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erratum

Nodulation of legumes by members of the β-subclass of Proteobacteria

Lionel Moulin, Antonio Munive, Bernard Dreyfus & Catherine Boivin-Masson

Nature **411,** 948–950 (2001).

In Figs 1 and 3 the tree branches were very faint. The corrected figures are shown below. □

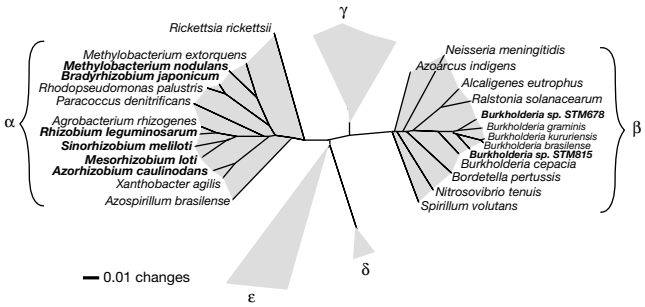

Figure 1

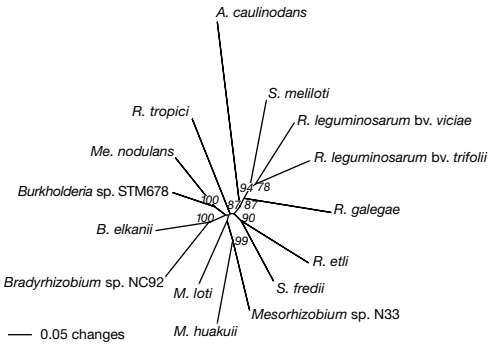

Figure 3

Enos Lake. This work was supported by NSF-NATO and NSF-International Research fellowships to J.W.B. and NSERC operating grants to D. Schluter.

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Nodulation of legumes by members of the β-subclass of Proteobacteria

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Members of the Leguminosae form the largest plant family on Earth, with around 18,000 species. The success of legumes can largely be attributed to their ability to form a nitrogen-fixing symbiosis with specific bacteria known as rhizobia, manifested by the development of nodules on the plant roots in which the bacteria fix atmospheric nitrogen, a major contributor to the global nitrogen cycle. Rhizobia described so far belong exclusively to the α-subclass of Proteobacteria, where they are distributed in four distinct phylogenetic branches[1,2]. Although nitrogen-fixing bacteria exist in other proteobacterial subclasses, for example *Herbaspirillum* and *Azoarcus* from the phylogenetically distant β-subclass, none has been found to harbour the *nod* genes essential for establishing rhizobial symbiosis[3,4]. Here we report the identification of proteobacteria from the β-subclass that nodulate legumes. This finding shows that the ability to establish a symbiosis with legumes is more widespread in bacteria than anticipated to date.

Recent taxonomic classifications portray rhizobia as belonging to three different branches of the α-subclass of Proteobacteria: the *Mesorhizobium-Sinorhizobium-Rhizobium* branch, the *Bradyrhizobium* branch and the *Azorhizobium* branch[1,5]. We recently described a fourth rhizobial branch within the α-subclass of Proteobacteria, containing methylotrophic rhizobial *Methylobacterium*[2]. To explore further the phylogenetic diversity of nitrogen-fixing legume symbionts, we characterized a collection of rhizobia isolated from tropical legumes. We found here that two bacteria isolated from nodules of *Aspalathus* and *Machaerium* plants were very distant from known rhizobia.

Strain STM678 was originally isolated from the South African legume *Aspalathus carnosa*, which was thought to be nodulated by bacteria of the *Bradyrhizobium* genus[6]. However, we performed phylogenetic analysis of gene sequences of the small subunit of ribosomal RNA (16S rRNA), and found that strain STM678 does not belong to any of the four branches of rhizobia described so far, nor even to the α-subclass of Proteobacteria, but instead belongs to the β-subclass of Proteobacteria (Fig. 1). From this analysis, we found the most closely-related sequences to that of strain STM678 (AJ302311) to be those of *Burkholderia kururiensis* (96.9% identity), *B. brasilense* (96.8% identity) and *B. graminis* (96.8% identity). Phylogenetic analyses of partial sequences of the 23S rRNA gene (AJ302313) and the *dnaK* gene encoding the chaperon heat shock protein (AJ302314) were consistent with the 16S rRNA analysis, thus unambiguously positioning strain STM678 in the *Burkholderia* genus within the β-subdivision of Proteobacteria.

