphids and can even be toxic to some aphids. Although aphids, once landed on chickpea plants, are able to temporally feed on, and transmit viruses to the chickpea plants, aphids cannot multiply on them. Thus there is no or minimum in field spread of viruses from plant to plant. Consequently, a major characteristic of virus disease of chickpea is scattered individually-diseased plants in the field. Significant damage can occur if the aphid population is high. Developing chickpea cultivars resistant to viruses need to consider these aspects of virus diseases of chickpea.

F. Bacteria

Many destructive plant diseases are caused by bacteria including wilts, soft rots, many cankers, and several stem and leaf spots, such as the halo blight of beans. These diseases are mainly due to Gram-negative plant bacterial pathogens, including *Pseudomonas* sp., *Xanthomonas* sp., *Erwinia* sp. and *Ralstonia solanacearum*, which use the type III secretion system to deliver a repertoire of effector proteins into their host cells (He and Jin, 2003). For legumes, bacterial diseases may be classified into three main groups: leaf blights and leaf spots, bacterial wilts, and more diverse symptoms such as sprout rot and dwarfing.

Bacterial leaf blights and leaf spots are often caused by infection with bacteria of the *Xanthomonas axonopodis* or *Pseudomonas syringae* species. Both of these species present a large number of pathovars that may infect one or more legume species. For example, *Xanthomonas axonopodis* pv. *phaseoli* (synonym *X. campestris* pv. *phaseoli*) cause common leaf blight of common bean, *X. axonopodis* pv. *glycines* affects soybean, *X. axonopodis* pv. *vignicola* causes the major disease of cowpea worldwide and *X.*

axonopodis pv. *alfalfae* affects alfalfa. Greater damage is more likely when early plant infection occurs, due to premature defoliation (Irigoyen and Garbagnoli, 1997). Disease is more severe under conditions of high temperature and rainfall.

Pseudomonas syringae pv. *pisi* is a seed-borne pathogen that causes pea bacterial leaf blight. Interestingly different pathovars of *P. syringae* can cause different diseases on the same host. For instance, *P. syringae* pv. *syringae* induces bacterial brown spot of common bean while halo blight of bean is caused by *P. syringae* pv. *phaseolicola* (Arnold *et al.*, 2011). The latter species (previously described as *P. medicaginis*) also causes bacterial stem blight of alfalfa (Harighi, 2007). These reports indicate that different diseases may be caused by the same pathogen, depending on the legume host species. *P. syringae* pv. *phaseolicola* also infects several other legumes, such as mung bean (*Vigna radiata*), pigeon pea (*Cajanus cajan*) and wild bean relatives (Taylor *et al.*, 1996) indicating that pathovar is not in a one-to-one relationship between bacterial pathogen and host species.

Bacterial wilt due to the Gram-positive bacterium *Curtobacterium flaccumfaciens* pv. *flaccumfaciens* mainly affects common bean, cowpea, and soybean. Natural infections also occur on other *Vigna* spp. and pea. Disease symptoms are described as gradual wilting of the seedling leaves, appearance of broad irregular yellow areas starting from the leaf margin and extending inwards, followed by necrosis of the parenchymal tissue. The bacterium is also able to colonize the seeds via vascular tissue. (Tegli *et al.*, 2002).

Ralstonia solanacearum is also a causal agent of bacterial wilt. *R. solanacearum* race 1 has the broadest host range, which includes solanaceous food crops and legumes. Bacterial wilt caused by *R. solanacearum* is the most important bacterial disease

affecting groundnut production in several countries including China, Indonesia, Vietnam and Uganda. It has been estimated that 10% of peanut fields are infested with the bacterial wilt pathogen in China, with higher incidence in south and central regions (Jiang *et al.*, 2007). The bacteria enter roots through wounds made at planting, cultivation, or induced by nematodes, insects, and the emergence of secondary roots. *R. solanacearum* is also able to infect *M. truncatula*, where it can actively alter root tip structure and colonize root tip cortical cells (Turner *et al.*, 2009). Once inside the roots, the bacteria multiply in the xylem vessels, and block them with cells and slime causing the plant to wilt.

Bacterial wilt of alfalfa is caused by the Gram-positive bacterium *Clavibacter michiganensis* ssp. *insidiosus* that infects alfalfa plants directly through stem and root wounds. Alfalfa crops that are uniformly infected with the bacterium may show no other symptoms than general poor growth or stunting, although individual infected plants may have distorted leaves and spindly stems (Eichenlaub and Gartemann, 2011). Infected seed, produced on infected plants, serves as a particularly important inoculum source and is the main pathway by which the bacterium is introduced into new production areas (Cormack and Moffatt, 1956; Samac *et al.*, 1998).

For alfalfa, the causal agent of dwarfing is *Xylella fastidiosa* (Daugherty *et al.*, 2010). While not being a major pathogen of alfalfa, this bacterium, transmitted by several species of xylem sap-feeding insects, is able to infect dozens of agricultural, native, and weedy plant species, being responsible of Pierce's disease of grapevines.

G. Insects

Insects are important pests of legumes throughout the world, with the relative importance of insect pests compared to other biotic stresses depending on the crop species and location (Edwards and Singh, 2006). A major class of insect pests is chewing insects of which in the case of legumes the most important tend to be storage insect pests particularly bruchid beetles including *Callosobruchus chinensis*, *C. maculatus*, *C. analis*, *Acanthoscelides obtectus* and *Bruchus incarnates* (Keneni *et al.*, 2011). These pests can cause tremendous damage because of their high fertility and short generation times (Southgate, 1979). Seeds damaged by storage insect pests suffer from poor germination and are no longer suitable for food. Other types of chewing insect pests of legumes can attack the pod and cause considerable damage, for example, *Nezara viriolvla* in soybean (Brier *et al.*, 1991) or various *Helicoverpa* spp. such as *Helicoverpa armigera* (War *et al.*, 2013) which attack chickpea and pigeon pea (Acharjee *et al*., 2010) and *Maruca vitrata* on cowpea (Higgins *et al.*, 2012).

Another important class of pests is sap-sucking insects that include xylem- or phloem-feeding insects and cell content feeders. In legumes sap-sucking insect pests include aphids, whiteflies, mirids, leafhoppers and psyllids. Soybean aphid (*Aphis glycene*), cowpea aphid (*A. craccivora*), spotted alfalfa (*Therioaphis trifolii*), bluegreen aphid (*Acyrthosiphon kondoi*) and pea aphid (*Acyrthosiphon pisum*) are examples of major aphid pests in legumes (Kamphuis *et al.*, 2013a). In many cases these aphids cause damage both by direct feeding and through virus transmission. A good example of the economic threat of aphids is the soybean aphid, which was first detected in North America in 2000 and was estimated to have cost $1.6 billion by 2008 (Kim *et al.*, 2008).

H. Nematodes

Several parasitic nematode species infect most important crops including legumes causing around 12% yield losses annually. Among these species, the cyst and root-knot nematodes are the most important worldwide causing more than $90 billion losses each year (Dhandaydham *et al.*, 2008). In addition, the nematodes *Pratylenchus* spp. and *Ditylenchus dispsaci* can also cause significant damage in many legume crops.

Several cyst nematodes can infect legumes including *Heterodera glycine*, *Heterodera trifolii*, *Heterodera cajani* and *Heterodera goettingiana*. The soybean cyst nematode *H. glycine* is one of the most important pests limiting soybean production worldwide (Koenning *et al.*, 1999) leading to $430 million losses a year in the US (Wrather *et al.*, 1997). Beside soybean, this parasite can infect other legumes including common bean, pea, peanuts and pigeon pea but not the model legume *M. truncatula* (Koenning *et al.*, 1999; Dhanbaydham *et al.*, 2009). By contrast, *M. truncatula* can be infected by *H. trifolii*, the clover cyst nematode, which is the most common cyst nematode of North America and the principal cause of cyst nematode infection of forage legumes reducing white clover yield from 12 to 34% approximately (Dhandaydham *et al.*, 2008). *H. goettingiana* is also an important parasite of pea and faba bean. Although it has a somewhat more specialized host range, *H. goettingiana* can also infect lentil, vetch and *Lathyrus* species (Tedford and Inglis, 1999).

Root-knot nematodes (*Meloidogyne* spp.) are obligate sedentary endo-parasites occurring on a large range of climatic conditions. More than 70 *Meloidogyne* species have been described. However, more than 95% of plant infection is caused by only four species, *M. incognita*, *M. arenaria*, *M. javanica* and *M. hapla* (Sasser *et al.*, 1983). *M. arenaria* is one of the most important pests of peanuts which can also be infected by *M. javanica*, *M. hapla* and *M. haplanaria* (Eisenback *et al.*, 2003). *M. arenaria* is also an important parasite of subterranean clover in Australia (Barbetti *et al.*, 2007). *M.*

trifoliophila is another important clover root-knot nematode that can also infect additional legumes genera including *Medicago*, *Lotus* and *Glycine* (Bernard and Jennings, 1997). Pea and common bean can be infected not only by *M. incognita*, *M. arenaria*, *M. javanica* and *M. hapla* but also by the recently described new species *M. pisi* and *M. phaseoli* that cannot infect peanuts and soybean (Charchar *et al.*, 2008a, 2008b). Important damage to chickpea can also be induced by root infection with *M. artiellia* (Castillo *et al.*, 2003). On the other hand, legume infection by root-knot nematode is often associated with an increased incidence of fusarium wilt in these species (France and Abawa, 1994; Castillo *et al.*, 2003).

II. CURRENT UNDERSTANDING OF PLANT PATHOGEN INTERACTION IN LEGUMES

Plants have developed sophisticated systems to detect and respond against the attack of potentially pathogenic microorganisms. To suppress this defense, pathogens deliver effectors into host cells. However, plants can also detect these effectors and suppress their effects (Jones and Dangl, 2006). Resistance can be conferred by single, race-specific resistance genes (R genes), usually conferring complete resistance, or by a number of minor genes resulting in a broad-spectrum incomplete resistance. The identification of genes underlying resistance is challenging and a detailed understanding of the interaction between plants and their pathogens at the genetic, histological and molecular level could be helpful to reach this objective. Knowledge of the mechanism of resistance conferred by a gene can help in the identification of the gene. As a recent example, the penetration resistance to *Erysiphe pisi* observed in pea *er1* lines, similar to

that observed in *mlo* mutants of cereals and other crops, has been crucial to discover that the *er1* gene is a member of the MLO gene family (Humphry *et al.*, 2011). In cases of complex resistance caused by the action of several mechanisms, scoring these mechanisms in segregating populations can result in a better scoring of the resistance trait and the identification of the genes controlling each of these mechanisms. This approach has been used to identify QTLs associated with specific mechanisms of resistance to broomrape in pea (Fondevilla *et al.*, 2010) and to various aphids in *M. truncatula* (Guo *et al.*, 2012; Kamphuis *et al.*, 2013b), and is being also used in the *D. pinodes*-pea pathosystem (Carrillo *et al.*, 2013b). Knowledge on the genetics and the mechanism of resistance is also important for estimating the durability of the resistance. Hypersensitive isolate-specific resistance, governed by single genes, is usually easily overcome by the pathogens while resistance to penetration and complex polygenic resistance are expected to be more durable. In this section we will review the current knowledge about legume-pathogen interactions and inheritance of resistance.

A. Histology

Resistance is a multicomponent event, being the result of a battery of avoidance factors and/or resistance mechanisms acting at different levels of the infection process. This is more easily observed in biotrophic fungi, where host cells and tissues are maintained alive. In contrast, the fast cell death and tissue disorganization caused by necrotrophic fungi have hampered histological studies of these kinds of pathogens. As a result, there is a better knowledge of the resistance mechanisms acting at the cellular level in biotrophic fungi such as mildews and rust than in necrotrophic fungi such as ascochyta or botrytis. However, significant advances have been obtained in the

characterization of resistance mechanisms acting in response to most pests and pathogens.

In general the main steps of the infection process of a pathogen are the deposition of infection units, germination, penetration into the host and colonization of host tissue. Pre-penetration mechanisms do not seem to be relevant in the case of ascochytas and powdery mildews infecting legumes. In contrast, several mechanisms of rust exclusion may occur prior to stomatal penetration (Sillero *et al.*, 2006). These include poor germling adhesion to the leaf surface (Mendgen, 1978), deviating micromorphology of the epidermal surface or guard cell that serves as cues in guiding the thigmo-sensing germ tube towards stomata (Wynn, 1976; Sillero and Rubiales, 2002) and leaf pubescence (Mmbaga *et al.*, 1994). Reduced induction of germination of broomrape seeds and spores of the soil-borne fungi *F. oxysporum* and *F. solani* has also been reported (Buxton, 1960; Kraft, 1974; Stevenson *et al.*, 1995; Rubiales *et al.*, 2004; Fernández-Aparicio *et al.*, 2012).

Penetration resistance due to hampered haustoria formation (prehaustorial resistance) has been described against powdery mildew in pea (Fondevilla *et al.*, 2006) and *M. truncatula* (Prats *et al.*, 2007) and against rust in chickpea (Sillero *et al.*, 2012), faba bean (Sillero and Rubiales, 2002), grass pea (Vaz Patto *et al.*, 2009), lentil (Rubiales *et al.*, 2013a), pea (Barilli *et al.*, 2009a), and *M. truncatula* (Rubiales and Moral, 2004). In many instances histology has not been performed, but the reported quantitative resistance, based on reduced infection frequency, prolonged latent period and reduced uredia/colony size not associated with macroscopically visible host cell necrosis (Statler and McVey, 1987; Sillero *et al.*, 2000, 2012; Vaz Patto and Rubiales, 2009; Rubiales *et al.*, 2011, 2013b; Trabanco *et al.*, 2012; Leitão *et al.*, 2013) is likely due to this pre-haustorial resistance, which is known to play a major role in the so-

called partial resistance, and may be more durable than resistance controlled by R genes (Niks and Rubiales, 2002).

