Bean rust, caused by the autoecious, macrocyclic, obligately parasitic rust fungus, *Uromyces appendiculatus*, is a major disease in many common bean production areas of the earth (2,23). Although the repeating spore stage, in which pustules with the rusty, brown urediniospores are produced that give this disease its common name is most commonly associated with rust, the black teliospores also are produced in most aging infections. The basidiospore, pycniospore, and white aeciospore stages are observed more rarely, but occur in many bean production areas of the United States and other places (17,23,31) and are indicative of genetic recombination in this fungus (2,23). Uredinal pustules can occur on all green areas of bean plants (23). Although rust has caused severe crop losses in the United States in some years, in other years only minor losses occur. Six to eight hours of wetness are required for infection to occur at the optimal infection temperature, about 20°C (23). So rust is commonly found in humid areas with sufficiently long dew periods, but it is rarely found in arid climates.

Control of bean rust is enhanced by crop rotation, sanitation, and early planting to avoid the longer late summer dew periods. Several fungicides control rust, but most efficient control occurs with host resistance (23). However, achievement of resistance that will be effective over time and distance is difficult due to the high degree of pathogenic variability in this fungus. Individual field collections of urediniospores, especially from rust susceptible dry beans, but also sometimes from snap beans, frequently contain several pathogenic races. Many such races have been identified in several major bean producing countries (5,13,23). Ninety such races have been isolated, identified, and maintained in liquid nitrogen storage at Beltsville since 1980. Most of these races are from United States field collections, but some are also from Africa, Asia, Europe, and other countries of the Americas. Descriptions have been published for many, but not all of these 90 races (1,5,13,19,20,22,28). Most of these races have only been found once, some a few times, but a few others, such as race 38 on bush snap beans and race 54 on pinto, navy, great northern and some other classes of dry beans are found frequently in the United States (16,19,28,30). These pathogenic races are defined by the descriptive reaction grades for the symptoms produced on each of a standard set of 19 differential cultivars. These reaction grades are assigned values of one to six that are representative of the various kinds of responses (23,25). Resistance to bean rust is expressed as immunity, with no visible symptoms (grade 1), chlorotic or necrotic, nonsporulating spots (grades 2, 2^-, 2^+, or 2^++) or as tiny, sporulating uredinia (grades 3 or 3,4 (23,25).

Many rust resistance genes are present in common bean for which the occurrence of a large number of pathogenic races is evidence. All such identified genes are dominant. A list of ten named rust resistance genes was assembled, defined, and published in 1996 as *Ur-1* through *Ur-10* (10). Since then a very important gene from plant introductions (PIs) 181996 and 190078 has been identified as *Ur-11* (20). Among these genes, this author has used *Ur-3, -4, -5, -6, and -11* in developing rust resistant germplasm and they control 44, 30, 70, 22, and 89 of the 90 races,
respectively (21,23,26,27). There is evidence from the rust reactions to these 90 races of the differential cultivars, other cultivars, and PIs that there are many, some broadly race effective, other unnamed resistance genes (1,5,13,19,22,23,28). By introgression of genes effective against multiple races, germplasm lines and cultivars can be developed that have multiple genes for resistance to each pathogenic race. Recently released BelDakMi-Rust and Mosaic Resistant-18 pinto is homozygous for the Ur-3, -4, -6, and -11 rust resistance genes, two of which, Ur-3 and -4, are effective against race 108, the only one of our 90 races not controlled by Ur-11. All other 89 races are controlled by Ur-11 and the other three genes provide one or two additional genes effective against most of these races.

Molecular random amplified polymorphic DNA (RAPD) markers and in a few cases sequence characterized amplified region (SCAR) markers have been identified for the Ur-3, -4, -5, -7, and -11 rust resistance genes (3,7,8,9,12,14,15). These markers are useful for identifying plants having these genes. However, these markers need be used with caution because false positive or negative results can be obtained due to insufficient closeness of the linkage between marker and gene as is apparently the case with IM (7,14) or lack of applicability to a major subgroup of the bean host, as occurs with Ur-4 in Andean beans (12).

