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Pathosystem Common Bean–*Uromyces appendiculatus*: Host Resistance, Pathogen Specialization, and Breeding for Rust Resistance

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ABSTRACT

Common bean (*Phaseolus vulgaris* L.) is the world's most important food legume for direct human consumption. This new world crop is adapted to many niches and it is grown in distinct regions and different seasons around the world. The common bean crop is grown by subsistence levels farmers with little technology as well as by farmers in developed nations that use high input technologies. Unfortunately, common bean yields are quite low compared to other grain legumes; e.g., soybeans and peas. One of the several factors contributing to this situation is the high number of pathogens that cause common bean diseases. Among these diseases is the common bean rust, incited by the basidomycete fungus *Uromyces appendiculatus* F. Strauss (syn. *U. phaseoli* G. Winter), which can cause great yield losses. We present here relevant information about the common bean rust including its etiology, epidemiology, the rust pathogen infection process, the symptomatology and genetic diversity of the pathogen. We also review progress on the control of the disease using cultural practices, biological and chemical methods. It is also reported and discussed the host resistance and pathogen specialization, genetics of host-pathogen interactions, the available molecular markers linked to rust resistance genes, and its utilization in marker assisted selection (MAS) for the development of rust resistance cultivars.

Keywords: common bean rust, gene pyramiding, host-pathogen interaction, marker-assisted selection, *Phaseolus vulgaris*, *Uromyces appendiculatus*

Abbreviations: ARS, Agricultural Research Service; **BIC**, Bean Improvement Cooperative; **BIOAGRO**, Instituto de Biotecnologia Aplicada à Agropecuária; **BJ**, 'BAT 93' × 'Jalo EEP 558' RIL population: common bean core mapping population; **BRW**, Bean Rust Workshop; **CBR**, common bean rust; **CNC**, Compuesto Negro Chimaltenango; **FAO**, Food and Agriculture Organization of the United Nations; **GRIN**, Germplasm Resources Information Network; **ITIS**, Integrated Taxonomic Information System; **LG**, linkage group; **MAS**, marker-assisted selection; **PC-50**, Pompadour Checa 50; **RAPD**, random amplified polymorphic DNA; **RR**, rust resistance; **SCAR**, sequence characterized amplified region; **TRAP**, targeted region amplified polymorphism; **UFV**, Universidade Federal de Viçosa; **USDA**, United States Department of Agriculture

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INTRODUCTION

Legumes used for human consumption include common bean (*Phaseolus vulgaris* L.), cowpea (*Vigna unguiculata*), pigeonpea (*Cajanus cajan*), chickpea (*Cicer arietinum*), lentils (L*ens culinaris*), peas (*Pisum sativum*) and broad beans (*Vicia beans*). However, the common bean is the most important worldwide. Dry beans were grown on approximately 27 million hectares in more than 120 countries in 2007 (http://faostat.fao.org). Common bean is also the main species among the other domesticate *Phaseolus* beans, which includes *P. lunatus* (lima bean), *P. coccineus* (scarlet bean), *P. acutifolisus* (tepary beans), and *P. polyanthus* (year-long bean). All of these beans are mainly grown and consumed in Latin America. However, common bean is grown and consumed throughout the entire world but principally in developing countries of Latin America, Africa, and Asia. The social value of the common bean is considerably high to millions of people in many countries and most especially in Latin America as well as in Eastern and Southern Africa (Pachico 1989; Wortmann *et al*. 1998; Broughton *et al*. 2003).

The cultivars released for these areas have to present a high spectrum of disease resistance, which is one of the

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main causes of yield and quality losses (Stavely and Pastor-Corrales 1989). This is especially true for small farms with low-technology inputs, which play an important role, as they account for the greatest fraction of the product for the world market supply (http://faostat.fao.org).

Among the most destructive diseases that attack common bean and cause serious damage we find bean rust which is incited by a highly variable pathogen, the fungus *Uromyces appendiculatus* F. Strauss (syn. *U. phaseoli* G. Winter). This disease is distributed throughout the world, but it effectively causes major production problems in humid tropical and subtropical areas and periodic severe epidemics in humid temperate regions (Stavely *et al.* 1989; Pastor-Corrales 2003).

Severe bean rust epidemics have been reported in Australia, China, the United States, and some areas of Europe. Major losses have occurred in Burundi, Ethiopia, Kenya, Malawi, Rwanda, South Africa, Tanzania, Uganda, and Zimbabwe. In Latin America, the bean rust is also a serious problem, major losses occurred in Argentina, Bolivia, Brazil, Colombia, Costa Rica, Cuba, Dominican Republic, Ecuador, El Salvador, Guatemala, Haiti, Honduras, Jamaica, Mexico, Nicaragua, and Peru (reviewed in Stavely and Pastor-Corrales 1989). In Brazil, the disease causes major losses in south, southeast and central areas, including the states of Paraná, Rio Grande do Sul, Santa Catarina, Minas Gerais, São Paulo, and Goiás (Souza *et al.* 2005a).

Disease losses worldwide measured in greenhouse and field conditions can vary from 18 to 100% (Stavely and Pastor-Corrales 1989; Staples 2000). According to Lindgren *et al.* (1995) a 1% increase in rust severity leads to yield losses of approximately 19 kg/ha. In Brazil, yield losses higher than 68% were detected in the state of Minas Gerais located in the southeast area of the country (Vieira *et al.* 2005).

The fungus *U. appendiculatus* can infect many species of the genera *Phaseolus*, including *P. acutifolius*, *P. caracalla*, *P. coccineus*, *P. lunatus*, *P. maculatus*, *P. nanus*, *P. ovatus*, *P. polystachyus*, and *P. vulgaris* (Arthur 1915; Hennen *et al.* 2005). The pathogen also infects other legume species including *Macroptilium atropurpureum*, *Vignia unguiculata*, *V. luteola*, *V. adenantha*, *V. vexillata*, and *V. angularis* (Almeida 1977; Chung *et al.* 2004). However, the prevalent host is the common bean.

Disease management practices for bean rust control include crop rotation, soil incorporation of bean debris, planting within recommended dates, growing resistant cultivars, and timely spraying of fungicides (Mmbaga *et al.* 1996a). In addition to being harmless to the environment, the use of resistant cultivars is an economically advantageous strategy as compared to chemical control. However, the wide variability of *U. appendiculatus* represents an obstacle to breeding programs aiming at the development of common bean cultivars with durable resistance to rust. The simultaneous introgression (pyramiding) of different rust resistance (RR) genes in the same genetic background has been proposed in order to obtain bean cultivars with durable and wide spectrum resistance (Coyne and Schuster 1975; Miklas *et al.* 1993; Alzate-Marin *et al.* 2005).

This review aims to report and discuss important aspects about the common bean rust etiology and epidemiology, the *U. appendiculatus* infection process, disease symptomatology and pathogen variability, cultural practices, biological and chemical control, host resistance and pathogen specialization, genetics of host-pathogen interaction, and breeding for RR assisted by molecular markers.

ETIOLOGY AND EPIDEMIOLOGY

U. appendiculatus belongs to the Basidiomycota subdivision of the Fungi kingdom, class Teliomycetes, order Uredinales, and family Pucciniaceae (http://www.itis.gov/index.html). The bean rust pathogen is an obligate parasite which has an autoecious and macrocyclic life cycle completed entirely on the bean host (Andrus 1931).

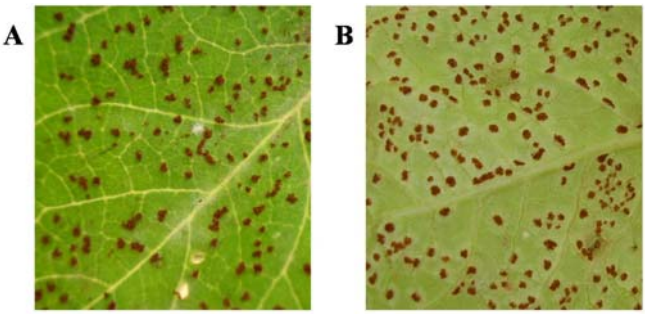

Fig. 1 ***Uromyces appendiculatus*** **uredia development on both adaxial (A) and abaxial (B) leaf surfaces of common bean (cv. 'US Pinto 111').**

The life cycle of the fungus is divided into five spore stages: spermatia, aeciospores, uredospores, teliospores, and basidiospores (Cummins 1978). The pathogen overwinters as teliospores in plant debris. The teliospore germinates to produce a basidium in which meiosis occurs and on which haploid basidiospores develop to infect the host (Gold and Mendgen 1984a). The basidiospores are windblown to susceptible plants where they germinate and penetrate the upper leaf surface through stomata. A layer of free water is necessary for germination and penetration to occur. On a susceptible bean cultivar, an appressorium is formed, penetration is direct and intercellular and intracellular hyphae are developed (Gold and Mendgen 1984b). When basidiospores infect bean leaves, it takes about six days at 22-26°C for a small chlorotic fleck containing the pycnium to develop. About seven days later, the pycnium produces droplets containing spermatia and receptive hyphae (Groth and Mogen 1978). Cross fertilization of a pycnium by pycniospores of the opposite mating type will begin aecium formation after about 10 days at 22-26°C. Aeciospores form in the white aecium and, upon their release, are able to infect the host plant. From eight to 10 days later, each aeciospores infection produces a uredium with uredospores (Andrus 1931; Groth and Mogen 1978). Pycnia and aecia are rarely observed under field conditions (Sherf and Macnab 1986; Hall 1991). An illustrative life cycle diagram of bean rust is available on McMillan *et al.* (2003).