Pre-penetration resistance has also been reported against broomrape in chickpea (Rubiales *et al.*, 2003b), faba bean (Pérez-de-Luque *et al.*, 2007), lentil (Fernández-Aparicio *et al.*, 2008a), pea (Pérez-de-Luque *et al.*, 2006a), and *M. truncatula* (Lozano-Baena *et al.*, 2007), and against ascochyta blight (Carrillo *et al.*, 2013b) and *Fusarium oxysporum* in pea (Beckman, 1987; Benhamou *et al.*, 1996). Pre-infection resistance impeding penetration of root-knot nematode was also described in peanut (Bendezu and Starr, 2003).

Post-penetration mechanisms can also reduce or stop pathogen growth. Hypersensitive response is an effective mechanism against biotrophic pathogens including powdery mildew (Fondevilla *et al.*, 2007b) and rust of legumes (McLean and Byth, 1981; Rubiales and Sillero, 2003; Singh and Schwartz, 2010). The death of the pea epidermal cell under attack by *Didymella pinodes* has also been related to smaller lesion size. The presence of protein cross-linking was also associated with reduced lesion size in this pathosystem (Carrillo *et al.*, 2013b). While a hypersensitive-like response may exist to stop fusarium wilt progression, the main post-penetration mechanisms are the formation of chemical and physical barrier within vascular tissue by the means of phytoalexin accumulation, lignification and accumulation of gums, gels or tyloses within xylem cells (Beckman, 1987; Tessier *et al.*, 1990; Benhamou *et al.*, 1996). As a result of these complex defense reactions, *F. oxysporum* progression was efficiently blocked at crown level in resistant pea accessions (Bani *et al.*, 2012). Post-haustorial resistance, where attached parasites fail to develop, also occurs against parasitic weeds, being usually identified with the presence of a darkening (necrosis) of the tubercles parasitising the host (Goldwasser *et al.*, 1999; Lane *et al.*, 1993; Pérez-de-

Luque *et al.*, 2005). The host vessels at the infection point are filled with mucilage-like substances that block the normal flux of water and nutrients between the host and the parasite (Pérez-de-Luque *et al.*, 2006b). Several post-penetration mechanisms have been postulated in peanuts, soybean and alfalfa resistant to the root-knot nematodes *Meloidogyne arenaria* and *M. incognita* including the development of a hypersensitive-like reaction (Dhanbyadham *et al.*, 2008) and the production of repellent or inhibiting substances that impede the establishment of feeding sites by the invading nematode (Bendezu and Starr, 2003).

Histological studies have been used to document the deployment of structural defences to insect pests. For example, trichomes have been shown to provide resistance to leafhoppers in alfalfa (Shade *et al.*, 1979), whereas in cowpea resistance to weevils correlated strongly with the thickness of the seed coat (Kitch *et al.*, 1991).

B. Transcriptomics

The first step in defense is the recognition of the pathogen by the plant, which activates signal transduction cascades that subsequently trigger transcription of plant defense genes (Park *et al.*, 2008). Gene expression studies provide information about the genes and metabolic pathways differentially regulated during plant-pathogen interactions and contribute to the identification of candidate resistance genes involved in each of these steps of the defense response (Foster-Hartnett *et al.*, 2007; Uppalapati *et al.*, 2009; Fondevilla *et al.*, 2011c; Samac *et al.*, 2011; Rispail *et al.*, 2013). Knowledge of the genes involved in defense can be useful for marker assisted selection (MAS) and also to select genes whose altered expression through transformation can result in an increased resistance.

Molecular studies in legume-pathogen interactions have been hampered by the limited knowledge of gene sequences in legumes and their pathogens. Thus, the first studies on gene expression as a response to a pathogen attack or elicitors in legumes were performed with a small set of genes for which the sequences were available (Ichinose *et al.*, 2001; Matsui *et al.*, 2004; Gao *et al.*, 2007). To increase the potential number of defence-related genes, cDNA libraries were generated from plants inoculated with various pathogens or from elicitor-treated tissues or cells. For this purpose, *M. truncatula* and soybean, have been extensively used providing a wide range of EST collections (Ameline-Torregrosa *et al.*, 2006; http://soybean.ccgb.umn.edu/documents/soy libraries/index EST.html.; http://medicago.toulouse.inra.fr/Mt/EST/, http://www.tigr.org/webserver tmp/libtc tmp/24749 1100190119.libs.; http://compbio.dfci.harvard.edu/cgi-bin/tgi/gimain.pl?gudb=medicago). The sequencing of the model plant species *M. truncatula* was also a major breakthrough. Several macro- then microarray platforms including EST sequences and all the consensus sequences available so far were developed (Tesfay *et al.*, 2006; Küster *et al.*, 2007).

These tools have been used to study the interaction between *M. truncatula*, soybean or cowpea with several pathogens and pests (Iqbal *et al.*, 2002; Moy *et al.*, 2004; Torregrosa *et al.*, 2004; Zou *et al.*, 2005; Foster-Hartnett *et al.*, 2007; Dita *et al.*, 2009; Uppalapati *et al.*, 2009; Huan *et al.*, 2012; Kamphuis *et al.*, 2012b; Studham *et al.*, 2013). The high degree of sequence homology among legume species, especially for expressed sequences, enables cross-species application of macro-and micro-arrays. A microarray consisting of a set of chickpea unigenes, grass pea ESTs, and lentil resistance gene analogs (RGAs) was used to study resistance response in chickpea and lentil against ascochyta blight (Coram and Pang, 2006; Mustafa *et al.*, 2009). A *M.*

truncatula microarray has also been successfully applied to study gene expression profiling resistant pea interactions with *D. pinodes* (Fondevilla *et al.*, 2011c). In parallel to microarray approaches that require previous knowledge of genome sequence, alternative approaches including suppressive subtractive hybridization (SSH), cDNA-amplified fragment lengh polymorphisms (AFLP) have also been used to identify legume defensive genes. As a result, many studies performed on *M. truncatula*, soybean, pea, chickpea, peanuts and common bean provided insight into the molecular basis of their resistance to several legume pathogens including *Fusarium oxysporum*, *Aphanomyces euteiches*, *Phytophthora sojae*, *Didymella pinodes*, *Ascochyta rabiei*, *Phakopsora pachyrhizi*, *Peronospora viciae* f.sp. *pisi*, pea seed-borne mosaic virus, bean common mosaic virus, common bacterial blight, *Orobanche crenata*, *Phelipanche aegyptiaca*, *Heterodera goettingiana*, *Spodoptera exigua* or *Helicoverpa armigera* (Nyamsuren *et al.*, 2003; Gao *et al.*, 2004; Cho *et al.*, 2005; Cadle-Davidson and Jahn, 2006; Nimbalkar *et al.*, 2006; Die *et al.*, 2007; Choi *et al.*, 2008; Darwish *et al.*, 2008; Singh *et al.*, 2008; Gupta *et al.*, 2009, 2010; Hiraoka *et al.*, 2009; Soria-Guerra *et al.*, 2010; Shi *et al.*, 2011; Veronico *et al.*, 2011; Feng *et al.*, 2012; Huang *et al.*, 2012a,b; Jaiswal *et al.*, 2012; Xu *et al.*, 2012; Rispail *et al.*, 2013; Toyoda *et al.*, 2013).

In *M. truncatula*, a high-throughput quantitative RT-PCR platform for the analysis of transcription factor gene expression has also been established (Kakar *et al.*, 2008) and used to identify potential early regulators of resistance to aphids (Gao *et al.*, 2010), rust (Madrid *et al.*, 2010), and chocolate spot (Villegas-Fernández *et al.*, 2013).

The development of new and less expensive sequencing techniques is facilitating *de novo* sequencing of genomes and transcriptomes in non-model organisms at a relative low cost. Their application in legumes will allow the identification of the genes involved in defense in legumes crops themselves. The genome of the model legumes

Medicago truncatula (Young *et al.*, 2011) and *Lotus japonicus* (Sato *et al.*, 2008) and of chickpea, pigeon pea and soybean are already available (Kim *et al.*, 2010; Schmutz *et al.*, 2010; Varshney *et al.*, 2011, 2013; Singh *et al*,. 2013;). Chickpea, faba bean, lentil, lupin, pea, peanut, pigeonpea and soybean transcriptomes have also been reported (Le *et al.*, 2007; Severin *et al.*, 2010; Franssen *et al.*, 2011; Garg *et al.*, 2011; Hiremath *et al.*, 2011; Libault *et al*., 2011; Duan *et al.*, 2012; Kaur *et al.*, 2012; O'Rourke *et al.*, 2013; Sharpe *et al.*, 2013; Verma *et al.*, 2013).

Next generation sequencing techniques (NGS) can be used with reduced complexity transcriptome techniques such as SSH, cDNA-AFLP, SuperSAGE or MACE to increase tremendously the amount of transcripts identified compared to cloning and Sanger sequencing approaches. For instance, the combination of SuperSAGE with NGS has already been applied to study the molecular aspects of resistance to ascochyta blight in several legumes (Almeida *et al.*, 2013; Fondevilla *et al.*, 2013b; García *et al.*, 2013; Madrid *et al.*, 2013b). The combination of SSH with NGS has also started to be applied in soybean to study the genes involved during the symbiotic interaction (Barros de Carvalho *et al.*, 2013) and in response to drought (Rodrigues *et al.*, 2012; Oliveira-Vidal *et al.*, 2012) but have not yet been used to study disease and pest resistance. In addition, RNAseq technique that involves the sequencing of all transcripts expressed in a given situation is not only a powerful tool to develop *de novo* transcriptomes but also to compare the whole transcriptome expression profiling in two different situations. Moreover, in the case of plant-pathogen interactions it offers the added advantage of simultaneously studying changes in gene expression in both the plant host and the infecting pathogen/pest. RNAseq has been extensively used to study plant-pathogen interactions in other crops or in legumes for other traits (Bagnaresi *et al.*, 2012; Wang *et al.*, 2012; Gao *et al.*, 2013; Zhu *et al.*, 2013) but rarely to study plant-

pathogen interaction in legumes (Kim *et al.*, 2011). Recently it was used to study the transcriptome of soybean aphid (Liu *et al.*, 2012) and this should serve as a basis for studying changes in both plant and aphid genes during infestation. Genome-wide transcriptome profiling techniques yield a high number of genes that are differentially expressed after the pathogen attack; however, the challenge is how to discern which of them are really relevant in defense and responsible for the resistant phenotypes. Studying their co-localization with QTLs and performing functional analysis could be helpful but the generation and application of high-throughput reverse genetic platforms in legume crops will be critical.

Several approaches have been developed for the functional analysis of candidate genes at the biochemical and physiological level. Originally, this was performed through two main transformation based-techniques, protein over-expression and promoter activity studies. More recently, the development of gene silencing technologies using interference RNA (RNAi) or virus induced gene silencing (VIGS) have improved the efficiency of functional analysis of candidate genes (Karchoo *et al.*, 2008; Wesley *et al*., 2001). However, these approaches require efficient genetic transformation protocols which are difficult for most legumes limiting mostly their application to model legumes. Alternatively to these transformation-based methods, several mutation-based functional genomics approaches have been developed. These approaches including targeting induced local lesion in genome (TILLING) and deletion-TILLING (de-TILLING), are based on the identification of mutants carrying specific mutation in candidate genes from large mutant collections obtained by chemical mutagenesis, fast neutron mutagenesis or saturating *Tnt1*-insertion mutagenesis respectively (Tadege *et al*., 2008, 2009). In legumes, these approaches have been initially developed for the model legumes *M. truncatula*, *Lotus japonicus* and soybean

(Perry *et al.*, 2003; Cooper *et al.*, 2008; Le Signor *et al.*, 2009; Rodgers *et al.*, 2009) allowing the characterization of genes required for plant growth and symbiosis (Hoffmann *et al.*, 2007; Horst *et al.*, 2007; Welham *et al.*, 2009; Credali *et al.*, 2013). This approach was also applied to test the function of the major soybean cyst nematode resistance locus, *Rhg4*, in soybean resistance to nematode (Liu *et al.*, 2011) The high potential of this approach in breeding has driven the development of TILLING platforms for pea (Dalmais *et al.*, 2008), common bean (Porch *et al.*, 2009) and peanut (Knoll *et al.*, 2011). While this approach have not been applied to study disease resistance in legumes, it already allowed the characterization of gene important for seed development in pea (D'Erfurth *et al.*, 2012) and seed quality in peanut and soybean (Hoshino *et al.*, 2010; Knoll *et al.*, 2011) which may be very interesting for a breeding perspective. Interestingly, the TILLING approaches can also be used on a natural germplasm collection instead of a mutant collection to identify interesting natural variants that can be introgressed to crops (Wang *et al.*, 2012). This alternative approach, the so-called EcoTILLING have not yet been used for legume breeding but showed promising result to breed melon and *Capsicum* species for virus resistance (Nieto *et al.*, 2007; Ibiza *et al.*, 2010).

C. Proteomics

Protein expression and activity is the result of gene expression, post-transcriptional and post-translational regulations. Thus, large differences between mRNA levels and protein accumulation may exist due to post-transcriptional regulation and protein turn-over (Gygi *et al.*, 1999). As a consequence, profiling of gene expression alone may not be sufficient to comprehend important biological processes

including the resistance mechanisms to pathogens and pests, and this should be complemented by the study of protein accumulation. The recent technological developments have allowed the establishment of quantitative and qualitative methods for large-scale protein profiling (Cánovas *et al.*, 2004). Traditionally, proteomic approaches were based on the separation of proteins according to their isoelectric points and molecular weights by two-dimensional electrophoresis followed by their identification by mass spectrometry techniques including peptide mass fingerprinting or *de novo* sequencing. This method has been and still is the most used comparative proteomic approach. Alternatively, several methods that do not require gel electrophoresis have been developed using chromatography-based techniques for separation of peptide mixtures before their identification by mass spectrometry (Lee *et al.*, 2013a). Shotgun proteomics, in which the whole cell lysate is digested for extended chromatographic separations for direct tandem mass spectrometric analysis, is one of the most widely used gel-free approaches for protein identifications. More recently, second generation proteomics allowing multiplex comparison of proteins and their quantification such as DIGE and iTRAQ have been established although only limited application of these methods have been described so far (Jorrín *et al.*, 2007; Schenkluhn *et al.*, 2010; Abdallah *et al.*, 2012; Castillejo *et al.*, 2012; Qin *et al.*, 2013).