To ensure that the *Burkholderia* strain STM678 was indeed a rhizobium, we checked its ability to re-nodulate a leguminous plant. Because seeds of the original host plant, *A. carnosa*, were not available, we selected as test plant *Macroptilium atropurpureum*, a tropical legume capable of establishing a symbiosis with diverse rhizobia. Over a three-week period, strain STM678 formed 5 to 20 nodules per plant on the roots of *M. atropurpureum* (Fig. 2). The nodules displayed the classical determinate nodule structure, with a central 'infected' tissue containing cells with intracellular bacteria and a peripheral tissue with vascular bundles (Fig. 2). Single colonies re-isolated from surface-sterilized nodules exhibited the characteristics of strain STM678, as assessed by 16S rDNA sequencing and *nodA* analysis using polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP; see below). Hence, Koch's postulates were verified. To eliminate the possibility that strain STM678 is a mixture of two different bacteria, a *Burkholderia* and a rhizobium, we isolated spontaneous mutants resistant to chloramphenicol, rifampicin and streptomycin, and showed by 16S rDNA and *nodA* PCR-RFLP analyses that the individual mutants retained the characteristics of both *Burkholderia* and rhizobia. Nodules induced on *M. atropurpureum* by strain STM678 were ineffective in terms of nitrogen fixation, probably because *M. atropurpureum* is not the original symbiotic partner of strain STM678. Supporting this conjecture, strain STM678 was indeed found to contain the *nifH* gene encoding dinitrogenase reductase, a key enzyme in nitrogen fixation (AJ302315). The highest identity values with other *nifH* genes were 81.2% (the α-Proteobacterium *Azorhizobium caulinodans*) and 81.1% (the β-Proteobacterium *Herbaspirillum seropedicae*).

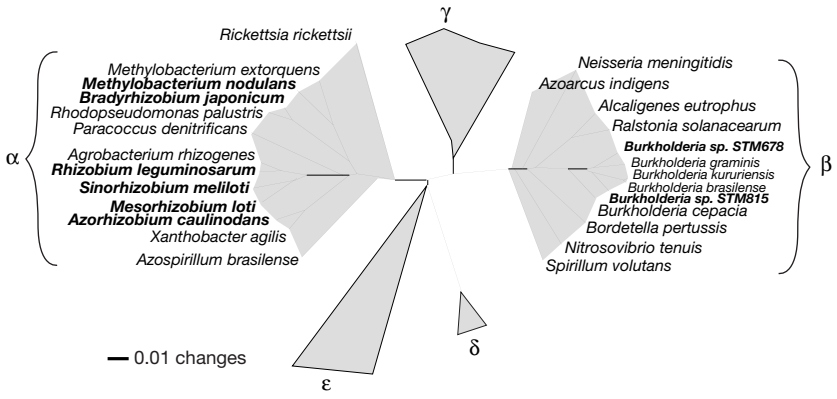

Figure 1 Unrooted 16S rDNA tree of Proteobacteria (purple bacteria). The figure shows the phylogenetic relationships between the different rhizobial genera—as represented by type species in bold—including the new rhizobial *Burkholderia* sp. strains. α, β, δ, γ and ε represent the different subdivisions of the Proteobacteria. The tree was constructed by using the neighbour-joining method and adapted from ref. 5. 16S rDNA sequences of published bacteria are available in GenBank. 16S rDNA from *Burkholderia* sp. STM 678 and *Burkholderia* sp. STM 815 are given in the text (AJ 302311 and AJ 302312).