The most commonly observed spore is the uredospore (vegetative spore). Uredospores are produced in uredia (pustules) on the upper or lower leaf surface (**Fig. 1**). They are light brown, unicellular, spiny, thin walled, and globoid to ellipsoid in shape. They may have two equatorial or superequatorial spores and measure 20-27 µm × 24-30 µm (Cummins 1978). The uredospores are capable of germinating to provide infectious hyphae that infect the host plant and form new uredium in which new uredospores are produced. The optimal temperature range for uredospores germination is 16-24°C. Germination occurs in the first six to eight hours in the presence of moisture. High temperatures (> 32°C) may kill the fungus and temperatures below 15°C retard the fungal development (von Alten 1983). Light intensity is an important factor for the fungal development. According to Augustin *et al.* (1972), infection is favored by low light intensity, about 2.0×10^{-5} µE $cm^{-2}s^{-1}$, for 18 hours. Several generations of uredospores are produced in a given season and reinfect bean plants. Uredospores thus serve as inoculum for secondary spread of the disease. Sporulation increases when infected plants are exposed to at least a 12-hour photoperiod and relative air humidity of 95% over 10-18 hours per day (Augustin *et al.* 1972). The fungus can produce 1,000,000 uredospores/cm^2 on leaves bearing two to 100 pustules/cm^2 (Yarwood 1961). The sporulation per unit of leaf area can vary inversely with uredia density (Imhoff *et al.* 1982). Dense infections also reduce uredia size (Harter and Zaumeyer 1941). Uredospores can survive up to 60 days under field conditions (Zambolim and Chaves 1974). They contain a water-soluble germination self-inhibitor, methyl *cis*-3,4-dimeth-oxycinnamate. This inhibitor is removed by washing spores with water and is counteracted

by a water-soluble substance in bean leaves (Allen 1972). The latent period, defined as the time from inoculation until 50% of the pustules on the adaxial leaf surface open, varies from seven to 15 days depending on temperature and humidity factors (Carrijo *et al.* 1980; Imhoff *et al.* 1982; Lindgren *et al.* 1995; Faleiro *et al.* 1999a; Souza *et al.* 2007a).

As the plant begins to mature, teliospores are produced in the uredium, converting it to a telium. Teliospores have a hyaline pedicel and are blackish brown, unicellular, have few to numerous verrucae, are rarely smooth, thick walled, and are globoid to broadly ellipsoid in shape. They may have a hyaline papilla over the pore and measure 24 µm × 30 µm. Only some races of the fungus produce teliospores (Harter and Zaumeyer 1941; Groth and Mogen 1978). Fusion of dikaryotic hyphae occurs in the teliospores of some *U. appendiculatus* races after they are formed (Andrus 1931; Harter and Zaumeyer 1941). The teliospores need a dormant period before they germinate.

The *U. appendiculatus* spores (uredospores and teliospores) can overwinter in bean debris and on wooden supports used for climbing bean plants (Davison and Vaughan 1963a). Uredospores can be transported by wind currents for long distances. They may provide primary, as well as secondary, inoculum during epidemics in bean growing world areas as Latin America and Africa where multiple cropping and planting dates provide a continuum of susceptible host tissue during favorable environmental conditions (Stavely and Pastor-Corrales 1989). The cropping systems, monoculture or association growing, also may influence the bean rust incidence (Hall 1991; Mmbaga *et al.* 1996a; Vieira *et al.* 2005).

Although *U. appendiculatus* does not grow in culture, viable spores can be preserved under laboratory conditions. Dry uredospores in plastic or glass tubes kept under dark conditions have been successfully stored at 5 ± 1°C and relative humidity < 50% for about one year in the Common Bean Breeding Program of the BIOAGRO-UFV, in Viçosa, MG, Brazil. Uredospores frozen at -80°C are stored for many years in the fungal collection maintained at the USDA-ARS Bean Project, in Beltsville, MD, USA (Dr. M. A. Pastor-Corrales, pers. comm.). Generally the inoculation using preserved spores is carried out when the primary leaves of the plants to be tested reached approximately 2/3 of their full development, about 10 days after sowing under greenhouse conditions (20 ± 5°C). The standard concentration of inoculum is 2.0×10^4 uredospores/mL of distilled water containing Tween-20 (0.05%, v/v). The inoculum solution can be sprayed on both leaf surfaces with a manual atomizer (e.g., atomizer De Vilbiss n° 15) adapted to an electric compressor. After inoculation the plants are transferred to a mist chamber (20 ± 1°C and relative humidity > 95%) where they are kept for approximately 48 h under a 12-hour light regime. In order to avoid contamination, plants inoculated with different isolates are kept in separate compartments of the mist chamber. After this period the plants are transferred to a greenhouse (20 ± 5°C), where they are kept until symptom evaluation (Carrijo *et al.* 1980; Souza *et al.* 2005a, 2007a). The inoculation can also be conduced using common bean excised leaves, as reported by Souza *et al.* (2005b). In the alternative method proposed by these authors, after inoculation, each leaf is placed in a Petri dish (90 × 15 mm) over a filter paper previously moistened with 3.0 mL of distilled water. The dishes are incubated in a BOD at 20°C, under a 12-hour light regime. Each filter paper is moistened again with 1.5 mL of distilled water in a regime of three-day interval up to the disease symptom evaluation.

Mersha and Hau (2008) recently studied the epidemics of common bean rust and their effects on host dynamics of common bean in three controlled greenhouse experiments, with and without fungicide sprays, on two susceptible cultivars ('Dufrix' and 'Duplika'). Bean plants were artificially inoculated with *U. appendiculatus* uredospores and temporal disease progress, as well as host growth dynamics (leaf size and defoliation), were monitored on a leaflet basis in comparison with non-inoculated plants sprayed with water only. The results showed that bean rust epidemics substantially affected host growth by reducing the total leaf area formed by 17.4-35.6 and 35.3-46.2% compared with healthy plants for cultivars 'Dufrix' and 'Duplika', respectively. Fungicide sprays mitigated the negative effect of bean rust, leading to a gain in leaf area of 17-21% compared with unsprayed plants in both cultivars in two experiments, while in another experiment, disease control had no effect in 'Dufrix', but a clear effect in 'Duplika'. In addition to growth depression, it was verified that rust also led to pronounced losses of leaf area as a result of reduced leaf size (leaf shrivelling) and accelerated defoliation.

INFECTION AND SYMPTOMATOLOGY

The *U. appendiculatus* uredospore infection process begins as a germ tube develops forming an appressorium upon physical contact with the edges of a stomatum (Pring 1980). The appressorium is induced by certain contact stimuli such as the stomatal outer lip or a scratch on a hydrophobic membrane (Staples 2000). The infection is most efficient on young leaves which have reached less than 70% of their final size (Harter and Zaumeyer 1941). An infection peg develops from the appressorium and pushes between the guard cells until the fungal cytoplasm is transferred into the substomatal vesicle (Mendgen and Hahn 2002). In most instances, only one infection hypha emerges from the substomatal vesicle. At the tip of the infection hypha, haustorial mother cell development is induced upon contact with a parachymatous cell (Mendgen 1978). The host cell is penetrated, a haustorium differentiates, and nutrients are transferred from the host to the haustorium and intracellular hypha (Mendgen 1979). Intracellular ramification proceeds throughout the host tissue, eventually forming a young uredium (Pring 1980).

The plant host physiology and biochemistry are affected during the pathogen infection. Respiration increases and photosynthesis decreases during infection, mainly after the sixth day (Raggi 1980). As reviewed by Stavely and Pastor-Corrales (1989), the activities of various enzymes such as peroxidase, catecholoxidase, glycolate oxidase, and glyoxalate reductase, increase during the infection. Quinines such as vitamin K, plastoquinones A, C, and O, and ubiquinones also increase during rust infection and development. In host hypersensitive reactions, deposition of tannins and death of affected host cells occur soon after infection. In general, infected plants become more sensitive to moisture stress as sporulation occur (Duniway and Durbin 1971).

In a recent proteomic study of *U. appendiculatus*, Cooper *et al.* (2006) used the 2-D nanoflow LC-MS/MS approach to identify more than 400 proteins in asexual uredospores. Knowledge of the proteins that differentiate life-cycle stages and distinguish fungal infection structures such as uredospores, germlings, appressoria, and haustoria are useful for understanding host-pathogen interactions. These proteins can also serve as targets for chemical inhibition of the fungus. According to the results obtained most of the proteins detected appear to have roles in protein folding or protein catabolism. Therefore, the authors present a model by which an abundance of heat shock proteins and translation elongation factors may enhance the spore's ability to survive environmental stresses and rapidly initiate protein production upon germination. It has also been verified that after germination of asexual uredospores there are changes in amounts of accumulated proteins involved in glycolysis, acetyl Co-A metabolism, citric acid cycle, ATP-coupled proton transport, and/or gluconeogenesis (Cooper *et al.* 2007).