Application of these proteomic-based techniques is particularly useful for the identification of proteins involved in stress-responses in plants (Gygi and Aebersold, 2000). These methods have thus been widely used to study plant response to both biotic and abiotic stresses in many species although they mainly focused on non-legumes such as *A. thaliana* and rice (Cánovas *et al.*, 2004). In legumes, these methods have been initially applied to establish the reference protein maps and their subcellular localization (Watson *et al.*, 2003; Komatsu and Ahsan, 2009; Katam *et al.*, 2010; Lee *et al.*, 2013a),

the protein content of seed and their modification during seed development (Komatsu and Ahsan, 2009; Thompson *et al.*, 2009; Nautrup-Pedersen *et al.*, 2010) and to study the symbiotic interaction with both rhizobium and mycorrhizal fungi (Mathesius, 2009; Recorbet *et al.*, 2010; Salavati *et al.*, 2012). The plant responses to abiotic stresses such as drought or salinity have also been widely studied at the proteomic level in legumes including *M. truncatula*, common bean, soybean, pea, peanuts, grass pea and lupin (Kav *et al.*, 2004; Pinheiro *et al.*, 2005; Jain *et al.*, 2006; Kottapalli *et al.*, 2009; Chattopadhyay *et al.*, 2011; Hakeem *et al.*, 2012; Mohammadi *et al.*, 2012; Staudinger *et al.*, 2012; Komatsu *et al.*, 2013; Subba *et al.*, 2013; Zadraznik *et al.*, 2013). By contrast, the changes induced by pathogens and pests have been far less studied at the proteomic level. The proteome response of *M. truncatula* has been studied following infection by *Aphanomyces euteiches* (Colditz *et al.*, 2004; Trapphoff *et al.*, 2009; Schenkluhn *et al.*, 2010), *Uromyces striatus* (Castillejo *et al.*, 2010b), *Orobanche crenata* (Castillejo *et al.*, 2009), and in pea against *O. crenata* (Castillejo *et al.*, 2004, 2012), *Erysiphe pisi* (Curto *et al.*, 2006), *Didymella pinodes* (Castillejo *et al.*, 2010a), *U. pisi* (Barilli *et al.*, 2012), downy mildew (Amey *et al.*, 2008) and *Acyrthosiphon pisum* (Carrillo *et al.*, 2013a). In addition, the proteome response of chickpea - *Fusarium oxysporum* (Palomares-Rius *et al.*, 2011) and soybean - *Phytophtora sojae* (Subramanian *et al.*, 2009; Zhang *et al.*, 2011) interactions have been studied. These studies indicated that pathogens and pests induced important changes in protein profiles involving many cellular processes and that some were common to all stresses belonging to a general battery of plant defenses, such as proteases, ROS detoxifying ezymes and PR proteins. The modulation of accumulation of these common elements within plant cells may thus be interesting candidates for breeding once these large-scale analyses are validated by more targeted studies.

While actual comparative proteomic approaches may detect changes in many proteins in response to biotic stresses, most studies performed to date only identified a limited number of candidate resistance proteins. Protein identification is based on the comparison of peptide charge and molecular weight to that of observed or expected peptides stored in genomic and/or proteomic databases. Thus protein identification requires the existence of such databases that thus far are available only for a limited number of legumes such as *M. truncatula* and soybean. In these model legumes, between 90 and 100% of the differentially accumulated proteins can be identified (Schenkluhn *et al.*, 2010; Zhang *et al.*, 2011), while in other species such as pea and chickpea, the percentage of protein identifications drop down to 51 to 56%, respectively (Palomares-Rius *et al.*, 2011; Castillejo *et al.*, 2012). A legume specific protein database containing sequences from seven legume species has been recently created (LegProt: http://bioinfo.noble.org/manuscript-support/legumedb), which yield a significant increase in the overall identification success rate respect to other public databases such as NCBI (http://www.ncbi.nlm.nih.gov/pubmed) (Lei *et al.*, 2011). The increased application of genome and transcriptome sequencing is contributing in the building of large legume databases that represent valuable resources for future protein identification. Our increased capacity to identify differentially accumulated proteins coupled with the constant improvement in protein sample preparation and separation will surely increase the application of these methods to non-model legume crops. Application of these proteomic methods allowed identification of endogenous elements that play an important role in resistance to diseases. These key differentially accumulated proteins could be mapped to identify quantitative protein loci (Bourgeois *et al.*, 2011) and associated with molecular breeding to select for genotypes containing high level of these proteins. Alternatively, their level may be modified artificially by

genetic transformation or the recently developed intra- or cis-genesis approaches (Holme *et al.*, 2013).

However, limiting the study to the differential accumulation of proteins is not sufficient since many post-translational modifications such as nitrosylation, nitration, phosphorylation or ubiquitination may modulate their activity as a response to stresses. Proteomic-based approaches for the detection of these post-translational modifications have been recently developed and applied in legumes (Du *et al.*, 2009; Trapphoff *et al.*, 2009; Melo *et al.*, 2011; Maiti *et al.*, 2012; Nguyen *et al.*, 2012; Serna-Sanz *et al.*, 2012; Begara-Morales *et al.*, 2013; Camejo *et al.*, 2013; Signorelli *et al.*, 2013). Most studies performed on pea, peanuts, soybean and the model legumes *M. truncatula* and *L. japonicus* aimed to determine the post-translational modification induced during the symbiotic interactions with rhizobia (Melo *et al.*, 2011; Maiti *et al.*, 2012; Nguyen *et al.*, 2012; Serna-Sanz *et al.*, 2012; Signorelli *et al.*, 2013) or in response to abiotic stress (Ortega-Galisteo *et al.*, 2012; Begara-Morales *et al.*, 2013; Camejo *et al.*, 2013). Interestingly, one study also evaluated the phospho-proteome in response to the soil-borne pathogen *A. euteiches* in cell cultures of *M. truncatula* showing specific phosphorylation on several pathogenesis-related proteins and of the carbohydrate metabolisms between others (Trapphoff *et al.*, 2009). Future application of these methods coupled or not with a comparative proteomic approach to legume resistance will surely increase our understanding of the regulation of protein activity in response to biotic stresses that will be very valuable to identify candidate resistance proteins.

D. Metabolomics

Metabolites are the end products of the cellular machinery. They are the direct consequence of protein activity that in turn depends on the actual level of gene expression and protein accumulation after the integration of post-transcriptional and post-translational regulations (Fiehn, 2002; Sumner *et al.*, 2003). As such, their levels can be considered the ultimate response of biological systems to genetic or environmental changes. Metabolomics is a set of methods allowing parallel analysis of the cell metabolome that is all metabolites produced by an organism at a given time (Fiehn, 2002). Thus, metabolomics offer the direct estimation of the phenotypic response that cannot be assessed by transcriptomic or proteomic approaches (Sardans *et al.*, 2011). As a consequence metabolomics can be very useful for a cultivar of purposes from basic to applied research. From a scientific point of view, metabolomics can provide important breakthroughs to clarify pathways and their interconnections, to uncover mechanisms of interaction between metabolites and development and to decipher the cellular adaptation to stresses (Fernie and Schauer, 2009; Sardans *et al.*, 2011). It is thus an interesting strategy to study legume resistance to diseases and pests. In legumes, metabolomics have mainly been applied to shed light on symbiotic interactions (Akiyama *et al.*, 2005; Desbrosses *et al.*, 2005; Barsch *et al.*, 2006; Strack and Fester, 2006; Zhang *et al.*, 2012; Ye *et al.*, 2013) and their adaptation/tolerance to abiotic stresses including drought, salinity and nutrient stresses (Broeckling *et al.*, 2005; Pinheiro *et al.*, 2005; Hernandez *et al.*, 2007, 2009; Charlton *et al.*, 2008; Sanchez *et al.*, 2011a, 2012). By contrast, only one study applied a large-scale metabolomics approach to characterize *Phytophtora sojae* resistance in soybean (McGarvery and Pocs, 2006). Although large-scale, comprehensive metabolomic studies have only begun to be applied to study disease and pest resistance, many studies aimed to characterize a specific subset of metabolites in response to biotic stresses. Most of these studies

targeted the phenylpropanoid pathway that contains many defense related molecules which indicated a prominent role of flavonoid related compounds in the defense reaction of lupin, alfalfa, soybean, and *L. japonicus* (Baldridge *et al.*, 1998; Shimada *et al.*, 2000; Lozovaya *et al.*, 2004; Saunders and O'Neill, 2004; Lygin *et al.*, 2009; Muth *et al.*, 2009; Morkunas *et al.*, 2010; Wojakowska *et al.*, 2013) and several examples have demonstrated their potential to increase resistance levels to disease by genetic transformation (He and Dixon, 2000; Wu and VanEtten, 2004; Lozovaya *et al.*, 2005). In addition, several reports indicated the emission of volatile compounds in response to spider mites in *L. japonicus* and lima bean, and demonstrated an important role of these molecules as chemo attractant of predators and as defense inducer in neighboring plants (Ozawa *et al.*, 2000a, 2000b; Arimura *et al.*, 2002, 2004). Altogether these studies highlighted the importance of secondary metabolites as defense effectors in legumes.

Seeds of root parasitic weeds (*Orobanche*, *Phelipanche*, *Striga*, and *Alectra*) require chemical stimulation from the host root to germinate. Several classes of plant secondary metabolites are known to induce seed germination of root parasitic weeds (Fernández-Aparicio *et al.*, 2011b). Dozens of strigol-related compounds, collectively called strigolactones, have been identified as germination stimulants for root parasitic weeds (Xie *et al.*, 2010) that play a major role in host specificity (Fernández-Aparicio *et al.*, 2011c). Of them, orobanchol, orobanchyl acetate, 5-deoxystrigol and fabacyl acetate are widely distributed in the Fabaceae (Yoneyama *et al.*, 2008). In addition to these, new metabolites such as peagol, peagoldione and peapolyphenols have been indentified in pea root exudates and soyasapogenol B and trans-22-dehydrocampesterol in common vetch root exudates showing a selective stimulation of *Orobanche* seed germination (Evidente *et al.*, 2009, 2010, 2011).

In addition to improving our understanding of important cellular processes such as plant resistance to stresses, metabolomics is potentially a powerful tool for plant breeding. Indeed, it could allow rapid molecular phenotyping, which could be applied to estimate molecular variability in plant collections and to select individuals with a specific level of a determined metabolite or set of metabolites (Fernie and Schauer, 2009). In this sense, the different defense-related metabolites identified by targeted metabolomics in legumes in addition to being candidate resistance metabolites for direct introgression in crops, can also be ideal metabolite markers for selection. The high phenotypic resolution of metabolomics is also very attractive for diagnostic purposes and product quality testing (Fiehn, 2002). Thus, it is the method of choice to estimate potential "unintended effect" on plant yield or on the levels of other cellular metabolites arising from genetic improvement (Fernie and Schauer, 2009) and has been applied for quality testing of transgenics in several plant species including soybean (Garcia-Villalba *et al.*, 2008; Schmidt *et al.*, 2011) and pea (Charlton *et al.*, 2004).

While the use of specific metabolomics approaches alone can be very useful to identify metabolite markers for resistance or elucidate molecular pathways or metabolic fluxes in response to environmental stimulus, its potential may be substantially increased by integrating them with other strategies. For instance, the simultaneous application of metabolomics, transcriptomics and/or proteomics or the integration of these data in a common database may allow a better understanding of the regulatory networks of metabolic pathways and improve gene annotation (Dixon, 2001; Fernie and Schauer, 2009; Sardans *et al.*, 2011). In legumes, such integrative approaches have been used in *L. japonicus*, *M. truncatula* and common bean in response to rhizobium and abiotic stresses (Bell *et al.*, 2001; Desbrosses *et al.*, 2005; Hernandez *et al.*, 2009; Sanchez *et al.*, 2011b) yielding important information on cellular responses to these

stimuli. Combining metabolomics with other approaches could be useful for breeding. While metabolomics can complement for breeding as a tool for metabolite-based selection, its benefits would be much greater if used with existing approaches such as MAS and genetic transformation (Fernie and Schauer, 2009). Indeed, they can be associated with genetic mapping to identify pathway-based QTLs from a population or with genome-wide association studies that take into account genetic diversity in a large germplasm collection to obtain molecular and metabolic association with important traits. Similarly, integrating metabolomics as a metabolite-based selection approach with other omics technologies including the recent development of NGS platforms could speed up and improve the efficiency of the breeding process (Fernie and Schauer, 2009). Thus incorporation of metabolomic approaches in legume breeding programs may increase in the coming years as both a selection and validation tool for higher efficiency in breeding, and reduce the time required for production of new varieties.

E. Pathogenic factors

The hypersensitive response is an effective defense against biotrophic plant pathogens, restricting access to water and nutrients, but can be exploited by necrotrophic pathogens to generate dead tissue around the infected area (Govrin and Levine, 2000). Hemi-biotrophic pathogens utilize aspects of both biotrophic and necroptrophic infection strategies involving an initial bitotrophic phase and then switching to the necrotrohpic phase, producing toxins to kill the host cells and thus complete its life cycle on the dead tissue. Secreted phytotoxins contribute to virulence or pathogenicity by disrupting host cells and inducing the release of nutrients to facilitate colonization of host tissues (Berestetskiy, 2008). The hemi-biotrophic lentil

anthracnose pathogen, *Colletotrichum truncatum*, expresses an effector gene, *CtNUDIX*, exclusively during the later biotrophic phase before switching to necrotophic phase, and elicits a hypersensitive response from the host plant (Bhadauria *et al.*, 2011). Overexpression of *CtNUDIX* in the pathogen causes incompability with the host plant (Bhadauria *et al.*, 2013).