At Beltsville, in this author's germplasm development program, controlled, multiple, individual race inoculations are still employed for maximum reliability (18). Each of the two primary leaves of each plant are inoculated with each of four selected, key races of the pathogen. Races are selected that will produce well proven reactions in the presence of pertinent, desired resistance genes. For instance, the Ur-3 gene produces a grade 2 reaction to the 44 races it controls. This Ur-3 reaction is epistatic to the grade 3,2 reaction produced to most of these races when Ur-11 is present. So race 53, which is controlled by Ur-3 and Ur-11, but not by Ur-4 or -6, can be used for detecting Ur-3. The Ur-6 gene produces grades 1 or 2 reactions that are epistatic to the grade 3,2 reaction usually produced by Ur-11, so race 47, which is controlled by Ur-6 and -11, but not by Ur-3 or -4 is used to detect Ur-6. The Ur-4 gene results in production of a distinct necrotic 2, 2 or 2,2 reaction by race 49 and 29 other races in the absence of other resistance genes. Race 49 produces the grade 3,2 reaction if Ur-11 is present without Ur-4, but if both Ur-4 and -11 are present a faint, chlorotic, grade 2 reaction is present. So race 49, which is not controlled by Ur-3 or -6 can be used to detect presence of Ur-4 with Ur-11. Race 67 is controlled by Ur-11 to produce a 3,2 reaction grade and is not controlled by Ur-3, -4, or -6 (28), so this race is always included in inoculations to detect presence of Ur-11. In addition to races 47, 49, 53, and 67, four additional appropriate races are always included for confirmatory data, one of which is race 108. In development of bean germplasm with the Ur-3, -4, -6, and -11 rust resistance genes and in all other resistant germplasm developed in this project, all F1 plants and every plant in all subsequent generations are inoculated with a least eight, and in some cases 12 individual pathogenic races of U. appendiculatus (23). For production of F1 plants, the more resistant parent is always used as the pollen parent in order to use the rust reactions of the F1 to confirm or deny whether the plant is from a cross or selfing, because all known rust resistances are dominant. Once a population of plants is obtained that is apparently homozygous for all desired rust resistance genes, subsequent progeny are tested with many additional key races of U. appendiculatus to confirm resistance to all available races.

For combining genes for resistance to bean common mosaic (BCMV), bean common mosaic necrosis (BCMNV), and bean golden mosaic (BGMV) viruses with rust resistance genes, inoculation and RAPD or SCAR markers are regularly employed. Rust inoculations are
performed when the primary leaves of seedlings are one to two third expanded and the inoculated plants are incubated in a dew chamber at 20°C overnight. The plants are then moved to a greenhouse at about 24°C and inoculated on that or the next day upon one of the monofoliolate leaves with a strain of BCMV, such as NL4 or US5, and on the other, paired leaf with BCMNV strains NL3 or NL5 (11,24). If the recessive bc-1^2, bc-2^2 or bc-3 genes are homozygous, moderately large, small, or no local lesion symptoms, respectively, will develop within less than 14 or 15 days when rust and mosaic symptoms are recorded (4). If the dominant I BCMV resistance gene is present without any of these recessive genes, the plant dies. Mosaic symptoms develop if the plant does not have one of the numbered homozygous recessive genes with bc-u or I and in the case of the US5 strain if a recessive gene is homozygous in the absence of I (11). If one of the recessive genes is present with I, it seems most practical to test for presence of I by using the very reliable RAPD or a SCAR primer-marker (6).

For BGMV resistance, only the excellent codominant RAPD marker is used for bgm-1 at Beltsville, because this virus does not yet occur north of Florida in the United States (29). This marker for the recessive bgm-1 gene is particularly useful because plants heterozygous for this gene as well as those homozygous for it in dominant or recessive condition are detectable. By use of this RAPD primer, it was possible to develop the first snap bean homozygous for bgm-1 along with rust resistance (27). As more resistance genes are identified for additional diseases, marker technology seems likely to enable development of lines and cultivars with combined resistances to many bean diseases.

Literature Cited