The fungus *U. appendiculatus* may infect leaves, pods, and, rarely, stems and branches. Symptoms usually appear first on the lower leaf surface as minute, whitish slightly raised spots about six days after inoculation. These spots enlarge to form mature reddish brown uredia which rupture the epidermis about two days later. Sporulation begins and

the uredia may attain a diameter of 1-2 mm about 12 days after inoculation. In some cases, secondary and tertiary uredia develop around the perimeter of these primary uredia. The entire infection cycle occurs within approximately 12-15 days (Stavely and Pastor-Corrales 1989).

U. appendiculatus uredospores may be released passively from open uredia and scattered by farm implements, insects, animals, and, mainly, wind currents. Black teliospores may form in the uredium. The teliosori become dark brown to black as teliospores replace uredospores. The bean rust fungus is not seed transmitted.

CULTURAL PRACTICES, BIOLOGICAL AND CHEMICAL CONTROL

No single control or disease management measure can be recommended as the only most efficient or cost-effective to prevent rust infection in all cases or different regions. Management of bean rust has relied primarily on three strategies: application of fungicides, host resistance, and various cultural practices. In addition, biological control also has been reported as potentially useful.

Cultural practices were once thought to have only a small effect on rust disease severity, but when they are combined with other methods in an integrated disease management system, they play an important role (Schwartz 1984). Although cultural practices are effective on reducing the amount of initial infection, when environmental conditions are favorable, the rate of rust infection increases and the high mobility of the rust spores often offset the initial benefits. In addition, the extent to which agronomic practices can be modified to lessen rust severity depends on the flexibility of the cropping system and pest management systems (Paula-Junior and Zambolim 1998).

Cultural controls include crop rotation and removal of old plant debris which may bear viable uredospores and teliospores (Vieira *et al.* 2005). Reduced plant density may also reduce rust incidence. Planting dates may be adjusted in certain production areas to avoid or decrease the incidence of rust infection. Such adjustment will minimize exposure to moderate or cool temperatures and long dew periods during the critical preflowering to flowering stage of plant development (Stavely and Pastor-Corrales 1989).

Biological control or utilization of antagonistic microorganisms, which may be applied to the phylloplane of the plant, has been used to suppress or inhibit disease development (Spurr-Junior and Knudsen 1985). This strategy of disease control has not been effectively used for bean rust, despite it has been considered as promising in the past.

Bacillus subtilis (Cohn) Praznowski and other *Bacillus* spp. have showed to be promising bean rust antagonists when applied before inoculation with *U. appendiculatus* uredospores under greenhouse conditions. According to Baker *et al.* (1983), when *B. subtilis* was sprayed on field-grown beans three times per week it caused 75% reduction in rust severity. In a study conducted by Mizubuti *et al*. (1995), the number of pustules per leaf, spore production per leaf area and the viability of the *U. appendiculatus* spores were all significantly reduced by previous application of *B. subtilis* cells.

Allen (1982) and Grabski and Mendgen (1986) have showed that the fungus *Verticillium lecanii* (Zimm.) Viegas penetrates, invades, and kills uredospores and teliospores of *U. appendiculatus*, and colonizes pustules. A 68% decrease in bean rust infection was obtained under greenhouse condition, but little control was obtained in the field (Grabski and Mendgen 1986).

Using light and electron microscopy, Assante *et al.* (2004) studied the interaction between the mycoparasite *Cladosporium tenuissimum* and *U. appendiculatus* at the host-parasite interface. Uredospore germination decreased upon contact with ungerminated *C. tenuissimum* conidia, possibly due to antibiosis mechanisms. Mycoparasite hyphae grew within the host spore, emptied its content, and emerged profusely forming conidiophores and conidia. Complete control of the bean rust was achieved by application of *C. tenuissimum* culture filtrates but not by applying conidial suspensions.

According to Stavely and Pastor-Corrales (1989), the inoculation of specific bean genotypes with specific races of *U. appendiculatus* to which they are not susceptible will protect against other races to which they are susceptible. This phenomenon is called "cross-protection" (D'Arcy *et al.* 2001).

Chemical control has been a mainstay in intensive production areas where bean growers manage their crop for maximum yield and quality. Numerous fungicides are effective in controlling rust, but proper timing of fungicide applications, which is essential to improve economic returns, requires good disease monitoring and a weather forecasting system (Steadman and Lindgren 1983; Schwartz *et al.* 1984; Hall 1991; Lindgren *et al.* 1995; Paula-Junior and Zambolim 1998). Fungicides are, however, costly, and are generally not utilized in the subsistence production systems of Africa and Latin America, where most of the world's common bean production occurs (Pachico 1989; Wortmann *et al*. 1998; Broughton *et al.* 2003). The use of fungicides is also usually restricted to production for export markets, but even then, several fungicide applications are required and high production costs are often considered impractical and/or not sustainable (Steadman *et al.* 1995). In addition, the growing awareness of environmental degradation due to pesticides makes chemical control a controversial strategy.

Bean rust reduces yields more severely when infection occurs before flowering. Chemical control is, therefore, most effective during early plant development. The disease has been controlled by dusting plants every 7-10 days with sulfur at a rate of 25-30 kg/ha after pustules first appear. A spray schedule of every seven-to-fourteen days is recommended for other preventive chemicals such as chlorothalonil, maneb/manex, mancozeb, bravo/terranil, endure, and headline/quadris. Other effective chemicals utilized in the past or still in use by some countries are bitertanol, triadimefon, propriconazole, triphenylphosphite, and oxycarboxin (Stavely and Pastor-Corrales 1989; Paula-Junior and Zambolim 1998). New fungicides like the strobilurins and new triazoles are also recommended. Currently, we have used fungicides with active ingredients like epoxyconazole, azoxystrobin, and tebuconazol.

PATHOGEN DIVERSITY

U. appendiculatus is a highly variable and is among the most pathogenically variable of all plant pathogens (Stavely and Pastor-Corrales 1989). It has been identified and reported in all bean production areas of the world (Pastor-Corrales 2001) and is characterized by highly diverse virulence phenotypes (Harter *et al*. 1935, Ballantyne 1978; Stavely 1984a; McCain *et al*. 1990; Mmbaga *et al*. 1996b; Souza *et al.* 2005a). Recent studies by Araya *et al*. (2004) confirm previous reports on *U. appendiculatus v*ariability, and indicated that sexual recombination, heterokaryosis, mutation, gene flow, and environmental factors may be acting simultaneously on bean rust pathogen populations worldwide.

The classification of *U. appendiculatus* into physiological races and the consequent knowledge of its virulence diversity is a basic step towards understanding the dynamics of the pathogen distribution and as a guide to the development of resistant cultivars. In this step it is also possible to identify which pathogen isolates can be used to monitor the resistance genes introgression in breeding programs (Pastor-Corrales 2001; Pastor-Corrales and Stavely 2002; Souza *et al.* 2007a).

One of the main difficulties hampering advances in the study of the rust pathogen diversity was the inadequate definition of differential cultivars used for classification of the physiological races of *U. appendiculatus*. Between 1941 and 1983, classification was based on the differential series proposed by Harter and Zaumeyer (1941). However, this series was later modified in order to facilitate the discrimi-

Table 1 Common bean varieties adopted as standard differentials for classification of *Uromyces appendiculatus* physiological races at the 1983 Bean Rust International Workshop (Stavely *et al.* 1983)

Common Bean Variety			
1.	U.S. 3	11.	Ecuador 299
2.	California Small White 643	12.	Mexico 235
3.	Pinto 650	13.	Mexico 309
4.	Kentucky Wonder 765	14.	Brown Beauty
5.	Kentucky Wonder 780	15.	Olathe
6.	Kentucky Wonder 814	16.	AxS 37
7.	Golden Gate Wax	17.	NEP 2
8.	Early Gallatin	18.	Aurora
9.	Mountaineer White Half Runner[a]	19.	51051
10.	Redlands Pioneer	20.	CNC

[a]Deleted from the list because of its similarity to 'Kentucky Wonder 780' (Stavely 1984a).

nation of certain isolates (Fisher 1952; Dias-Filha and Costa 1968; Augustin and Costa 1971; Pereira and Chaves 1977; Ballantyne 1978).

During the "Bean Rust Workshop" (BRW), held in 1983, 35 researchers from different countries proposed a series of 20 cultivars as the international differential standard for *U. appendiculatus* (Stavely *et al.* 1983) (**Table 1**). In 1984, cv. 'Mountainer White Half Runner' was eliminated from this series because it was very similar to the 'Kentucky Wonder 780' (Stavely 1984a). Characterization of Brazilian isolates based on those 19 differential cultivars was accomplished by Mora-Nuñes *et al.* (1992), Santos and Rios (2000) and Souza *et al.* (2005a). In their work, Mora-Nuñes *et al.* (1992) concluded that eight out of the 19 cultivars ('Kentucky Wonder 814', 'Early Gallatin', '51051', 'NEP 2', 'Ecuador 299', 'Olathe', 'Mexico 309' and 'Redlands Pioneer') were sufficient to discriminate and classify isolates collected in Brazil. Using these eight cultivars, Faleiro *et al.* (1999a) characterized 13 races of this fungus in the Brazilian state of Minas Gerais.