Necrotophic fungi are known to produce phytotoxic metabolites and peptides that are usually required for pathogenicity. Phytotoxins that affect a broad range of plant species are known as non-host-specific toxins (non-HSTs), whereas HSTs affect only a particular plant species or more often genotypes of that species (Stergiopoulos *et al.*, 2013). A wide range of secondary phytotoxic metabolites has been reported from legume pathogens. Ascochitine has been isolated from *Ascochyta fabae* and *A. pisi* (Foremska *et al.*, 1990; Beed *et al.*, 1994), ascosalitoxin from *A. pisi* (Evidente *et al.*, 1993), solanapyrones A-C from *A. rabiei* (Alam *et al.*, 1989; Chen and Strange, 1991), pinolidoxin, pinolide, and herbarumin II from *Didymella pinodes* (Cimmino *et al.*, 2012), lentisone from *A. lentis* (Andolfi *et al.*, 2013), botrytone from *Botrytis fabae* (Cimmino *et al.*, 2011). Those secondary metabolites often show phytotoxicity to host both host and non-host plants (Evidente *et al.*, 1993), and are frequently assumed to play a role in pathogenesis (Kaur, 1995). However, critical evidence is still lacking on the requirements of these toxins in causing diseases. Preliminary results using genetically defined toxin-deficient mutants showed that the phytotoxins solanapyrones produced by *Ascochyta rabiei* are not essential for ascochyta blight development. Nevertheless, all pathogenic isolates of *A. rabiei* produce the toxins. Thus, the toxins could play critical roles in competition and survival of the pathogen (Zerroug *et al.*, 2010).

Some preliminary progress has also been made on identifying potential effectors in aphids, including those attacking legumes (Hogenhout and Boss, 2011). Mass spectrometry of proteins in aphid saliva and salivary glands revealed a diversity of proteins, and some of these proteins appear to have effector functions (Harmel *et al.*, 2008; Carolan *et al.*, 2009; Nicholson *et al.*, 2012). Conserved proteins among insects, such as glucose oxidase (GOX), elicit plant defense responses (Tian *et al.*, 2012). RNAi has been used to show that specific salivary gland transcripts are essential for pea aphid feeding on plants (Mutti *et al.*, 2008). These studies provided evidence that aphid effectors are likely to suppress plant defense, modulate plant processes to aid aphid colonization, or may be recognized by specific plant resistance proteins, resulting in effector-triggered immunity (Hogenhout and Bos, 2011).

Extensive genome, transcriptome and proteome studies have shown that plant-parasitic nematodes secrete many additional effectors. Some are involved in suppression of plant defences, while others can specifically interact with plant signalling or hormone pathways to promote the formation of nematode feeding sites (Haegeman *et al.*, 2012). Like other plant pathogens, nematodes are thought to interfere with ubiquitin-proteasome (responsible for the selective degradation of proteins) pathway in the host cell, by secreting effectors involved in this pathway. Several such proteins have been described from *Heterodera glycines* (Gao *et al.*, 2003). The putative members of the ubiquitin–proteasome complex secreted into plant tissues might represent a mechanism of cellular regulation and mitigation of host defences to promote parasitism by nematodes (Davis *et al.*, 2004).

A variety of different effectors have been identified that may participate in manipulation of the auxin pathways, leading ultimately to a change in local auxin levels. The first effectors identified that may play a role in manipulating auxin levels were the

CMs, which may interfere locally with the hormone biosynthesis. The expression of *Meloidogyne javanica* CM in soybean hairy roots profoundly affects root formation and development of the vascular system (Doyle and Lambert, 2003). Other identified effectors could, theoretically, play a role in manipulating auxin levels, such as GSTs, an important detoxification enzymes that can also bind auxin and flavonoids in plants, modulating their trafficking in the cell (Moons, 2005). Nematode effectors can interfere with plant signalling pathways to either block, modulate or hijack cellular processes. One of the most striking examples of this process is the production by nematodes of proteins/peptides that mimic endogenous plant peptide hormones, such as cytokininsc found in *M. incognita* and *H. schachtii* secretions, which could stimulate the plant cells to activate the cell cycle, and hence promote the formation of giant cells and syncytia (De Meutter *et al.*, 2003).

Pseudomonas syringae, in particular pv *pisi*, is one of the few models that allowed us to understand gene-for-gene resistance in *Arabidopsis*, making these data paradigms for other plants. This phytopathogenic bacterium can suppress both pathogen-associated molecular pattern-triggered immunity and effector-triggered immunity by injection of type III effector proteins into host cells (Block and Alfano, 2011). The type III secretion system is a protein secretion apparatus used by animal and plant pathogens or mutualists to deliver T3E virulence proteins directly into host cells, where they can modulate the host's physiology and manipulate the host immune system. Strains of *P. syringae* pv. *syringae* secrete syringolin A, a product of a mixed non-ribosomal peptide/polyketide synthetase, that has been identified as a virulence factor (Groll *et al.*, 2008). Some of these effectors are able to elicit resistance and are thus called avirulence proteins (Avr). They have various enzymatic activities including cysteine proteases, mono-ADP-ribosyltransferases, phosphothreonine lyase, E3 ligase,

and protein tyrosine phosphatase (Block *et al.*, 2008). Several pathogenic factors required for *Ralstonia solanacearum* to infect *M. truncatula* susceptible lines have been described (Turner *et al.*, 2009). The infection process requires two type III effectors, Gala7 and AvrA, which are involved at different stages of infection. Whereas the Gala7 effector is necessary for bacterial propagation in the vessels and wilting development, the AvrA effector is required for the first steps of infection: root growth arrest, root tip swelling, and epidermal cell death followed by bacterial cortex colonization and vessel colonization (Turner *et al.*, 2009). To date, only a few effectors from *R. solanacearum* have been shown to have pathogenicity functions. Gala T3Es are the best characterized; they have an F-box and a Leu-rich repeat domain and interact with ASK (part of the host ubiquitin/proteasome pathway) proteins in Arabidopsis to promote disease (Angot *et al.*, 2006).

A number of proteins termed nodulation outer proteins (Nops) are delivered by the pathogen via the type III secretion system (Marie *et al.*, 2003). This type of protiens has also been found in *Rhizobium* species. In interactions with some hosts Nops act somewhat like virulence factors, while in other potential hosts they appear to act like avirulence factors by triggering a rapid defense response against the pathogen. To investigate the function of NopL, Bartsev *et al.* (2004) expressed nopL in tobacco plants. When these plants were inoculated with Potato virus Y, expression of the defense proteins class I chitinase and β-1,3-glucanase was suppressed and virus accumulation was enhanced. Similarly, expression of nopL in transgenic *Lotus japonicus* plants also suppressed accumulation of class I chitinase. The demonstration that NopL is a repressor of plant defense reactions suggests that invading rhizobia can manipulate metabolic pathways of their hosts using mechanisms that are common to pathogens.

III. ADVANCES AND CHALLENGES FOR EXPLOITATION OF SOURCES OF RESISTANCE IN LEGUME BREEDING

A. Sources of resistance and their utilization in breeding

The success in developing varieties resistant to biotic stresses depends on the availability of good sources of resistance, the availability of an accurate method to score the disease and also on the inheritance of the resistance. While complete or high levels of resistance conferred by single genes can be easily introduced into varieties by backcrossing, the introgression of quantitative resistance governed by several minor genes/QTLs is challenging. In monogenic resistances the individuals carrying the gene are easily identified in breeding populations while quantitative polygenic traits are usually highly influenced by the environment and the genotype cannot be directly inferred from the phenotype. As a result, most resistant varieties available so far are based on major genes although some successes have been obtained in some polygenic resistances.

Complete monogenic resistance has been reported in common bean and soybean against rusts with closely linked markers identified that are readily used in marker assisted backcrossing (Faleiro *et al.*, 2004; Miklas *et al.*, 2006; Hyten *et al.*, 2007; García *et al.*, 2008). By contrast, most of the rust resistance reactions described so far in most cool season legumes are incomplete, with the genetic basis of these resistances largely unknown in most cases. In faba bean the most frequently reported resistance against *U. viciae-fabae* is incomplete non-hypersensitive resistance (Sillero *et al.*, 2000, 2006, 2010). Mapping studies have been initiated to identify QTLs and to develop

molecular markers useful in MAS for the resistance (Torres *et al.*, 2006), but no results are available yet. On the other hand, the recently identified hypersensitive resistance is controlled by genes with major effects (Sillero *et al.*, 2000). Different genes might be available in the different accessions displaying hypersensitivity reported so far (Sillero *et al.*, 2000). However, genetic analysis only been performed with one of them allowing the identification of markers linked to a resistance gene (*Uvf-1*) (Avila *et al.*, 2003).

In pea only incomplete resistance has been described against both *U. viciae-fabae* (Chand *et al.*, 2006) and *U. pisi* (Barilli *et al.*, 2009a, b). Partial resistance to *U. viciae-fabae* has been assigned to a single major gene (*Ruf*) (Vijayalakshmi *et al.*, 2005). Similarly, a QTL explaining 63% of the resistance to *U. pisi* has been identified flanked by 2 RAPDs (Random Amplification of Polymorphic DNA) (Barilli *et al.*, 2010). However, in both cases, these markers were not close enough to allow a reliable MAS approach for rust resistance.

Lentil resistance against *U. viciae-fabae* has been reported mainly as partial resistance, although some hypersensitive resistant sources have also been described (Negussie *et al.*, 2005; Rubiales *et al.*, 2013a). Monogenic resistance has been described (Erskine *et al.*, 1993) and preliminary information on chromosome location and associated molecular markers is being produced. Similarly, only incomplete resistance was identified in chickpea against *U. ciceris-arietini* (Sillero *et al.*, 2012). A QTL explaining 81% of the resistance has been described and assigned hypothetically to a single gene (Madrid *et al.*, 2008). In peanut, genetic studies indicated that resistance to the rust fungus *Puccinia arachidis* is complex and polygenic, with 12 QTLs reported (Khedikar *et al.*, 2010). Resistance to rust has also been identified in *Lathyrus sativus* (Vaz Patto and Rubiales, 2009) and *L. cicera* (Vaz Patto *et al.*, 2009) germplasm but

genetic studies have only recently been initiated by generation of proper mapping populations (unpublished).

Complete or high level of resistance to *E. pisi*, conferred by the genes *er1*, *er2* or *Er3* is available in pea (Harland, 1948; Heringa *et al.*, 1969; Fondevilla *et al.*, 2007a, 2011b). In addition, lines with different levels of incomplete resistance to powdery mildew have been reported in pea and wild relatives (Pal *et al.*, 1980; Sharma, 1992; Dang *et al.*, 1994; Thakur *et al.*, 1996; Fondevilla *et al.*, 2007a). However, most pea cultivars resistant to *E. pisi* developed so far relies on the complete resistance conferred by gene *er1*. A recent study indicates that resistance provided by *er1* is due to a loss of function of PsMLO1, a MLO (Mildew Resistance Locus O) gene (Humphry *et al.*, 2011). However, resistance conferred by *er1* is not operative against *E. trifolii* infecting peas whereas *er2* is (Fondevilla *et al.*, 2013a). Molecular markers linked to these three genes in coupling and repulsion phase are available (Fondevilla *et al.*, 2008a; Katoch *et al.*, 2010; Pereira *et al.*, 2010). The first pea cultivar with gene *Er3* has been recently developed. Resistance to powdery mildew (*E. pisi*) in *M. truncatula* also involved three distinct loci (Ameline-Torregrosa *et al.*, 2008).

Partial resistance to *Peronospora viciae* has been reported in faba bean (Thomas and Kenyon, 2004) although there is no information on the sources of resistance or its genetic control. Both race-specific (Thomas *et al.*, 1999) and partial resistance (Stegmark, 1994) have been reported in pea. A combination of dominant, recessive and intermediate resistance genes are involved (Stegmark, 1992).

The low levels of resistance identified so far against ascochyta blights and the polygenic nature of resistance has hampered the release of legume varieties highly resistant to these diseases (Rubiales and Fondevilla, 2012). This task has also been hampered by the presence of the higher levels of resistance in wild types with

undesirable agronomic traits. Resistance to *Ascochyta pisi* in pea is controlled by a major gene modified by minor genes or QTLs (Darby *et al.*, 1985; Dirlewanger *et al.*, 1994). Some levels of incomplete resistance against *Didymella pinodes* and *Phoma medicaginis* have been reported (Ali *et al.*, 1978; Kraft, 1998; Wroth, 1998; Prioul *et al.*, 2003; Fondevilla *et al.*, 2005). The majority of the genetic studies concerning resistance to *D. pinodes* in pea have concluded that resistance is a polygenic trait (Fondevilla *et al.*, 2008a) associated with numerous QTLs explaining from low to moderate percentage of the variation of the trait (Timmerman-Vaughan *et al.*, 2002; Tar'an *et al.*, 2003; Prioul *et al.*, 2004; Fondevilla *et al.*, 2008b, 2011a). Resistance to *D. pinodes* in *M. truncatula* is also quantitative (Madrid *et al.*, 2013a). By contrast, the resistance of *M. truncatula* to two races of *Colletotrichum trifolii* is mainly governed by a single major locus (Ameline-Torregrosa *et al.*, 2008).

Incomplete resistance to *Didymella lentis* is also available in lentil germplasm. Single genes, either dominant (Ford *et al.*, 1999) or recessive (Chowdhury *et al.*, 2001) have been reported together with molecular markers flanking the resistance genes. In addition, at least five QTL for blight resistance have been mapped that together accounted for 50% of phenotypic variation (Rubeena *et al.*, 2006). Incomplete resistance to *Didymella rabiei* is available in chickpea germplasm (Reddy and Singh, 1984; Singh and Reddy, 1994) and is being used in breeding programs. Early inheritance studies concluded that resistance could be controlled by one, two or three genes (Singh and Reddy, 1993; Collard *et al.*, 2001; Chen *et al.*, 2004). More recently, two major QTLs have been identified (Santra *et al.*, 2000; Tekeoglu *et al.*, 2002; Collard *et al.*, 2003; Rakshit *et al.*, 2003; Udupa and Baum, 2003; Cho *et al.*, 2004; Iruela *et al.*, 2006). Incomplete levels of resistance to *Didymella fabae* have been identified in faba bean germplasm (Sillero *et al.*, 2001; Tivoli *et al.*, 2006; Rubiales *et*

al., 2012) that are being used in breeding programmes to develop improved cultivars. Both major gene (Rashid *et al.*, 1991; Kohpina *et al.*, 2000; Kharrat *et al.*, 2006) and polygenic inheritance (Román *et al.*, 2003; Avila *et al.*, 2004) have been reported.

Incomplete resistance to *Botrytis fabae* has been reported in faba bean germplasm (Bond *et al.*, 1994; Hanounik and Robertson, 1988; Villegas *et al.*, 2009, 2011, 2012). No QTLs or associated molecular markers have been reported so far, although molecular characterization of the resistance is in progress (Villegas *et al.*, 2013).

With the exception of phytophthora root rot for which both monogenic race-specific resistance and quantitative resistance have been described (Lee *et al.*, 2013c; Zhang *et al.*, 2013), resistance to root rot is exclusively quantitative and of complex inheritance (Montoya *et al.*, 1997; Grunwald *et al.*, 2003; Roman-Aviles and Kelly, 2005; Zhao *et al.*, 2005, 2009; Ellis *et al.*, 2013; Hamon *et al.*, 2013). Resistance to *Fusarium solani* f.sp. *pisi* in pea is quantitatively inherited. A number of QTLs and their associated markers have been identified (Coyne and Pilet-Nayel, 2008). Genetic resistance to *Aphanomyces euteiches* in pea is known to be quantitative and largely influenced by interactions with environmental conditions (Shehata *et al.*, 1983). Up to 7 QTLs have been associated with partial field resistance (Pilet-Nayel *et al.*, 2002, 2005) and a major QTL has been described for the root response in *M. truncatula* (Djébali *et al.*, 2009). In the case of *Rhizoctonia solani*, moderate resistance to some anastomosis groups has been found in *M. truncatula* (Anderson *et al.*, 2013).

Resistance to *Fusarium oxysporum* has been identified in many legume crops. In most cases, resistance was found to be mono- or oligogenic although quantitative resistance has also been described in pea, lentil and common bean (Hijano *et al.*, 1983; Salgado *et al.*, 1995; Sharma and Muehlbauer, 2007; Bani *et al.*, 2012). Race-specific

resistance to *F. oxysporum* f.sp. *pisi* is controlled by single dominant genes for some of which markers are available (McPhee *et al.*, 1999; Grajal-Martin and Muehlbauer, 2002; McClendon *et al.*, 2002; Okubara *et al.*, 2002). Resistance to verticillium wilt is quantitative (Pennypacker, 2000; Vandemark *et al.*, 2006; Ben *et al.*, 2013b). In *M. truncatula*, resistance to verticillium wilt caused by *Verticillium albo-atrum/V. alfalfae* is controlled by several QTLs (Ben *et al.*, 2013b; Negahi *et al.*, 2013a). Resistance to *Sclerotinia trifoliorum* in faba bean has been shown to be governed by a single dominant gene (Lithourgidis *et al.*, 2005).

Resistance against broomrapes in legumes is difficult to access, scarce, of complex nature and of low heritability, making breeding for resistance a difficult task (Rubiales, 2003; Rubiales *et al.*, 2006; Pérez-de-Luque *et al.*, 2009). Still, a number of cultivars with various levels of resistance to *Oorbanche crenata* and/or *O. foetida* have been released by different faba bean breeding programs (Kharrat *et al.*, 2010; Pérez-de-Luque *et al.*, 2010; Maalouf *et al.*, 2011). Little resistance was available within pea germplasm against *O. crenata* (Rubiales *et al.*, 2003a), but was identified in *Pisum* spp. (Pérez-de-Luque *et al.*, 2005; Rubiales *et al.*, 2005), which were successfully hybridized with cultivated pea (Rubiales *et al.*, 2009) resulting in the recent release of the first resistant cultivars. Resistance in lentil has only recently been reported (Fernández-Aparicio *et al.*, 2008a, 2009b). Resistance is also very limited in *Lathyrus sativus* (Fernández-Aparicio *et al.*, 2011a) and *L. cicera* (Fernández-Aparicio *et al.*, 2009a). However, resistance is frequent in common vetch and chickpea germplasm and cultivars (Gil *et al.*, 1987; Rubiales *et al.*, 2003b; Fernández-Aparicio *et al.*, 2008b) as well as in their wild relatives (Rubiales *et al.*, 2004, 2005; Sillero *et al.*, 2005). Resistance to *Phelipanche aegyptiaca* has been found in purple vetch (*Vicia atropurpurea*) (Goldwasser *et al.*, 1999). Resistance to *Striga gesnerioides* and *Alectra vogueli* has

been identified in cowpea, resulting in the release of resistant cultivars (Singh and Emechebe, 1990; Singh *et al.*, 1993). A gene-for-gene interaction has been demonstrated in *S. gesnerioides*-cowpea interaction (Li and Timko, 2009) and molecular markers associated with race-specific resistance genes have been identified, and several sequence-confirmed amplified regions (SCARs) have been developed (Li *et al.*, 2009).

Good progress has been made in legumes in identifying and characterising genetic resistance to aphids, particularly in *M. truncatula* and soybean (Kamphuis *et al.*, 2013b). Thus in *M. truncatula*, strong resistance to bluegreen, spotted alfalfa aphid and different pea aphid biotypes, has been identified and shown to be mediated by single dominant genes (Klingler *et al.*, 2005, 2007; Gao *et al.*, 2008; Stewart *et al.*, 2009). In addition, QTLs underlying more moderate forms of resistance to cowpea, spotted alfalfa, bluegreen and pea aphids have also been identified (Klingler *et al.*, 2009; Guo *et al.*, 2012; Kamphuis *et al.*, 2012a, 2013b). In soybean a number of single dominant resistance genes, that provide strong resistance to specific biotypes of soybean aphid, have been identified (Hill *et al.*, 2004; Mian *et al.*, 2008; Zhang *et al.*, 2010; Jun *et al.*, 2012). In addition two QTLs mediating resistance to soybean aphid have also been identified (Zhang *et al.*, 2009).

In many cases, genetic resistance to key chewing insect pests has been hard to find (Edwards and Singh, 2006). However, screening of wild accessions has yielded some success. For instance, effective resistance to the Mexican bean weevil mediated by a single dominant gene has been found in wild lines of bean (van Schoonhoven *et al.*, 1983) and resistance to pod fly and pod wasp has been found in wild relatives of pigeon pea (Sharma *et al.*, 2003). In addition, various QTLs that mediate some degree of resistance to various chewing insects have been identified (Yesudas *et al.*, 2010; Xin *et*

al., 2012). There is little resistance to *Bruchus pisorum* in pea germplasm. The highest resistance has been found in *Pisum fulvum* (Byrne *et al.*, 2008). A line of pea exhibiting the 'Neoplastic pod' wild allele, that enables the formation of callus tissue in response to the presence of *B. pisorum* eggs on pods, resulting in a reduced larval survival, has also been reported (Berdnikov *et al.*, 1992).

Resistance against bacterial wilt incited by *Ralstonia solanacearum* is considered essential in peanut (Jiang *et al.*, 2007). Althought available resistant cultivars have relatively low yields with poor resistance or tolerance to other constraints, such as foliar diseases and drought, there is prospect for exploitation of these sources of resistance to breed for bacterial wilt resistance (Jiang *et al.*, 2013). Resistance to *R. solanacearum* in *M. truncatula* is governed by major QTL located on chromosome 5 (Vailleau *et al.*, 2007). Comparison of parental line sequences revealed 15 candidate genes with sequence polymorphisms, but no evidence of differential gene expression upon infection. These data indicate that the quantitative resistance to bacterial wilt, which contains a cluster of seven R genes, is shared by different accessions and may act through intralocus interactions to promote resistance (Ben *et al.*, 2013a)

Resistances to bacterial blights and bacterial wilts are usually considered as quantitative in the allogamous tetraploid lucerne (Li and Brummer, 2012). Resistance to *Pseudomonas syringae* pv. *phaseolicola* is one of the best underdstood systems todate, in different species. *P. syringae* effectors AvrB and AvrRpm1 are recognized by phylogenetically distinct resistance nucleotide-binding site and leucine-rich repeats (NB-LRR) proteins in *Arabidopsis* (Mackey *et al.*, 2002; Belkhadir *et al.*, 2004) and soybean (Ashfield *et al.*, 2003). By screening common bean genotypes of various geographical origins, it was shown that no genotypes recognized AvrB. By contrast,

multiple genotypes responded to AvrRpm1, and two independent R genes conferring AvrRpm1-specific resistance were mapped (Chen *et al.*, 2010). Race-specific resistance to *Pseudomonas syringae* pv. *pisi* controlled by single dominant genes has been reported in pea (Bevan *et al.*, 1995; Hunter *et al.*, 2001). Race non-specific resistance has also been reported (Schmit *et al.*, 1993). *Pisum abyssinicum* accessions are resistant or partially resistant to all races including race 6, for which there are no known commercial resistant cultivars. This resistance is controlled by a major recessive gene together with a number of modifiers (Elvira-Recuenco *et al.*, 2001).

Resistance to pea seed-borne mosaic virus has been reported in pea and several recessive resistance genes have been identified (Hagedorn and Gritton, 1973; Khetarpal *et al.*, 1990). Molecular markers associated with the resistance gene are now available (Gao *et al.*, 2004). Resistances to pea enation mosaic virus, white lupin mosaic virus and bean yellow mosaic virus have also been reported (Provvidenti and Hampton, 1993; Yu *et al.*, 1995, 1996). Resistances to bean yellow mosaic virus and bean leaf roll virus have been identified in faba bean (Gadh and Bernier, 1984; Bond *et al.*, 1994; Kumari and Makkouk, 2003).

Resistance to the nematodes *Ditylenchus dipsaci*, *Pratylenchus thornei* and *P. neglectus* has been reported in faba bean (Hanounik *et al.*, 1986; Di Vito *et al.*, 2001) although no information is so far available on the genetics of resistance. Resistance to *P. neglectus* was also described in alfalfa (Baldridge *et al.*, 1998). Resistance to root-knot and cyst nematodes has been identified in many legume species. Monogenic resistance to *M. hapla* and *M. arenaria* has been described in alfalfa and peanut respectively (Burow *et al.*, 2001; McCord, 2012). By contrast, several sources of quantitative resistance have been found in soybean against *M. arenaria*, *M. incognita* and the soybean cyst nematode *H. glycine* and incorporated into breeding programs

(Luzzi *et al.*, 1994; Davis *et al.*, 2009; Fourie *et al.*, 2013). Resistance to *Heterodera glycine* was also found in common bean cultivars and in wild relative species of *Vigna angularis* (Smith and Young, 2003; Kushida *et al.*, 2013). Resistance to *H. ciceri*, was not found in chickpea and lentil although resistance was detected in several wild relative of chickpea (Erskine *et al.*, 1993; Di Vito *et al.*, 1996). Resistance was also described in pea against its cyst nematode *H. goettingiana* (Veronico *et al.*, 2011).

B. Advances and challenges for marker-assisted breeding

The identification of molecular markers linked to the resistance genes, or if possible, located within the genes, could be used to replace or reduce screening tests in breeding populations, thus reducing the time and effort required to develop new cultivars. Molecular markers can be used to detect the individuals carrying resistance genes at seedling stages and avoid tedious inoculations and screenings. Molecular markers can also be also used for pyramiding several genes conferring resistance to a disease, in order to increase the durability of the resistance conferred by each individual gene, or to combine genes conferring resistance to different diseases in the same cultivar.

To identify molecular markers linked to major genes the development of a complete map of the species is not needed. A simple technique called Bulked Segregant Analysis (BSA) (Michelmore *et al.*, 1991) can be used. This technique consists of creating two bulks, each containing homozygous individuals for each of the alleles of the gene, and identifying molecular markers polymorphic between these bulks. Alternatively, near isogenic lines differing for the gene of interest can be used. BSA has been successfully used to identify molecular markers linked to resistance genes in legumes such as resistance genes for powdery mildew in pea (Timmerman-Vaughan *et*

al., 1994; Tiwari *et al.*, 1998; Janila and Sharma, 2004; Fondevilla *et al.*, 2008a), rust in faba bean (Avila *et al.*, 2003), fusarium wilt and ascochyta blight in lentil (Ford *et al.*, 1999; Chowdhury *et al.*, 2001; Eujayl *et al.*, 2008), anthracnose, bacterial blight and rust in common bean (Bai *et al.*, 1997; Young *et al.*, 1997; Alzate-Marin *et al.*, 1999a; Park *et al.*, 1999; Faleiro *et al.*, 2000) and frogeye leaf spot in soybean (Mian *et al.*, 1999), among others.

In the case of quantitative traits controlled by several genes with minor effects the identification of the genes controlling the trait is a difficult task by traditional approaches. The development of QTL analysis was a major breakthrough in the characterisation of the inheritance of quantitative traits, enabling the identification of important genomic regions and their contribution to phenotypic variation. In addition, the mapping of QTLs is a useful tool to identify molecular markers linked to the resistance genes that could be used to assist breeding or to identify the gene itself.