Another aspect hindering the study of common bean rust was the use of different scales for evaluating the symptoms incited by the pathogen. Several authors proposed different evaluation scales (Harter and Zaumeyer 1941; Crispín and Dongo 1962; Davison and Vaughan 1963b; Stavely *et al.* 1983; Faleiro *et al.* 1999b). The scale proposed by Davison and Vaughan (1963b) was the most widely used throughout the world. In Brazil, modifications in this scale were proposed (Junqueira-Netto *et al.* 1969; Pereira and Chaves 1977; Carrijo *et al.* 1980). A standard scale of reaction grades was proposed by Stavely *et al.* (1983) which has been the most widely adopted (**Table 2**).

Besides the distinct differential series and evaluation scales, another factor hindering the classification of the physiological races of the fungus was the nomenclature attributed to them. The terminology used for this purpose was not uniform. Most authors arbitrarily designated the races by successive numbers (Harter and Zaumeyer 1941; Fisher 1952; Zúñiga and Victoria 1975; Stavely 1984a). In Brazil, the nomenclature was usually given by a number preceded by a capital letter that represented the geographical area where the races were identified (Dias-Filha and Costa 1968; Junqueira-Netto *et al.* 1969; Augustin and Costa 1971; Coelho and Chaves 1975; Carrijo *et al.* 1980). In Australia, Ballantyne (1978) attributed a lower case letter to each differential cultivar, whereas the designation was given by the letters corresponding to the differential cultivars to which the races were compatible.

In an attempt to facilitate the classification of *U. appendiculatus* races, Faleiro *et al.* (1999b) developed a simplified procedure that considered only the eight cultivars proposed by Mora-Nuñes *et al.* (1992). In addition, the authors proposed the use of an evaluation scale with three reaction degrees and a numeric system for the nomenclature of the races. By using this procedure, the authors grouped the 86 races that had been previously identified by Stavely (1984a), Mora-Nuñes *et al.* (1992) and Faleiro *et al.* (1999a) into 66 races.

During the 3rd BRW held in 2002, a new differential series was proposed for *U. appendiculatus* (Steadman *et al.* 2002). This series contained six Andean and six Mesoamerican bean cultivars (**Table 3**). In addition, a new binary nomenclature system was proposed for designation of the races, in which the evaluation scale was divided in only two reaction degrees: resistant and susceptible (Steadman *et al.*

Table 3 International differential series and the binary system of nomenclature adopted at the 3rd Bean Rust International Workshop as standard for classification of *Uromyces appendiculatus* physiological races (Steadman *et al.* 2002).

Gene Pool	Entry	Resistance Gene[a]	Binary System Value
Andean	A. Early Gallatin	*Ur-4*	1
	B. Redlands Pioneer	*Ur-13*	2
	C. Montcalm	*Ur-?*	4
	D. PC-50	*Ur-9*, *Ur-12*	8
	E. Golden Gate Wax	*Ur-6*	16
	F. PI 260418	*Ur-?*	32
Mesoamerican	A. Great Northern 1140	*Ur-7*	1
	B. Aurora	*Ur-3*	2
	C. Mexico 309	*Ur-5*	4
	D. Mexico 235	*Ur-3*$^{+}$	8
	E. CNC	*Ur-?*	16
	F. PI 181996	*Ur-11*	32

[a]See **Table 4** for references and information about the resistance genes; *Ur-?* = unnamed genes.

Table 2 The bean rust grading scale with the additional interpretative symbols adopted at the 1983 Bean Rust International Workshop (Stavely *et al.* 1983).

Grade[a]	Definition	Symbol[b]
1	Immune, having no visible symptoms	I
2	Necrotic or chlorotic spots, without sporulation, and less than 0.3 mm in diameter	HR
2+	Spots, without sporulation, 0.3-1.0 mm diameter	HR
2++	Spots, without sporulation, 1.0-3.0 mm diameter	HR
2+++	Spots, without sporulation, greater than 3.0 mm diameter	HR
3	Uredia less than 0.3 mm diameter	R
4	Uredia 0.3-0.5 mm diameter	MR
5	Uredia 0.5-0.8 mm diameter	MS
6	Uredia larger than 0.8 mm diameter	S
N+, N++, etc.	Necrotic spot of appropriate size surrounding uredosori of appropriate size	R
-3, -4, etc.		MR[c]

[a]When several reaction grades are present as evaluation results, they are recorded in order of predominance. Intensity is recorded separately, using the modified Cobb Scale (Stavely 1985).

[b]I = immune; HR = hypersensitive or highly resistance; R = resistance, reactions having any of the grades 2 with grade 3 present or predominant with some grade 4; MR = moderately resistance, grade 4 predominant and no grade 5 uredia; MS = moderately susceptible, uredia larger than grade 4, but none larger than grade 5; S = susceptible, grade 6 uredia (Stavely and Pastor-Corrales 1989).

[c]This reaction first described by Harter and Zaumeyer (1941) is characterized by a uredium in the center of a necrotic spot; whether R, MR, or other is determined by the size of uredium as described before.

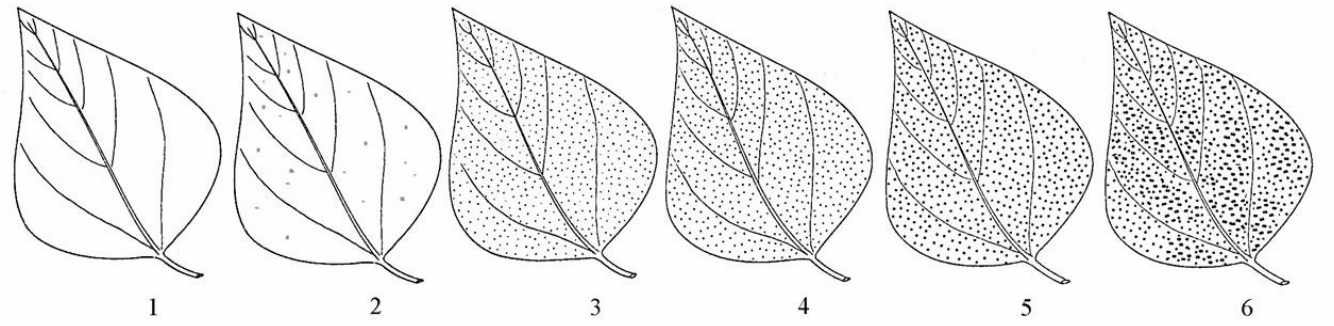

Fig. 2 Graphic diagram proposed by Castaño (1985) to symptom evaluation of the common bean rust. Scale: 1 - no pustules (immunity); 2 - necrotic spots without sporulation; 3 - pustules undergoing sporulation with a diameter less than 300 μm; 4 - pustules undergoing sporulation with a diameter ranging from 300 μm to 499 μm; 5 - pustules undergoing sporulation with a diameter ranging from 500 to 800 μm; 6 - pustules undergoing sporulation with a diameter greater than 800 μm. The plants that predominantly presented degrees 3 or lower are classified as resistant, whereas those with predominant degrees 4 or higher are considered susceptible.

2002). The reaction degrees to the disease are evaluated on the basis of Stavely *et al.* (1983). The lesions in both surfaces of the primary leaves can also be determined with the aid of a graphic representation diagram (Castaño 1985) (**Fig. 2**), as adopted by Souza *et al.* (2007a). Each race is designated by two numbers separated by a hyphen. The first number is obtained by the sum of the binary values attributed to the susceptible Andean cultivars of the set. The second number is obtained by the sum of the binary values of the susceptible Mesoamerican cultivars.

In the new differential series, cultivars 'Early Gallatin', 'Redlands Pioneer', 'Golden Gate Wax', 'Aurora', 'Mexico 309', 'Mexico 235' and 'CNC', which were proposed in the 1983 BRW, were maintained. Cultivars 'Montcalm', 'PC-50', 'PI 260418', 'Great Northern 1140' and 'PI 181996' were added to the new series. The wide adoption of this system can contribute to the elaboration of an internationally standardized classification methodology, facilitate the exchange of information, and the cooperative use of the results obtained by different research groups throughout the world.

Re-characterization of *U. appendiculatus* isolates collected in the USA, South Africa, Honduras, Argentina and Mozambique was already accomplished with the new procedure (Steadman *et al.* 2002; Acevedo *et al.* 2004; Jochua *et al.* 2004). Souza *et al.* (2007a) report the first work using the standard system for classification of *U. appendiculatus* physiological races in Brazil utilizing *U. appendiculatus* single-pustule isolates obtained from the fungal collection of BIOAGRO/UFV.