Many studies have been carried out to identify molecular markers linked to genes/QTLs controlling resistance to diseases and pest in legumes. However, only in some cases where major genes or QTLs have been detected, have molecular markers been used to assist breeding. Those include resistance to pests and soybean mosaic virus in soybean (Walker *et al.*, 2002, 2004; Shi *et al.*, 2009), bacterial blight, rust, virus, anthracnose, angular leaf spot, white mould and root rot in common bean (Alzate-Marin *et al.*, 1999b; Stavely, 2000; Yu *et al.*, 2000; de Oliveira *et al.*, 2005; Ender *et al.*, 2008; Navarro *et al.*, 2009) and powdery mildew in pea (for the development of pea cultivar Eritreo carrying *Er3* gene). The efficiency of a marker in MAS depends on the distance between the flanking markers and the target gene that estimates the number of expected recombinants between the gene and the marker. In legumes the distance between the

markers and the gene/QTL controlling resistance are in most cases still too large for efficient MAS.

The accuracy of a QTL analysis is highly influenced by the number of individuals forming the segregating population, the accuracy in scoring the trait and the availability of highly dense maps. The first maps in legumes were done with morphological markers, isozymes and a few DNA markers containing mainly anonymous, low reproducible markers such as RAPDs or RFLPs (Restriction Fragment Length Polymorphism) (Torres *et al.*, 1993; Simon and Muehlbauer, 1997; Weeden *et al.*, 1998; Chowdhury and Slinkard, 1999; Rubeena *et al.*, 2003). The use of more reproducible molecular markers such as SSRs (Simple Sequence Repeats) is increasing the quality of the maps and allowing comparison between maps (Pozarkova *et al.*, 2002; Loridon *et al.*, 2005; Millán *et al.*, 2010; Fondevilla *et al.*, 2011a; Yang *et al.*, 2012). Some of the mapped SSRs are located within genes but there is still a need for increasing the saturation of the existing legumes maps with gene-based markers. The de novo assembly of transcriptomes using NGS techniques is allowing the massive identification of SSRs located within genes of interest. As a result, more than 2,000 loci containing repetitive elements have been identified in the pea transcriptome and more than 1,000 in the faba bean transcriptome (Kaur *et al.*, 2011), although they are still not included in genetic maps.

NGS techniques are also allowing the massive identification of gene-based SNPs (Single Nucleotide Polymorphisms). The development of high-throughput genotyping techniques for SNPs such as IlluminaGoldenGate assay, KASPar, Affymetrix SNP array or High Resolution Melting Curve is accelerating the introgression of these kinds of markers in genetic maps. Illumina GoldenGate assays are available for pea (Deulvot *et al.*, 2010), chickpea (Gaur *et al.*, 2012), common bean (Blair *et al.*, 2013), cowpea

(Muchero *et al.*, 2013) and lentil (Sharpe *et al.*, 2013) as well as a KASPar assays for faba bean (Cottage *et al.*, 2012). However, more effort is needed to increase the number of SNPs mapped especially for genes involved in defence. Mapping of defence related genes can be helpful for identifying candidate genes through their co-localization with QTLs as has been done in the case of ascochyta blight resistance in pea (Timmerman-Vaughan *et al.*, 2002; Prioul-Gervais *et al.*, 2007). Coding regions are also the best-conserved sequences in different species and therefore gene-based markers are the most suitable for comparative mapping. *M. truncatula* has a high level of synteny and homology with legume crops (Choi *et al.*, 2004; Phan *et al.*, 2007). The identification of the homologous region of a QTL in the model legume *M. truncatula* through microsynteny is a good approach to identify candidate genes involved in the control of resistance, or at least molecular markers more tightly linked with resistance, in species with less saturated maps. This approach has been used for example in resistance to ascochyta blight in chickpea (Madrid *et al.*, 2012), for identifying in pea the *SYM2* gene involved in symbiosis (Gualtieri *et al.*, 2002) and in resistance to broomrape (Cobos *et al.*, 2013).

QTLs detected in one particular environment or population may be useful for a specific condition or cross. However, the most promising QTLs for MAS are those that are stable in different environments and genetic backgrounds. To identify common genomic regions controlling a trait, the QTLs identified for the trait in different crosses by traditional QTL linkage mapping using bi-parental populations can be compared. This approach has been used for example in pea for identifying common genomic regions controlling resistance to ascochyta blight, earliness and architectural traits in different pea maps (Fondevilla *et al.*, 2011c). However to discern whether a QTL is really the same as other identified in a different population a set of robust common

anchored markers are needed to compare accurately the different maps. SNPs and SSR markers are suitable markers for this approach. Another interesting strategy to identify molecular markers linked to a trait and not specific for a particular segregating population is emerging through association mapping (AM). In AM approach, based on linkage disequilibrium, a germplasm collection is genotyped with densely distributed genetic marker loci covering all chromosomes and evaluated for one or several traits. Analysing the association between the markers profiles and the phenotypes, molecular markers associated with the traits can be identified. However, in order to avoid false positives the structure of the population used needs to be well known and taken into account (Rafalski, 2010). Recently an AM approach was performed in cowpea to identify loci controlling delayed senescence, biomass and grain yield under drought stress (Muchero *et al.*, 2013). The advances in the development of high-throughput genotyping techniques for SNPs or DArT (Diversity Arrays Technology) markers and the development of germplasm core collections offers now the opportunity of developing genome wide association mapping not only to find associations between traits and DNA polymorphisms but also to unravel the origin of genetic correlations among phenotypic traits, that is, pleiotropy versus genetically linked genes (Zhao *et al.*, 2011).

C. *In vitro*-based breeding approaches

Tissue culture and its derived *in vitro* approaches are an important complement to traditional breeding methods. *In vitro* culture techniques offer not only tools for multiplication and maintenance of improved plant materials but also to increase and screen the genetic variability available either directly through somaclonal variation and mutagenesis or indirectly by overcoming incompatibility barriers allowing wide

hybridization outside the sexual compatibility barrier boundaries (Svetleva *et al.*, 2003). As such, these methods have potential for breeding. However, all these methods are dependent on efficient *in vitro* regeneration procedures to recover fertile plants from tissue explants or isolated protoplasts, which have proven to be arduous in legumes (Choudhary *et al.*, 2009). Indeed, most legumes are recalcitrant to regeneration from both organogenesis and embryogenesis (Anand *et al.*, 2001; Chandra and Pental, 2003). This recalcitrance is the main bottleneck against the routine production of transgenic plants for many legumes, for both new cultivar releases and functional genomic studies since advances in molecular genetics, i.e. manipulation of endogenous gene expression or insertional mutagenesis, require efficient transformation systems (Somers *et al.*, 2003). It has also hampered the development and release of new cultivars by *in vitro* selection. Thus many efforts have been made to optimize protocols of *in vitro* regeneration in many legumes including alfalfa (Li *et al.*, 2009), common bean (Veltcheva *et al.*, 2005), cowpea (Bakshi *et al.*, 2012), chickpea (Tripathi *et al.*, 2013), grass pea (Ochatt *et al.*, 2002), lentil (Newell *et al.*, 2006; Sarker *et al.*, 2012), *L. japonicus* (Barbulova *et al.*, 2005), *M. truncatula* (Duque *et al.*, 2006), moth bean (Choudhary *et al.*, 2009), mung bean (Sivakumar *et al.*, 2010), pea (Zhihui *et al.*, 2009), pigeonpea (Krishna *et al.*, 2010) and soybean (Loganathan *et al.*, 2010). Although in many cases, the recovery of fertile plants is still low, it allowed application of genetic transformation and other tissue culture derived techniques to generate genetic diversity such as somaclonal variation, *in vitro* mutagenesis, and wide hybridization in some legume species. Here we review the advances gained in *in vitro* related approaches to improve legume genetic variability and their application to improve crop resistance to diseases and pests.

D. Wide hybridization

Wide hybridization or interspecific hybridization corresponds to the creation of a hybrid between plants from different species and/or genera. As such it offers the breeder the possibility to broaden the genetic variability outside the species range and to take advantage of the large genetic variability found in related wild species that often contain interesting traits such as resistance to diseases and pests. While hybridization of two closely related species may provide fertile plants, in most cases these species are completely or partially sexually incompatible leading to sterile hybrids. Two alternative *in vitro*-based approaches, embryo rescue and protoplast fusion, have been developed to circumvent these problems and overcome the sexual incompatibility barrier.

Embryo rescue is a set of *in vitro* culture methods, including embryo, ovule and ovary or pod culture that promotes the development of viable plants from immature or weak embryos, which would have otherwise prematurely aborted. This approach is thus particularly useful when wide hybridization with species from the secondary or tertiary gene pool is possible but may lead to only a very limited number or immature embryos. In these cases, efficient embryo rescue may improve their viability and increase their number by *in vitro* callus induction and subsequent plantlet regeneration (Pratap *et al.*, 2010). This method has been widely used allowing the creation of viable interspecific hybrids in many legume species including lentils (Fratini and Ruiz, 2006; Tullu *et al.*, 2011), common bean (Veltcheva *et al.*, 2005), chickpea (Clarke *et al.*, 2006), pigeonpea (Pratap *et al.*, 2010), grass pea (Hammett *et al.*, 1994) and *Trifolium* (Abberton, 2007) among others.

In cases where the reproductive barrier cannot be overcome by embryo rescue, protoplast fusion or somatic hybridization may be applied to break the incompatibility.

This method offers the possibility not only to overcome sexual incompatibility by enabling hybridization between more distantly related species and even outside the genera boundary but also to generate novel combinations of nuclear and/or cytoplasmic genomes by allowing asymmetric crosses between species with different ploidy (Tian and Rose, 1999). Protoplast fusion is thus a very attractive tool for breeders that can increase the available genetic diversity of crops. However, this method is more challenging than embryo rescue and requires several critical steps including the availability of a method to generate large numbers of protoplasts, the establishment of optimal conditions for growth and fusion of protoplasts and efficient plantlet regeneration protocols (Sonntag *et al.*, 2009). This method has been largely applied in species amenable to protoplast culture and *in vitro* regeneration such as alfalfa and barrel medic in which asymmetric and/or intergenera hybridizations have been reported, including the crosses *M. truncatula* x *M. scutellata*, *M. sativa* x *Onobrychis viciifolia* or *M. sativa* x *Lotus corniculatus* (Li *et al.*, 1993; Kaimori *et al.*, 1998; Tian and Rose, 1999). Protoplast fusion was also attempted to obtain somatic hybrids from interspecific hybridization of other legume species such as soybean (Hammatt *et al.*, 1992) and lupin (Sonntag *et al.*, 2009) or from intergenera hybridization between legume species or between legume and non-legume species including the crosses between *Pisum sativum* x *Lathyrus sativus* (Durieu and Ochatt, 2000), *Vicia faba* x *Helianthus annus* (Schnabl *et al.*, 1999) or *L. corniculatus* x *Oryza sativa* (Nakajo *et al.*, 1994) although regeneration of viable hybrid plantlets has not been reported yet.

Despite its potential, this method has not been extensively applied in breeding mainly due to the difficulty of regenerating viable and fertile plants from the somatic hybrid and due to the instability of the new characters that are often lost in subsequent generations by chromosome elimination (Mizukami *et al.*, 2006). The difficulty of

plantlet regeneration of most legume species through *in vitro* culture is also shared by the embryo rescue approaches that limited the broad application of *in vitro* based wide hybridization to breeding programmes although important progress in legume regeneration has already been made. Consequently, most studies of wide hybridization have been limited in demonstrating the possibility to obtain viable hybrids while a very limited number of studies have applied these approaches to introgress interesting traits to legume crops. Wild relative species are important sources of resistance to many diseases and pests. Thus application of wide hybridization methods offers the theoretical possibility to transfer and introgress resistance traits from wild species to legume crops.

Introgression of resistance to a number of pests and diseases has been described in chickpea, common bean, lentil, pea, soybean, white clover, *Vigna* spp. and pigeonpea. Anthracnose resistance has successfully been transferred from the wild species *Lens ervoides* to lentil through embryo rescue (Fiala *et al.*, 2009). In addition, the *L. culinaris* x *L. ervoides* lines may possess higher degree of resistance to *Stemphylium botryosum* (Tullu *et al.*, 2011). Genes for resistance to *Bruchus pisorum*, *Didymella pinodes*, *Orobanche crenata* and *Erysiphe pisi* have been transferred from *Pisum fulvum* or other wild *Pisum* to *P. sativum* through sexual hybridization (Fondevilla *et al.*, 2007a; Byrne *et al.*, 2008; Rubiales *et al.*, 2009). Resistance to root rot and rust have been introgressed in chickpea from *Cicer reticulatum* (Singh *et al.*, 2005; Madrid *et al.*, 2008). Resistance to ascochyta blight in chickpea may be introgressed from *C. reticulatum* and *C. echinospermum* (Collard *et al.*, 2001, 2003). Resistance to *Sclerotinia sclerotiorum*, *Colletotrichum lindemuthianum*, *Phaeoisariopsis griseola*, *Xanthomonas campestris* pv. *Phaseoli* and several viruses including bean mosaic virus and bean golden yellow mosaic virus has been introduced

into common bean from species of its secondary gene pool including *Phaseolus coccineus, P. costaricensis, P. polyanthus* and *P. acutifolius* (Mahuku *et al.*, 2002, 2003; Miklas *et al.*, 2006; Singh *et al.*, 2009; Teran *et al.*, 2013). Resistance to clover cyst nematode, root-knot nematode and several viruses including the peanut stunt virus, clover yellow vein virus and alfalfa mosaic virus has been introduced to white clover from *Trifolium ambiguum* and/or *T. nigrescens* (Abberton, 2007). Resistance to root-knot nematode *M. arenaria* was also successfully introgressed in peanut from the wild related species *Arachis cardenasii* (Mallikarjuna and Hoisington, 2009). In pigeon pea, varying level of resistance or tolerance to the cyst nematode *Heterodera cajani*, *Phytophthora drachsleri* and *Helicoverpa armigera* were transfered from the secondary gene pool species *Cajanus acutifolius* and *C. scarabaeoides* (Saxena, 2008). Resistance to several races of the cyst nematode *Heterodera glycine* was also conferred to soybean by crossing a *Glycine max* cultivar to one accession of its closely related *G. tormentella* (Riggs *et al.*, 1998).

Other resistances were more difficult to transfer such as the resistance to *Phytophthora drechsleri* f.sp. *cajani* that was recovered from crosses of pigeonpea with the tertiary gene pool species *C. platycarpus* that were made using embryo rescue. The progenies also showed potential for conferring resistance to several insect pests including pod borer and bruchid (Mallikarjuna *et al.*, 2011). Embryo rescue was also used to introgress resistance to late leaf spot, rust and to several peanuts viruses including peanut mottle virus, peanut stripe virus and peanut bud necrosis virus in from *Arachis glabrata* into peanut (Mallikarjuna, 2003). Similarly, resistance to early and late leaf spot disease and to the tobacco caterpillar *Spodoptera litura* was successfully transferred to peanuts from of *A. kempff-mercadoi* (Mallikarjuna *et al.*, 2004). Moderate resistance to late leaf spot was also detected in hybrid lines between *A. hypogaea* x *A.*

kretschmeri (Mallikarjuna and Hoisington, 2009). Embryo rescue was also applied to transfer the *Vigna mungo* resistance to yellow mosaic virus to *V. radiata* (Gosal and Bajaj, 1983). Transfer of aphid and weevil resistance to fertile S_1 generation of alfalfa was obtained by protoplast fusion of *Medicago rugosa* x *M. sativa* (Mizukami *et al.*, 2006). Resistance to various aphid species has been transferred from wild accessions to *M. truncatula*, which in addition to being a model legume is an annual pasture crop in parts of the world (Crawford *et al.*, 1989).

E. Somaclonal variation and in vitro selection

Tissue culture, such as callus cultivation, organogenesis and somatic embryogenesis, has the capacity to generate genetic variation in plants by inducing active transposition of retro-transposons, changes in DNA methylation profile or other spontaneous mutations (Larkin and Scowcroft, 1981; Jain, 2001). Such somaclonal variation is not desirable for some applications such as genetic transformation or massive micropropagation, but can be useful from a breeding perspective (Jain, 2001; Svetleva *et al.*, 2003). This method can broaden the genetic variability available and coupled with an efficient screening method, the so called *in vitro* selection, offer new approaches to improve legume crops (Svetleva *et al.*, 2003) which have already been explored to produce agronomically useful somaclones in pea, pigeonpea and others (Griga *et al.*, 1995; Chintapalli *et al.*, 1997). These approaches are thus considered an important complement to classical breeding methods (Svabova and Lebeda, 2005).

In vitro selection involves the use of toxins, herbicides or other stress-inducing substances to select resistant variants or somaclones in cell culture. This approach can considerably shorten the time of selection under *in vitro* pressure (Jain, 2001). The

simplicity and ease in exposing a large number of cells to a uniform dose of stressor and in identifying a resistant variant have attracted considerable attention among breeders (Chawla and Wenzel, 1987). This technique is particularly attractive to tackle plant resistance to fungal and bacterial diseases, contributing in the identification of resistant individuals within a highly susceptible population (Svabova and Lebeda, 2005). For instance, application of this approach led to the release of novel cultivars resistant to *Fusarium* wilt in banana, celery, tomato, and others, which illustrate the usefulness of these approaches in breeding (Jain, 2001). Despite its attractiveness, this approach had not been used extensively in legumes except in alfalfa where several studies aimed to identify variants for resistance to *Colletotrichum trifolii* (Cucuzza and Kao, 1986), *Verticillium albo-atrum* (Latunde-Dada and Lucas, 1988; Frame *et al.*, 1991; Koike and Nanbu, 1997), and mainly to *Fusarium solani* and *F. oxysporum* f. sp. *medicaginis* (Hartman *et al.*, 1984; Arcioni *et al.*, 1987; Binarova *et al.*, 1990). In addition to these studies on alfalfa, *in vitro* selection was used to screen for resistance against soil-borne pathogenic fungi in other legume species including chickpea and pea for resistance to *F. oxysporum* (Parkash *et al.*, 1994; Sharma *et al.*, 2010) and soybean against *F. solani* (Jin *et al.*, 1996). In all cases, higher resistance levels compared to the parental lines were found although complete resistance was not reported (Sharma *et al.*, 2010). Higher resistance to *A. rabiei* was also detected in a genetic variant of chickpea (Singh *et al.*, 1999).

Besides application of *in vitro* selection to screen for somaclonal variants in tissue culture, this approach can also be used to rapidly screen for resistance in progenies obtained by conventional breeding and transgenesis as a complement to field testing (Jain, 2001). It can also be applied in combination with induced mutagenesis to increase genetic diversity of a crop obtained by somaclonal variation.

F. Induced mutagenesis

Induced mutagenesis is a potent approach to rapidly increase the mutation rates within crop genomes to create novel genetic variation. Originally, mutation was obtained through seed treatment with chemical mutagens such as Ethyl Methane Sulfonate (EMS) or physical mutagens such as Gamma-ray irradiation that induced point mutations and deletions in the genome, respectively (Ahloowalia *et al.*, 2004). More recently, additional mutagenesis approaches including fast neutron irradiation, insertional mutagenesis using T-DNA or transposon tagging and gene silencing technologies have been developed primarily for functional genomic approaches although they can also be very valuable resources for breeding (Tadege *et al.*, 2009; Kurowska *et al.*, 2011). The adoption of induced mutagenesis as a legume breeding strategy became widespread as early as 1940 and is still an important approach in many breeding programmes (Upadhyaya *et al.*, 2011). Indeed, mutants obtained are highly valuable breeding resources that can be directly released as new varieties or serve as breeding materials to transfer new traits to elite cultivars by crossing and selection (Ahloowalia *et al.*, 2004). As a result, more than 450 registered varieties in 29 legume species have been obtained (Kharkwal *et al.*, 2010). Most of these varieties were bred for agronomic characters such as growth habit, plant architecture and time to flowering with the finality to improve yield (Ahloowalia *et al.*, 2004). As a consequence, many induced-mutagenesis programmes have been oriented toward the nitrogen fixation and the symbiotic interaction with rhizobia and arbuscular micorrhyzal fungi (Sagan *et al.*, 1994; Bhatia *et al.*, 2001). The direct influence of pest and disease resistance on yield and quality has also driven intensive efforts to develop new mutant-derived varieties with higher resistance to diseases and pests (Micke, 1984; Ahloowalia *et al.*, 2004). For

instance, a joint initiative of the Food and Agriculture Organization (FAO) and the International Atomic Energy Agency (IAEA) drove the creation of many mutagenesis-based breeding programmes in numerous developing countries from 1970 onwards to breed new legume varieties most of which targeted traits for higher disease resistance, together with higher yield (Micke, 1984). This led to the identification of several lines or cultivars with increased resistance to different diseases including cultivars of mungbean resistant to yellow mosaic virus and cercospora leaf spot, black gram resistant to YMV, chickpea resistant to ascochyta blight and fusarium wilt (Ahloowalia *et al.*, 2004; Salimath *et al.*, 2011), common bean resistant to rust, anthracnose , halo blight and bean common virus (Fadl, 1983; Micke, 1984), lupin resistant to fusarium wilt (Micke, 1984), soybean resistant to cyst nematodes and virus (Micke, 1984; Ahloowalia *et al.*, 2004) and pea resistant to powdery mildew, rust, fusarium wilt and aphanomyces root rot (Gritton and Hagedorn, 1979; Fadl, 1983; Pereira and Leitao, 2010; Sharma *et al.*, 2010). The establishment of high-throughput methods to identify mutated sites in mutants such as targeted-induced local lesion in genome (TILLING) and deletion-TILLING platforms allow identification of mutants carrying mutation in specific genes which is a powerful reverse genetic approach (Porch *et al.*, 2009; Tadege *et al.*, 2009). Besides their usefulness as functional genomic tool to clarify gene function in model species, these methods can be applied directly for breeding. These approaches may be applicable to most plant species to screen for mutations in specific genes within chemical or physical mutant collections (Kurowska *et al.*, 2011). Thus, they can identify new mutants carrying interesting alleles of key defensive genes and more importantly they can be used to transfer candidate genes from model to legume crops by identifying new alleles of these candidate genes in these species (Rispail *et al.*, 2010).

G. Genetic transformation

Genetic transformation or genetic engineering is a method to transfer new traits to any organism. Indeed, it expands the sources of genes for plant improvement to all organisms, far beyond the gene pool normally accessible via sexual hybridization. This method not only offers the possibility to introduce new genes but also to manipulate the expression of endogenous genes to create new phenotypes useful to study gene function and breeding (Somers *et al.*, 2003). In general, the presence of very rigid and thick cell walls coupled with efficient DNA repair systems has made the process of genetic transformation of some plant species quite challenging. Despite these difficulties, it is possible to transform many plant species provided that an efficient protocol of *in-vitro* regeneration is available (Somers *et al.*, 2003). Many if not all legume species can be genetically transformed including pea, chickpea, soybean, *M. truncatula*, peanuts, common bean and cowpea albeit with limited recovery rates in most cases (Chandra and Pental, 2003; Somers *et al.*, 2003; Popelka *et al.*, 2004). Given the high potential of genetic transformation in plant breeding, large efforts have been made to improve the efficiency of transformation in many legumes. Legume transformation has been mainly performed by *Agrobacterium tumefaciens* but in some cases, especially in common bean, micro-particle bombardment has been used to deliver exogenous DNA to embryogenic or organogenic tissues (Chandra and Pental, 2003). Despite these efforts, in most cases the recovery rate of transgenic lines is still low and about 1% depending on the transformation method and the legume species (Broughton *et al.*, 2003; Chandra and Pental, 2003). This low recovery rate is generally ascribed to the recalcitrant nature of most legumes to *in vitro* regeneration, which is the main bottleneck to the application of genetic transformation in legumes (Somers *et al.*, 2003). To shorten the generation

time of stable transgenic lines and solve the very low plantlet rooting rates of some legume species, micrografting, as originally described for pea mutants (Murfet, 1971) of transgenic regenerated shoots onto non-transgenic roots was used with some success in pea, lentil and chickpea (Gulati *et al.*, 2002; Chakraborti *et al.*, 2009; Hassan *et al.*, 2009). For instance this method reduced the generation time by 3 months in pea (Hassan *et al.*, 2009). Alternatively, *in-planta* transformation that may circumvent the need of tissue culture regeneration steps has been reported for *M. truncatula* (Trieu *et al.*, 2000), peanuts (Rohini and Rao, 2000), pea (Chowrira *et al.*, 1998), pigeonpea (Ramu *et al.*, 2012), cowpea (Adesoye *et al.*, 2008), as well as lentil and soybean (Chowrira *et al.*, 1996).

All these efforts toward improving *in vitro* regeneration and transformation procedures allow the creation of transgenic lines with improved resistance to pests and diseases in several legumes (Table 1). By contrast to induced-mutagenesis that targeted mainly fungal diseases, genetic transformation was mainly aimed to confer resistance to insect and virus diseases. Thus one of the main transformation strategies was the introduction of derivatives of *cry1* genes from *Bacillus thuringiensis* conferring resistance to many pod borer insects. To improve the efficiency of resistance, the strategy has moved from the transfer of a single *cry1* gene to constructs containing *cry* genes with different modes of action. This strategy has worked well for corn and cotton to increase the durability of the resistance and is especially important for insect resistance management (Roush *et al.*, 1998). So far, the transgenic lines of pigeonpea, chickpea, cowpea and soybean resistant to the lepidopterans *Helicoverpa armigera*, *Spodoptera litura*, *Maruca vitrata*, *H. zea*, *Anticarsia gemmatalis* and *Pseudoplusia includens*, respectively, contain only single *cry* genes, although in some cases an option

exists for stacking two different genes by crossing (Walker *et al.*, 2000; Sanyal *et al.*, 2005; Surekha *et al.*, 2005; Adesoye *et al.*, 2008; Ramu *et al.*, 2012; Table 1).

Expression of *cry3a* in alfalfa provides resistance to *Hypera postica*, the alfalfa weevil (Tohidfar *et al.*, 2013). Alternatively, high levels of resistance to different bruchids was also shown in transgenic pea, chickpea, adzuki bean and cowpea expressing high levels of common bean α-amylase 1 inhibitor in seeds (Schroeder *et al.*, 1995; Ishimoto *et al.*, 1996; Sarmah *et al.*, 2004; Solleti *et al.*, 2008).

Although several strategies have been used to confer resistance to viruses in legumes, the most frequently used method consists of expressing viral coat or other viral genes within the host. This resulted in protection of pea against pea enation mosaic virus and alfalfa mosaic virus (Chowrira *et al.*, 1998; Timmerman-Vaughan *et al.*, 2001), of clover against the bean yellow mosaic virus (Chu *et al.*, 1999), of bean against bean golden mosaic virus (BGMV) (Bonfim, 2007) and of soybean against the soybean mosaic virus and the bean pod mottle mosaic virus (Reddy *et al.*, 2001; Wang *et al.*, 2001).