GENETICS OF HOST-PATHOGEN INTERACTION

Studies on variation patterns of the common bean seed protein phaseolin, alloenzymes and morphological evidences revealed the existence of a Mesoamerican and an Andean gene pool (Gepts *et al*. 1986; Singh *et al.* 1991a, 1991b). The Andean cultivars originated in the Andean region of South America, while the Mesoamerican beans were domesticated from wild populations in Mexico and the rest of Central America. Using both phenotype (virulence diversity) and genotype (RAPD markers) analysis of 90 *U. appendiculatus* isolates from thirteen Latin-American countries, Araya *et al*. (2004) were able to distinguish two major groups, namely the Andean and the Mesoamerican, and a third, the intermediate group. Although Andean and Mesoamerican isolates were virulent on landraces from either gene pool, more individual Andean isolates displayed greater regional or geographic specificity than Mesoamerican isolates, showing differential virulence to bean landraces from different gene pools. This phenomenon, previously also observed by Sandlin *et al*. (1999), demonstrates a clear differentiation of the pathogen population along similar lines as its host and suggests parallel evolution in the bean rust pathosystem. Intermediate virulence groups of *U. appendiculatus* races, observed by Braithwaite *et al.* (1994), Maclean *et al*. (1995), Sandlin *et al*. (1999), and Araya *et al*. (2004), provide evidence of a transition area between these two gene pools in both the common bean host and rust pathogen isolates. It is therefore possible that ongoing adaptation between pathogen and host is responsible for the characterization of these major groups (Araya *et al*. 2004).

The use of resistant cultivars is certainly the main component of the integrated pest management of bean rust. Pyramiding of resistance genes from both Andean and Mesoamerican gene pools is an important strategy for developing complementary and durable resistance to a large number of *U. appendiculatus* races (Stavely and Pastor-Corrales 1989; Pastor-Corrales and Stavely 2002; Araya *et al*. 2004). The large number of virulence patterns of *U. appendiculatus*, some of which are unique to certain countries, requires the use of specific resistance genes in different regions (Ballantyne 1978; Araya *et al*. 2004; Souza *et al*. 2005a; Liebenberg *et al*. 2006; Acevedo *et al*. 2008; Alleyne *et al.* 2008).

Several reports indicate that resistance to bean rust is controlled by major single dominant genes (Augustin *et al.* 1972; Ballantyne 1978; Christ and Groth 1982a; Sayler *et al*. 1995; Corrêa *et al*. 2000; Faleiro *et al*. 2000a, 2000b; Alzate-Marin *et al*. 2004; Souza *et al*. 2007b, 2007c), single recessive gene (Zaiter *et al.* 1989), two genes (Finke *et al*. 1986), two complementary dominant genes (Grafton *et al*. 1985), and by genes with minor effect (Edington *et al*. 1994). The gene-to-gene relationship has been shown to occur in the *U. appendiculatus–P. vulgaris* host-pathogen interaction (Christ and Groth 1982a, 1982b). Resistance genes effective against multiple races of the pathogen are organized in clusters or complex loci (Stavely 1984b; Stavely and Grafton 1985).

At least 13 dominant RR genes have been identified so far (genes *Ur-1* to *Ur-13*) (see **Table 4**). These genes have been named according to a nomenclature proposed by Kelly *et al*. (1996). Ballantyne (1978) proposed the first permanent symbols *Ur-1* and *Ur-2* for *Ur-A* and *Ur-B* present in the cultivars 'Gallaroy Genotype I' and 'Gallaroy Genotype II', respectively, and the symbol *Ur-2*^{2} for *Ur-E* (gene derivate from cultivar 'AxS 37'; 'AxS 37' = 'Actopan'/'Sanilac Selection 37'). Also, Ballantyne (1978) proposed the Symbol *Ur-3* for the gene *Ur-M* from cultivar 'Aurora'. In addition to these 13 genes, other unnamed RR genes, in 'BAC6' (Jung *et al.* 1996), 'Ouro Negro' (Corrêa *et al*. 2000; Faleiro *et al.* 2000a), 'Dorado' (Miklas *et al.* 2000, 2002), and 'PI 260418' (Pastor-Corrales 2005; Pastor-Corrales *et al.* 2008) have been identified. Genetic evidence supports that *Ur-3* is linked in repulsion to *Ur-11* (Stavely 1998), *Ur-4* and *Ur-5* are independent from each other and from *Ur-3* and *Ur-11* (Kelly *et al.* 1996); and *Ur-4* is independent from *Ur-6* (Stavely and Kelly 1996). Allelism tests showed that *Ur-ON* present in 'Ouro Negro' is distinct from genes *Ur-5* ('Mexico 309'), *Ur-11* ('Belmidak RR3', de-

Table 4 Rust resistance genes in common bean (*Phaseolus vulgaris*).

Resistance gene	Gene Pool[b]	Cultivar	LG[d]	Observation[e, f]
Ur-1	MA	B 1627 (Gallaroy Genotype I)	?	Discovered by Ballantyne (1978). 'Gallaroy' is derived by '643' x 'Sanilac'. *Ur-1=Ur-A*.
Ur-2	MA	B2090 (Gallaroy Genotype II)	?	Discovered by Ballantyne (1978). 'Gallaroy' is derived by '643' x 'Sanilac'. *Ur-2=Ur-B*.
Ur-2²	MA	B2055	?	'B2055' posses only the gene *Ur-E* derivates from 'AxS 37' ('Actopan' x 'Sanilac Selection 37', with genes *Ur-E* and *Ur-F*). *Ur-B* and *Ur-E* are allelic (Ballantyne 1978).
Ur-3	MA	Aurora[c]	B11	Discovered by Ballantyne (1978). 'Aurora' posses two genes linked (*Ur-M* and *Ur-N*); *Ur-M=Ur-3*. *Ur-3* is resistant to 43/87 races of the USDA-ARS Bean Project (Beltsville, MD, USA). It is also found in the Mesoamerican cultivars 'Mexico 235', 'NEP 2', and '51051'.
Ur-3⁺	MA	Mexico 235[c]	B11	Resistant to 43/87 races of the USDA-ARS Bean Project.
Ur-3	MA	NEP 2	B11	Resistant to 43/87 races of the USDA-ARS Bean Project. 'NEP 2' possess the genes *Ur-F*, *Ur-I*, *Ur-J* and *Ur-K*. *Ur-J* is allelic or closely linked in repulsion phase to gene *Ur-H* of 'Cornell 49242'. Gene *Ur-I* is allelic to *Ur-3* (Ballantyne 1978).
Ur-4	A	Early Gallatin[c]	B6	Discovered by Ballantyne (1978). *Ur-4=Ur-C*. Resistant to 30/87 races of the USDA-ARS Bean Project.
Ur-5	MA	Mexico 309[c]	B4	Block of eight tightly linked rust resistance genes found by Stavely (1984a). Resistant to 68/87 races of the USDA-ARS Bean Project.
Ur-6	A	Golden Gate Wax[c]	B11	Found by Ballantyne (1978) and Grafton *et al.* (1985). *Ur*-6=*Ur-G*. Is also found in cultivar 'Olathe'. Resistant to 15/87 races of the USDA-ARS Bean Project.
Ur-7	MA	Great Northern 1140[c]	B11	Discovered by Augustin *et al.* (1972). Is also found in cultivar 'Pinto US-5'. Resistant to 8/87 races of the USDA-ARS Bean Project.
Ur-8	A	U.S. 3	?	Discovered by Christ and Groth (1982a, 1982b). Resistant to 15/87 races of the USDA-ARS Bean Project.
Ur-9	A	PC-50[c]	B1	Discovered by Finke *et al*. (1986). Moderate susceptible to 75/87 races of the USDA-ARS Bean Project.
Ur-10	A/MA	Cape and Resisto	?	Discovered by Webster and Ainsworth (1988). It confers moderate resistant to 16/87 races of the USDA-ARS Bean Project.
Ur-11	MA	PI 181996[c]	B11	Discovered by Stavely (1998) as *Ur-3²*. Tightly linked with *Ur-3*. Resistant to 86/87 races of the USDA-ARS Bean Project.
Ur-12	A	PC-50[c]	B7	Discovered by Jung *et al.* (1998). Condition adult plant resistance (APR).
Ur-13	A	Kranskop	B8	Discovered by Lienberg and Pretorius (2004). 'Kranskop' shares an ancestor with 'Redlands Pioneer' (Lienberg *et al.* 2006).
Ur-13	A/MA(?)	Redlands Pioneer[c]	B8	Described by Lienberg and Pretorius (2004). Despite 'Redlands Pioneer' has been considered as an Andean common bean cultivar (Steadman *et al*. 2002), the gene *Ur-13* appears to be of Mesoamerican origin (Liebenberg *et al.* 2006).
Ur-?[a]	A	PI 260418[c]	?	Important Andean source from Bolivia (Pastor-Corrales 2005). Confers resistance 87/87 races of the USDA-ARS Bean Project. Tentatively named as *Ur-14*.
Ur-?	A	Montcalm[c]	?	Pedigree: 'Great Northern #1' x 'Dark Bed Kidney' (McClean and Myers 1990).
Ur-?	MA	CNC[c]	?	Composite of Guatemalan black beans (McClean and Myers 1990). One single gene conferring resistance to race 49 was detected by Rasmussen *et al*. (2002). Resistant to Andean races (Sandlin *et al*. 1999).
Ur-?	MA	Ouro Negro	B4	Discovered by Faleiro *et al*. (2000a, 2000b). Confers resistance to 13/13 Brazilian races (Faleiro *et al.* 1999) and 22/24 races tested in the USDA-ARS Bean Project (Alzate-Marin *et al*. 2004). Temporary named as *Ur-OuroNegro* or *Ur-ON*.
Ur-?	MA	Dorado (DOR 346)	B4	Reported by Miklas *et al.* (2000). Gene temporary named as *Ur-Dorado108* which confers resistance to races 108 of the USDA-ARS Bean Project (Miklas *et al.* 2002).
Ur-?	MA	Dorado (DOR 346)	B11	Reported by Miklas *et al.* (2000). Gene temporary named as *Ur-Dorado53* which confer resistance to races 53 of the USDA-ARS Bean Project (Miklas *et al.* 2002).
Ur-?	MA	BAC6	B11	Described by Jung *et al.* (1996). Gene temporary named as *Ur-BAC6* (Miklas *et al.* 2002).