Overexpression and introduction of several PR genes such as chitinase and glucanase or genes from the phenylpropanoid pathway were also found to provide increased resistance in alfalfa against *Phoma medicaginis* (He and Dixon, 2000; Samac *et al.*, 2004) and in soybean against the cyst nematode *Heterodera glycine* (Ornatowski *et al.*, 2004). Transgenic pea lines expressing chitinase and/or β1,3-glucanase are also under evaluation for their usefulness against other fungal diseases (Hassan *et al.*, 2009; Amian *et al.*, 2011). There is good potential for transformation to help tackle recalcitrant plant pathogens. In the case of *Rhizoctonia solani*, a moderate degree of resistance to AG8 was found in specific *M. truncatula* accessions and molecular genetic

analysis demonstrated that this was due, in part, to recruitment of the ethylene signalling pathway (Penmetsa *et al.,* 2008; Anderson *et al.,* 2010). By identifying transcription factors in ethylene signalling that were involved in mediating the moderate resistance, it was possible to generate high levels of resistance to *R. solani* AG8 by expressing these genes in composite transgenic plants using *A. rhizogenes* without having deleterious effects on growth and development or symbiotic interactions with rhizobium (Anderson *et al.*, 2010).

From the foregoing it can be concluded that resistance to pests and diseases can be obtained through genetic transformation of legumes. While uncommon, some adverse effects have been occasionally detected in transgenic lines such as the unexpected immune reaction in mice consuming peas expressing the α-amylase 1 inhibitor (Prescott *et al.,* 2005) although this early finding has not been confirmed in a more extensive study (Lee *et al.*, 2013b).

Most of the transgenic lines generated so far are still at the laboratory stage except the Bt soybean (Miklos *et al.,* 2007) which is close to commercialisation in Brazil, the BGMV resistant bean (Bonfim *et al.,* 2007) which is in advanced field trials in Brazil, the pod borer resistant chickpea (Acharjee *et al.*, 2010) which is in early field trials in India, and the pod borer resistant cowpea (Higgins *et al.*, 2012) which is in multi-location field trials in Nigeria. This is partly due to social (public) and political concerns (lower rate of investment in legume crops compared to other crops such as cotton, canola and maize) that have nearly completely blocked the release of transgenic cultivars of any kind in Europe. The low level of public acceptance and the related high cost of de-regulating a transgenic crop have meant that legumes (with the exception of soybean) are struggling to become a choice for farmers who want to diversify. Two of the main public concerns about transgenics are the use of antibiotic selectable markers

that are perceived to have undesirable side effects and the mixing of genetic material from species that cannot hybridize (Holme *et al.*, 2013). It is possible to obtain marker-free transgenic plants as recently shown for alfalfa. The development of intragenesis or cisgenesis approaches based on the insertion of genes from related or even the same species to modify endogenous expression level may relieve some of this social pressure (Ferradini *et al.*, 2011; Holme *et al.*, 2013). However, this seems unlikely in the short to medium term despite science-based assurances of safety, including from the WHO.

In summary, the future importance of legumes as high protein foods and their role in sustainable farming may eventually contribute to the acceptance of genetically modified legumes.

IV. CONCLUSIONS AND FUTURE PROSPECTS

Diseases and pests are the main causes of yield reduction of legume impairing both yield and quality of the crops. Of the different control methods available, breeding for resistance is the most efficient, economical and environmentally friendly. Crop improvement is thus a crucial element for sustainable agriculture that can be performed by several approaches from classical breeding to genetic engineering. However, a detailed understanding of host-pathogen interactions and of the efficient resistance mechanisms at the cellular, genetic and molecular levels are required to improve the efficiency of the breeding process.

Although development of genomic resources in legumes has been relatively slow compared to other crops such as cereals, the wide applicability of MAS has

already been demonstrated in soybean and to a much lesser extent in common bean, cowpea and pea and are still in early infancy stage for most other legumes (Miklas *et al.*, 2006; Millan *et al.*, 2006; Muehlbauer *et al.*, 2006; Torres *et al.*, 2006; Vaz Patto *et al.*, 2006; Rispail *et al.*, 2010). However, one encouraging fact is that exceptional progress has already been made in generating ample genomic resources in all the major pulse crops. The recent development of large scale phenotyping, genome sequencing and analysis of gene, protein and metabolite expressions will be of great help in further deciphering plant-pathogen interactions and identifying key resistance components not only in model legumes but also in economically important legume crops.

The continuous decrease in cost and the increased performance of many post-genomic tools including next generation sequencing platforms now allow the application of such large-scale approaches having already resulted in the public release of genomic and/or transcriptomic information of several legumes such as soybean, common bean, chickpea, cowpea, peanuts, pea, grasspea and pigeonpea, in addition to the model legumes *Medicago truncatula* and *Lotus japonicus* (Sato *et al.*, 2008; Branca *et al.*, 2011; Franssen *et al.*, 2011; Garg *et al.*, 2011; Hiremath *et al.*, 2011; Varshney *et al.*, 2011, 2013; Young *et al.*, 2011; Kaur *et al.*, 2012, 2013; Almeida *et al.*, 2013; Barros-Carvalho *et al.*, 2013; Cannon, 2013). This will no doubt contribute to unraveling key components of legume defense and resistance to pathogens and pests in the near future. These key components could then be introgressed into the crops through plant breeding. At this stage, biotechnological approaches such as marker-assisted selection, tissue culture, *in vitro* mutagenesis, and genetic transformation could contribute to speeding up classical breeding and overcome major problems such as lack of natural sources of resistance and sexual incompatibility for more efficient crop

improvement. However, progress in deploying these resources, especially to legumes, has been depressingly slow.

This will be complemented by efforts to sequence the genomes of key pathogens and pests such as the genome sequences of *Fusarium oxysporum* (Ma *et al.*, 2010), *Verticillium albo-atrum* (Klosterman *et al.*, 2011), *Botrytis cinerea* (Staats and van Kan, 2012), *Rhizoctonia solani* (Wibberg *et al*., 2012), *Phytophthora sojae* (Tyler *et al*., 2006), *Macrophomina phaseolina* (Islam *et al*., 2012), *Ralstonia solanacearum* (Remenant *et al.*, 2010), *Pseudomonas syringae* (*Gardiner et al., 2013*), *Curtobacterium flaccumfaciens* (Flanagan *et al*., 2013), *Xylella fastidiosa* (Zhang *et al*., 2011), *Meloidogyne incognita* (Abad *et al*., 2008), *Meloidogyne hapla* (Opperman *et al*., 2008), alfalfa mosaic virus (Cornelissen *et al*., 1983), cucumber mosaic virus (Owen *et al*., 1990), pea enation mosaic virus (Demler and de Zoeten, 1991), bean leafroll virus (Domier *et al.*, 2002), pea seed-borne mosaic virus (Johansen *et al*., 1991) and the pea and soybean aphids (International Aphid Genomics Consortium, 2010; Liu *et al.*, 2012). In addition, further in-depth studies on the biology of the pests and pathogens are required. Indeed comprehensive information on host status and virulences are often incomplete, with insufficient knowledge on the existence of races and on their distribution. This is a major limitation for any breeding programme. In turn, the available information on levels of resistance and the responsible mechanisms is often incomplete. As a consequence, resistance breeding will be efficiently accelerated only after significant input to improve existing knowledge on biology of the causal agents as well as on the plant.

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Tables

Table 1 List of some legumes genetically engineered for biotic stress

Legume target	Biotic stress	Gene(s)	Reference(s)
Arachis hypogaea	Tomato spotted wilt virus (TSWV)	Nucleocapsid from TSWV	Magbanua *et al.*, 2000
	Sclerotinia minor	Oxalate Oxidase from barley	Livingstone *et al.*, 2005
	Elasmopalpus lignosellus	Cry 1Ac	Singsit *et al.*, 1997
Cajanus cajan	*Spodoptera litura*	*cry1 E-C*	Surekha *et al.*, 2005
	Helicoverpa armigera	*cry1 AcF*	Ramu *et al.*, 2012
Cicer arietinum	*Helicoverpa armigera*	*cry1Ac*	Sanyal *et al.*, 2005
	Helicoverpa armigera	Cry2Aa	Kar *et al.*, 1997 Acharjee *et al.*, 2010
	Aphis croccivora	*Allium sativum* leaf agglutinin	Chakraborti *et al.*, 2009
	Callosobruchus maculatus *Callosobruchus chinensis*	Alpha-amylase inhibitor from bean	Sarmah *et al.*, 2004
Glycine max	*Sclerotinia sclerotiorum*	Oxalate oxidase (Germin (gf-2.8) fom wheat)	Donaldson *et al.*, 2001
	Sclerotinia sclerotiorum	Oxalate decarboxylase	Cunha *et al.*, 2010
	Bean Pod Mottle Virus (BPMV)	Capsid polyprotein from BPMV	Reddy *et al.*, 2001
	Heterodera glycines	Chitinase from *Manduca sexta*	Ornatowski *et al.*, 2004
	Helicoverpa zea *Anticarsia gemmatalis* *Pseudoplusia includens*	*cry1Ac*	Walker *et al.*, 2000 Miklos *et al.*, 2007
	Heterodera glycines	Promoter of chalcone synthase from Promoter of Phenylalanine Ammonia Lyase	Narayanan *et al.*, 1999
	Soybean Mosaic Virus (SMV)	Coat protein gene from SMV	Wang *et al.*, 2001
Lupinus angustifolius	*Colletotrichum lupine* *Pleiochaeta setosa*	Anti-apoptotic baculovirus gene *p35*	Wijayanto *et al.*, 2009
Medicago sativa	*Phoma medicaginis*	Resveratrol synthase from *A. hypogaea*	Hipskind and Paiva, 2000
	Phoma medicaginis	Isoflavone *O*-Methyltransferase	He and Dixon, 2000
	Phoma medicaginis	Endochitinase (ech42)	Samac *et al.*, 2004
	Pratylenchus penetrans	Oryzacystatin I and II	Samac and Smigocki, 2003
Medicago	Alfalfa Mosaic Virus	Virus coat protein from	Jayasena *et al.*, 2001

truncatula	(AMV)	AMV	
Phaseolus acutifolius	*Zabrotes subfasciatus*	Arcelins-1, Arcelins-5	Zambre *et al.*, 2005
Phaseolus vulgaris	Bean Golden Mosaic Virus (BGMV)	*Rep-TrAP-REn*, *BC1* (viral genes)	Aragão *et al.*, 1998 Bonfim *et al.*, 2007
Pisum sativum	*Bruchus pisorum*	Alpha-amylase inhibitor (alpha-AI-1 and 2)	Schröeder *et al.*, 1995 Morton *et al.*, 2000
	Pea Seed-borne Mosaic Virus (PSbMV)	Replicase (*NIb*) from PSbMV	Jones *et al.*, 1998
	Pea enation Mosaic virus	Coat protein from PeMV)	Chowrira *et al.*, 1998
	Alfalfa Mosaic Virus (AMV)	Coat protein from AMV	Timmerman-Vaughan *et al.*, 2001
Trifolium subterraneum	Bean yellow Misaic Virus (BYMV)	Coat protein from BYMV	Chu *et al.*, 1999
Vigna unguiculata	*Maruca vitrata*	Cry1Ab Cry 1Ab	Adeboye *et al.*, 2008 Higgins *et al.*, 2012
	Callosobruchus maculatus	Alpha-amylase inhibitor from bean	Lee *et al.*, 2013b Solleti *et al.*, 2008
Vigna angularis	*Callosobruchus chinensis*	Alpha-amylase inhibitor from bean	Ishimoto *et al.*, 1996

Table 2. List of genes and/or QTLs identified in the various legumes against the various pests and diseases

Legume species	Biotic Stresses	Genetic Bases of resistance	References
Lentil (*Lens culinaris*)	Ascochyta Blight (*Ascochyta lentis*)	Major genes	Chowdhury *et al.*, 1991; Ford *et al.*, 1999
		QTLs	Rubeena *et al.*, 2006
Common Bean (*Phaseolus vulgaris*)	Fusarium Root-rot (*Fusarium solani* f. sp. *phaseoli*)	QTLs	Freyre *et al.*, 1998; Roman-Aviles and Kelly, 2005
Chickpea *(Cicer arietinum)*	Ascochyta blight (*Ascochyta rabiei*)	Major genes	Singh and Reddy, 1993; Collard *et al.*, 2001; Chen *et al.*, 2004
		QTLs	Santra *et al.*, 2000; Tekeoglu *et al.*, 2002; Collard *et al.*, 2003; Rakshit *et al.*, 2003; Udupa and Baum 2003; Cho *et al.*, 2004; Iruela *et al.*, 2006
	Fusarium wilt (*Fusarium oxysporum* f. sp. *ciceri*)	Major genes	Sharma and Muehlbauer, 2007
Faba bean *(Vicia faba)*	Ascochyta blight (*Ascochyta fabae*)	Major genes	Rashid *et al.*, 1991; Kophina *et al.*, 2000; Kharrat *et al.*, 2006
		QTLs	Román *et al.*, 2003; Avila *et al.*, 2004
Pea	Ascochyta blight (*Ascochyta pisi* Race C)	A major gene modifed by minor genes or QTLs	Darby *et al.*, 1985; Dirlewanger *et al.*, 1994
	Ascochyta blight	QTLs	Timmerman-Vaughan *et al.*, 2002; Tar'an *et al.*, 2003; Prioul *et al.*, 2004; Fondevilla *et al.*, 2008b

(*Pisum sativum*)	(*D. pinodes*)	Major genes	Clulow *et al.*, 1991
	Fusarium wilt (*Fusarium oxysporum f. sp. pisi*)	Major genes	McClendon *et al.*, 2002; Okubara *et al.*, 2005
		QTLs	McPhee *et al.*, 2012
Soybean (*Glycine max*)	Fusarium root rot (*Fusarium graminearum*)	QTLs	Ellis *et al.*, 2012
	Phytophthora sojae	QTLs	Lee *et al.*, 2013c
		Major genes	Zhang *et al.*, 2013
Barrel Medic (*Medicago truncatula*)	Verticillium wilt (*Verticillium albo-atrum*)	QTLs	Ben *et al.*, 2013b
Cowpea (*Vigna unguiculata*)	Fusarium wilt (*F.o. tracheifilum*)	Major genes	Pottorff *et al.*, 2013