[a] *Ur-?* = unnamed genes.
[b] Andean (A), Mesoamerican (MA).
[c] Differential cultivar (Steadman *et al*. 2002).
[d] The linkage groups designated as B1-to-B11 in the BJ common bean core map (Freyre *et al*. 1998; Miklas *et al*. 2002; Kelly *et al*. 2003; Miklas *et al*. 2006) correspond to the *P. vulgaris* chromosomes 1-to-11, respectively (Pedrosa *et al.* 2003, 2006, 2008).
[e] The Bean Improvement Cooperative BIC, List of genes *Phaseolus vulgaris* L., 2008; prepared by T.G. Porsch. Available online: http://www.css.msu.edu/bic/PDF/Bean%20Genes%20List%202008.pdf.
[f] USDA-ARS, National Genetic Resources Program, Germplasm Resources Information Network GRIN; Online Database: http://www.ars-grin.gov/cgi-bin/npgs/html/dno_eval_acc.pl?83042+490345+13.

rived from 'PI 181996') (Alzate-Marin *et al.* 2004), and *Ur-3⁺* ('Mexico 235') (Souza *et al.* 2007c). Most genes characterized so far confer resistance to multiple races of *U. appendiculatus*, corroborating the evidence that they are organized in clusters of race-specific genes.

Several RAPD markers associated with genes conferring resistance to rust in common bean have been identified, as described in **Table 5**. Many SCAR markers have been developed to increase the reproducibility of the RAPD molecular markers (**Table 5**). These molecular markers have been used for mapping *Ur* genes in the integrated common bean map (**Fig. 3**).

The groups of Mesoamerican genes *Ur-5/Ur-Dorado53/Ur-ON* and *Ur-3/Ur-7/Ur-11/Ur-Dorado108/Ur-BAC6* have been mapped in linkage groups (LG) B4 and B11, respectively. The genes *Ur-3* and *Ur-11*, and also the gene *Ur-Dorado108* map to the end of LG B11, located next to the *Co-2* locus, which is related to resistance to anthracnose. The gene *Ur-BAC6* is located near to the *Ur-7* locus, and they do not appear to be close to *Ur-Dorado108*, *Ur-3*, and *Ur-11* loci on LG B11. The Andean genes *Ur-4*, *Ur-6*, *Ur-9*, *Ur-12*, and *Ur-13* were mapped to LG B6, B11, B1, B7, and B8, respectively (Miklas *et al.* 2002; Kelly *et al.* 2003; Miklas *et al.* 2006; Wright *et al*. 2008). Park *et al*. (2008) observed a possible allelic interrelation between *Ur-7* present in Mesoamerican cultivar 'Great Northern 1140' and *Ur-6*

Table 5 Molecular markers linked to rust resistance genes in common bean (*Phaseolus vulgaris*).

Molecular marker	Product size (bp)	Distance (cM)	Linkage phase	Resistance gene	Resistance source	SCAR	LG[a]	Reference[b]
RAPD K14	620	2.2	Coupling	*Ur-3*	NEP 2	SCAR K14	B11	Haley *et al.* 1994; Nemchinova and Stavely 1998
RAPD A14	1,100	0.0	Coupling	*Ur-4*	Early Gallatin	-	B6	Miklas *et al.* 1993
SCAR A14	1079/800	0.0	Codominant	*Ur-4*	BelMiDak-RR-9 and BelMiDak-RMR-11	-	B6	Mienie *et al.* 2004
RAPD F10	970	2.1	Coupling	*Ur-5*	B-190	-	B4(?)	Haley *et al.* 1993
RAPD I19	460	0.0	Coupling	*Ur-5*	B-190	SCAR I19	B4	Haley *et al.* 1993; Melotto and Kelly 1998
SCAR I19	460	3.31	Coupling	*Ur-5*	Mexico 309	-	B4	Souza *et al*. 2007b
RAPD BC06	308	1.3	Coupling	*Ur-6*	Olathe	SCAR BC6	B11	Park *et al.* 2003a, 2004
RAPD AG15	300	2.0	Coupling	*Ur-6*	Olathe	-	B11(?)	Park *et al.* 2003a, 2004
RAPD AY15	200	7.7	Repulsion	*Ur-6*	Olathe	-	B11(?)	Park *et al.* 2003a, 2004
RAPD AA11	500	0.0	Coupling	*Ur-7*	GN1140	-	B11(?)	Park *et al.* 1999a, 2003b
RAPD AD12	537	0.0	Coupling	*Ur-7*	GN1140	SCAR AD12	B11	Park *et al.* 1999a, 2008
RAPD AF17	900	0.0	Coupling	*Ur-7*	GN1140	-	B11(?)	Park *et al.* 1999a
RAPD AB16	850	2.2	Coupling	*Ur-7*	GN1140	-	B11(?)	Park *et al.* 1999a
RAPD AD9	550	2.2	Coupling	*Ur-7*	GN1140	-	B11(?)	Park *et al.* 1999a
RAPD AB18	650	2.4	Repulsion	*Ur-7*	GN1140	-	B11(?)	Park *et al.* 1999a
RAPD J13	1,100	5.0	Coupling	*Ur-9*	PC-50	-	B1	Jung *et al.* 1998
RAPD A04	1,050	8.6	Coupling	*Ur-9*	PC-50	-	B1	Park *et al.* 1999b
RAPD AC20	490	0.0	Coupling	*Ur-11*	PI 181996	-	B11(?)	Johnson *et al.* 1995
RAPD AE19	890	6.2	Repulsion	*Ur-11*	PI 181996	-	B11(?)	Johnson *et al.* 1995
RAPD AE19	890	1.0	Repulsion	*Ur-11*	BelMiDak RR-3	SCAR AE19	B11	Souza *et al.* 2002; Queiroz *et al.* 2004c; Liebenberg *et al.* 2008
RAPD GT02	450	0.0 and 5.4	Coupling	*Ur-11*	BelMiNeb1 and BelMiNeb3	SCAR UR11-GT2	B11	Boone *et al.* 1999
SCAR SQ4	1,440	?	Coupling	*Ur-11/Co-2*	PI181996/Cornell 49-242	-	B11	Awale *et al*. 2008
SCAR KB126	430/405	1.6	Codominant	*Ur-13*	Kranskop	-	B8	Mienie *et al*. 2005
SCAR KB85	310/288	9.2	Codominant	*Ur-13*	Kranskop	-	B8	Mienie *et al*. 2005
SCAR KB4	436, 250/186	13.8	Codominant	*Ur-13*	Kranskop	-	B8	Mienie *et al*. 2005
RAPD AJ16	250	12.5	Coupling	*Ur-BAC6*	BAC6	-	B11	Jung *et al*. 1996
RAPD F10	1,072	7.0	Coupling	*Ur-ON*	Ouro Negro	SCAR F10	B4	Corrêa *et al.* 2000; Faleiro *et al*. 2000a
RAPD BA08	530	6.0	Coupling	*Ur-ON*	Ouro Negro	SCAR BA8	B4	Corrêa *et al.* 2000; Faleiro *et al*. 2000a
RAPD X11	550	5.8	Coupling	*Ur-ON*	Ouro Negro	-	B4(?)	Faleiro *et al*. 2000a
TRAP F7R1	150	3.0	Coupling	*Ur-115M* (*Ur-5?*)	115M	-	B4(?)	Wright *et al.* 2008

[a] The linkage groups designated as B1-to-B11 in the BJ common bean core map (Freyre *et al*. 1998; Miklas *et al*. 2002; Kelly *et al*. 2003; Miklas *et al*. 2006) correspond to the *P. vulgaris* chromosomes 1-to-11, respectively (Pedrosa *et al.* 2003, 2006, 2008).
[b]Another consulted source: The Bean Improvement Cooperative - BIC, SCAR markers linked with disease resistance traits in common bean - *Phaseolus vulgaris*; updated on May, 2008. Available online: http://www.css.msu.edu/bic/PDF/SCAR%20Markers%202008.pdf.

present in Andean cultivar 'Olathe', based on the fact that the band generated by SCAR AD12 linked to *Ur-7* was also amplified a DNA fragment for cultivar 'Olathe'.

Clustering is also observed when the positions of RR genes are compared with those conferring resistance to anthracnose (*Co*) and BCMV (Miklas *et al*. 2006) (**Fig. 3**). For instance, the Andean RR gene *Ur-9* and the anthracnose resistance gene *Co-1* co-localized on LG B1 (Kelly and Vallejo 2004; Miklas *et al.* 2006). The Mesoamerican genes *Ur-5* and *Co-3*/*Co-9*, and gene *Ur-ON* from cultivar 'Ouro Negro' and *Co-10* co-localized on LG B4 (Faleiro *et al.* 2000b; Alzate-Marin *et al*. 2003), and *Ur-3* co-localized with *Co-2* on LG B11, suggesting that these genes derived from common ancestral gene sequences (Geffroy *et al*. 1999; Faleiro *et al.* 2000b, 2003; Miklas *et al.* 2006). Recent works show that SCAR SQ4 linked to the *Co-2* anthracnose resistance gene is closely linked to *Ur-11* (Awalle *et al.* 2008). According to Geffroy *et al*. (1999) and Liebenberg *et al*. (2006) the knowledge of the positions of resistance genes, whether singly or in clusters, and analysis of the composition of these clusters, will contribute to understanding of the mechanisms and time-span involved in the co-evolution of pathogen and host resistance. The linkage groups designated as B1-to-B11 in the BJ common bean core map correspond to the chromosomes 1-to-11, respectively (Pedrosa *et al.* 2003, 2006, 2008).

The proper characterization of the RR genes is essential for the pyramiding of genes from Mesoamerican and Andean gene pools in order to broaden the spectra of the RR genes presently used (Liebenberg *et al*. 2006; Pastor-Corrales *et al.* 2008).

RUST CONTROL BY PLANT RESISTANCE

The main goals of common bean breeding programs throughout the world are to increase on yield and disease resistance. Genetic resistance associated with disease control management techniques is the most effective, inexpensive and ecologically correct strategy for controlling common bean diseases such as rust (Stavely and Pastor-Corrales 1989; Paula-Junior and Zambolim 1998).

In the last few decades, DNA markers have been used to assist different steps of common bean breeding programs aimed at developing cultivars resistant to rust. Isozymes and DNA-based markers have been used to study the genetic diversity of the rust fungus (Lu and Groth 1988; Linde *et al.* 1990a, 1990b; McCain *et al.* 1992; Groth *et al.* 1995; Maclean *et al.* 1995; Faleiro *et al.* 1998) and also for mapping

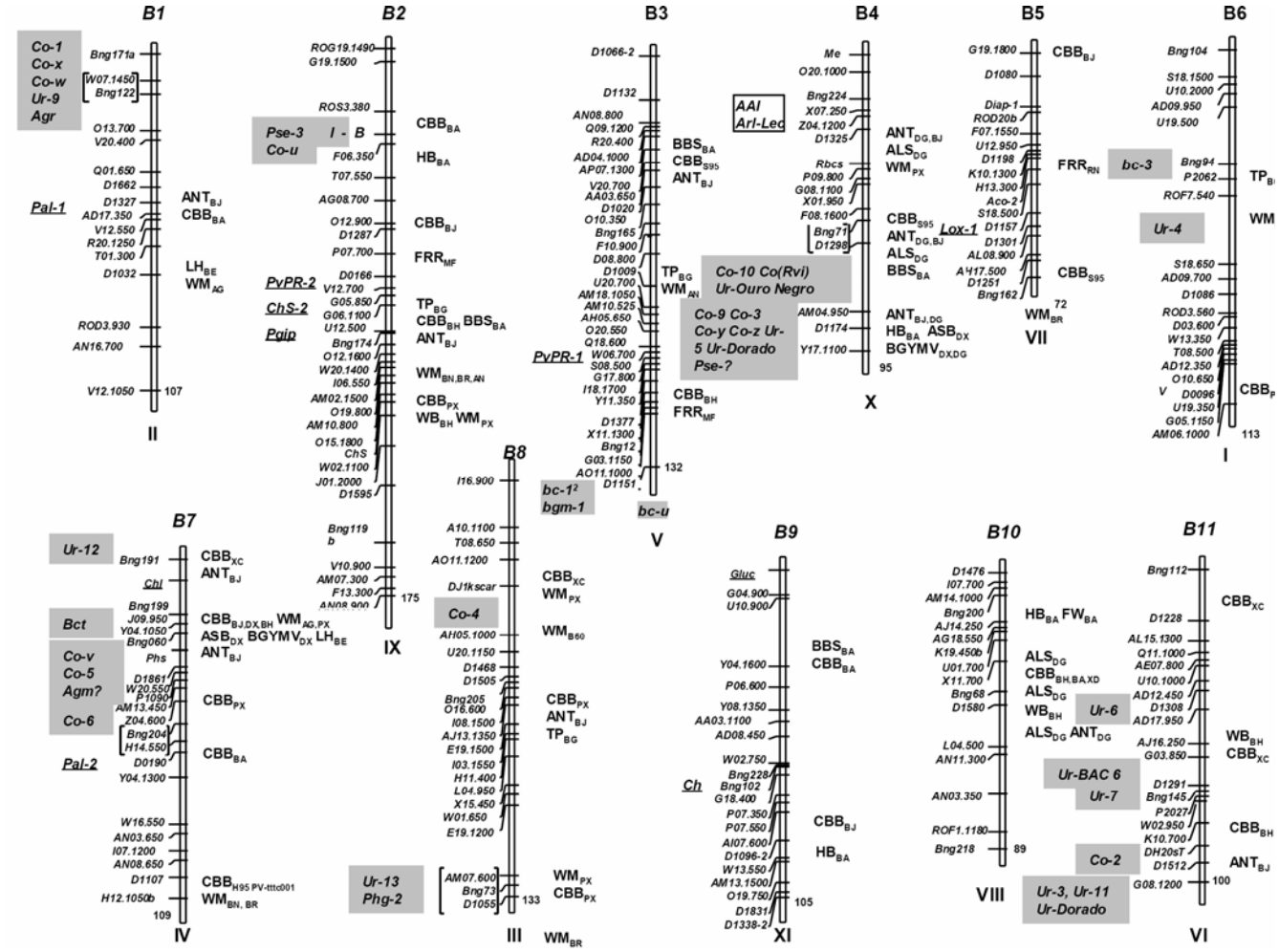

Fig. 3 Comprehensive genomic map of disease and insect resistance genes and QTL in common bean (Miklas *et al*. 2006). The linkage groups correspond to the core map version of Freyre *et al*. (1998), and resemble the maps presented by Kelly *et al*. (2003), and Kelly and Vallejo (2004). Directly to the left of each linkage group are the framework molecular markers (smaller font), the monogenic disease resistance genes (shaded boxes), defense-related genes (underlined), and arcelin, lectin and α-amylase inhibitor genes (clear box). The *Co* are anthracnose resistance loci, *Ur* rust resistance loci (*Ur-Dorado*, *Ur-Ouro Negro*, and *Ur-BAC 6* refer to the line source of unnamed genes), *Pse* halo blight resistance loci, *I* and *bc* are dominant and recessive genes respectively for resistance to BCMV, *Phg* angular leaf spot resistance locus, and *Bct* is a locus for resistance to BCTV (Miklas *et al*. 2006). The linkage groups designated as B1-to-B11 in the BJ common bean core map correspond to the chromosomes 1-to-11, respectively (Pedrosa *et al.* 2003, 2006, 2008).

and characterizing resistance genes to *U. appendiculatus* and other important bean pathogens (Haley *et al.* 1993; Miklas *et al.* 1993; Stavely 2000; Faleiro *et al.* 2000a; Caixeta *et al.* 2003; Faleiro *et al.* 2003; Kelly *et al.* 2003; Queiroz *et al.* 2004a, 2004b, 2004c; Souza *et al.* 2005c, 2005d, 2007b).

As already mentioned in this review, the RR genes are race-specific and the interaction of their expression products with the pathogen follow the gene-to-gene theory proposed by Flor (1971). The co-evolution between the common bean and the rust pathogen led to the diversification of both the resistance genes in the host and the avirulence genes in the pathogen.

Considering the high genetic and physiological variability of *U. appendiculatus*, the combination of different RR genes in the same variety is an important strategy for obtaining effective and durable genetic resistance to rust (Johnson 1984; Stavely and Pastor-Corrales 1989; Kelly *et al.* 1995; Souza *et al.* 2005c, 2007b). This combination can be accomplished by gene pyramiding, when the genes are combined in the same cultivar, or by using multilines, when different genes are transferred to different lines of the same cultivar (lines with the same genetic background but harboring distinct RR genes). Different breeding strategies can be used to combine resistance genes, however, for gene pyramiding, knowledge about the organization of the genes and also the use of proper selection tools are of paramount importance.

In autogamous species the allelic organization of the genes prevent the use of more than one allele of a specific gene in the same cultivar unless alternative alleles are incorporated in the cultivar by recombinant DNA techniques and transformation. On the other hand, the organization of tightly linked genes allows for the recombination of more than one gene in the same chromosome and their pyramiding by conventional breeding methods.

Developing a strategy for the selection of resistance genes depends on their organization in the genome. The introduction of different genes closely linked in a genic block is easier than the introduction of different genes which are dispersed in the genome. However, during the gene pyramiding process, the association of different resistance genes present in distinct genitors in the same genic block would be more difficult than the association of the same genes if they are located in different regions of the genome (Michelmore 1995).

Selection for resistance genes can be accomplished by inoculation of segregating progenies with spores of a single race or from a mixture of races. When a single race is used one specific resistance gene will be selected (vertical resistance), and other genes with secondary effect may be lost during the breeding process. This type of genetic erosion is critical when dealing with pathogens presenting high genetic variability like *U. appendiculatus*. Inheritance studies have indicated that major and minor genes are involved with resistance to this pathogen (Faleiro *et al.* 2000a, 2000b;

Souza *et al.* 2002; Faleiro *et al.* 2003). In this case, selection using one single race will inevitably lead to loss of genes of minor effect along the breeding process.

Gene pyramiding using only conventional breeding methods has not been effective mainly due to the difficulties in selecting genotypes harboring different resistance genes which demand multiple or serial inoculations of the same plant or population (Michelmore 1995). This limitation affects the breeding process as a whole and also decreases the accuracy and efficiency of the selection process (Bigirimana and Höfte 2001). Epistatic interactions between different resistance genes can also affect the selection process (Singh *et al.* 2001).

Pyramiding of resistance gene has been proposed as a control measure mainly for pathogens with high genetic and physiological variability (Coyne and Schuster 1975; Miklas *et al.* 1993; Kelly and Miklas 1998; Faleiro *et al.* 2004; Souza *et al.* 2005c), like *U. appendiculatus*. Monogenic resistance is often overcome by new races of the pathogens which appear in the growing regions (Stavely and Pastor-Corrales 1989). Pyramiding of individual resistance genes or of gene blocks can be used for obtaining resistance to the same pathogen (durable resistance) (Kelly and Miklas 1998; Faleiro *et al.* 2000a; Souza *et al.* 2005c, 2007b), or to different pathogens (multiple resistance) (Faleiro *et al.* 2004; Ragagnin *et al.* 2005). One of the main limitations of the method is the proper selection of plants containing all the alleles of interest. In the pyramiding of resistance genes for the same pathogen the phenotype could be the same whether one or more R genes are present in the host. In the other situation, the pyramiding of resistance genes for different pathogens, the main limitation is the screening of each single plant simultaneously for different pathogens. A more difficult situation is found when one intends to pyramid major genes and minor genes as the former can mask the effect of the latter (Faleiro *et al.* 2004; Souza *et al.* 2005c). These limitations can be overcome by the use of molecular markers linked to the resistance genes. However, for each resistance allele a specific marker or markers need to be identified. The use of flanking markers tightly linked to the locus of interest makes selection even more robust (Faleiro *et al.* 2003). Other problems associated with the use of gene pyramiding process for the development of cultivars resistant to pathogens are the high-cost and hard work, the time defendant, and the difficulties in transfer quickly the genes of interest to new commercial cultivars.

Experimental evidence demonstrates that gene pyramiding confers more effective resistance to the host plant than that conferred by the sum of the resistance present in the progenitor plants (Yoshimura *et al.* 1995; Huang *et al.* 1997; Singh *et al.* 2001). According to Schafer and Roelfs (1985), the probability that a pathogen will overcome a gene pyramid of four to six genes is extremely low. In order for this to happen, independent mutations in the pathogen genome must occur and they should be combined in the same genetic background, or they could occur simultaneously or sequentially in the genome of a specific pathogen isolate. Nelson (1979) argues that resistance resulting from the partial action of several resistance genes exerts a low selection pressure on the pathogen and for this reason it tends to last for a long period of time. Although this concept is not fully accepted by the scientific community there are experimental data supporting the existence of partial effects of different resistance genes in some pathosystems (Brondy *et al.* 1986; Pedersen and Leath 1988). According to the theory presented the duration of resistance will depend on the number of genes to be overcome by the pathogen.

Epidemiology data also support the use of gene pyramiding as an effective strategy for disease control. By studying the pathosystem *Melampsora lini-Linum marginale* in Australia, Thrall and Burdon (2003) demonstrated that there is an inverse correlation between pathogen fitness, as measured by the number of spores produced, and the number of avirulence genes present in its genome. The authors observed that the pathogen populations which were able to infect a greater number of host populations were less aggressive than pathogen populations which were able to infect a lower number of host populations. This indicates that the inactivation of several avirulence genes in the pathogen compromises its adaptability. This is a positive aspect from the epidemiological perspective because it indicates that gene pyramiding can potentially keep the disease below an economical damage level and also prevent its fast dissemination.

Molecular markers are often used to aid gene pyramiding during the breeding process (Miklas *et al.* 1993; Stavely 2000; Kelly *et al.* 2003; Ragagnin *et al.* 2005; Souza *et al.* 2007b). This allows the proper identification of the different resistance alleles present in one specific genotype. With the use of molecular markers not only the multiple and sequential inoculations can be avoided but also the confounding effect of potential epistatic interaction that might happen among the different resistance genes present in the same genetic background (Michelmore 1995; Bigirimana and Höfte 2001; Singh *et al.* 2001; Toenniessen *et al.* 2003).

The main steps of a MAS gene pyramiding breeding program aiming at disease resistance are: (i) identification of the most prevalent and virulent races of the pathogen in the region of interest and characterization of the most promising resistance sources for that region; (ii) determination of the disease resistance inheritance mode by crossing the resistance sources and the susceptible cultivar; (iii) identification of molecular markers tightly linked to the various disease resistance alleles; (iv) development of lines harboring the R genes and the molecular markers of interest; this process is often done by backcrossing; (v) identification of markers that can specifically identify the resistance alleles to avoid false positives; and (vi) pyramiding of resistance alleles by intercrossing the lines obtained. During this process, the following activities must also be considered: (i) continuous characterization of the variability of the pathogen and the host; (ii) characterization and introduction of new resistance sources in the breeding program; and (iii) identification of molecular markers linked to the resistance genes present in the new resistance sources (Alzate-Marin *et al.* 2005).

Pyramiding of disease resistance genes has been successfully accomplished by common bean breeding programs. Kelly *et al.* (1995) reported the pyramiding of five resistance alleles (*I*, *bc-u*, *bc-1*^{2}, *bc-2*^{2} and *bc-3*) which confer resistance to bean common mosaic virus (BCMV). The USDA-ARS Bean Project, in Beltsville, MD, USA, in collaboration with Experimental Stations in Michigan, Nebraska and North Dakota developed 52 bean lines with genes conferring resistance to BCMV and/or to rust, with distinct allelic combinations and different genetic backgrounds (Pastor-Corrales 2003). Other common bean and also cowpea lines with gene pyramids for one or more diseases have been reported (Beaver *et al.* 2003; Coyne *et al.* 2003; Kelly *et al.* 2003).

In the Common Bean Breeding Program of the BIOAGRO-UFV, molecular markers were used to assist the transfer of rust and anthracnose resistance genes from the black seeded cultivar 'Ouro Negro' ('Honduras-35') to the "carioca-type" cultivar 'Rudá' (Faleiro *et al.* 2004). Ragagnin *et al.* (2005) expanded these efforts and transferred genes for resistance to rust (*Ur-ON*), anthracnose (*Co-4, Co-6* and *Co-10*) and angular leaf spot (*Phg-1*) to the "carioca-type" cultivars 'Rudá' and 'Pérola'.

In the specific case of rust, the breeding program conducted at BIOAGRO/UFV is also using the MAS approach for development of lines with specific RR genes *Ur-ON*, *Ur-5*, and *Ur-11* aiming posterior introgression and pyramiding in Brazilian commercial cultivars (Alzate-Marin *et al*. 2004; Faleiro *et al*. 2004; Ragagnin *et al*. 2005; Souza *et al*. 2005c, 2007b). Until recently, the gene *Ur-ON* has been used as the only source for resistance to *U. appendiculatus* in that breeding program. The RAPD marker X11 (Faleiro *et al.* 2000a) and the SCAR markers F10 and BA08 have been used for its indirect selection (Corrêa *et al.* 2000).

Later, another RR gene was characterized, the gene *Ur-11*, which was then also introgressed into the 'Rudá' background (Souza *et al.* 2002). Aiming at assisted selection of *Ur-11*, the RAPD marker AE19 was validated in a F_2 population derived from the cross 'Rudá' × 'Belmidak RR-3' (Alzate-Marin *et al.* 2004). Later, this marker was converted into a SCAR marker (SCAR AE19) by Queiroz *et al.* (2004c). In the study of Souza *et al.* (2007b) the SCAR marker SI19 was validated as linked to gene *Ur-5* from cultivar 'Mexico 309'. It was also verified that this marker can be used for the indirect selection of gene *Ur-5* in the presence of genes *Ur-ON* and *Ur-11*.

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