Nodulation of legumes by rhizobia is controlled by a set of bacterial nodulation (*nod*) genes involved in the production of lipo-chitooligosaccharides (Nod factors) that act as signalling molecules for nodulating specific legume hosts[3,4]. The *nodABC* genes are responsible for the synthesis of the core structure of the Nod factor[7,8], and as such are present in all rhizobia. We thus looked for the presence of *nodABC* genes in the nodulating *Burkholderia* strain STM678 by PCR amplification (see Methods). Sequencing of the amplified DNA revealed a genetic organisation of *nodAB* genes similar to that found in other rhizobia; that is, with *nodAB* in the same orientation and overlapping and preceded by a NodD-dependent regulatory sequence (*nod* box). A *nodC*-like sequence was found immediately downstream of *nodB*. However this sequence is unlikely to correspond to a functional *nodC* gene as it lacks the ~600-base-pair 5′ end of known *nodC* genes. We obtain evidence for the presence elsewhere in the STM678 genome of a longer *nodC* sequence that probably corresponds to the functional *nodC* gene (AJ306730). Such genetic unlinkage of *nodABC* genes in rhizobia is not unprecedented[9]. The sequences of strain STM678 *nodAB* genes (AJ302321) revealed very high similarities with rhizobial Nod protein sequences available in databases, with values ranging from 62.8% (*Sinorhizobium meliloti*) to 77.6% (*Methylobacterium nodulans*) for NodA and from 55.6% (*Rhizobium galegae*) to 70.9% (*Mesorhizobium* sp. N33) for NodB. To examine whether these genes were functional, we constructed a *nodA* mutant by introducing a *lacZ*-kanamycin-resistance cassette into the *nodA* gene of strain STM678. The *nodA* mutant did not form any nodules after inoculation on *M. atropurpureum*, even after 30 days, indicating that the *nod* genes that we disrupted are required for nodulation of the *Burkholderia* sp. strain STM678.

By screening among bacteria isolated from root nodules collected from various legumes in French Guiana, we found a second *Burkholderia* rhizobium, strain STM815, isolated from the legume *Machaerium lunatum*. 16S rDNA sequencing (AJ302312) of this strain revealed the following sequence identities with its closest phylogenetic neighbours: 96.9% (*Burkholderia kururiensis*), 96.8% (*B. brasilense*), 96.6% (*B. graminis*) and 96.9% (strain STM678). These data clearly show that strain STM815 belongs to the *Burkholderia* genus, and most probably to a different species to strain STM678. The nodulation ability of STM815 was confirmed, as described for strain STM678, by inoculation of *M. atropurpureum* in axenic conditions and by re-isolation and characterization of the bacteria isolated from the induced nodules.

The β-subdivision of Proteobacteria contains many bacteria that interact with eukaryotes, including human pathogens, such as *Neisseria* and *Bordetella*, and plant-associated bacteria. These latter bacteria include pathogenic *Ralstonia solanacearum*, rhizospheric *Burkholderia* and endophytic *Azoarcus*. However the β-Proteobacteria had not been reported to include rhizobia, bacteria capable of nodulating leguminous plants. Here, we have identified two rhizobia belonging to the *Burkholderia* genus. These bacteria were isolated in different continents, from legumes belonging to different Papilionoideae tribes, and probably correspond to two distinct species. We have shown that the genetic control of nodulation by the *Burkholderia* sp. strain STM678 involves *nod* genes. Moreover, this strain has been shown to produce Nod factors[10]. Hence rhizobia from both the α- and β-Proteobacteria (now termed α- and β-rhizobia) use the same strategy for establishing symbioses with legumes.

Furthermore, we have performed phylogenetic analyses that indicate a much smaller phylogenetic distance between the *nodAB* genes of strain STM678 and other rhizobia (Fig. 3) than between the 16S rRNA genes of α- and β-Proteobacteria (Fig. 1). This suggests that the presence of *nod* genes in both α- and β-rhizobia probably occurred through horizontal gene transfer. This transfer may have occurred after the appearance of legumes on Earth, about 70 million

Figure 2 Nodules of *Macroptilium atropurpureum*, three weeks after root inoculation with *Burkholderia* sp. strain STM678. **a**, Root segments with nodules; **b**, longitudinal section showing the typical structure of a determinate nodule with a central zone containing infected cells (iz), and a peripheral region with vascular bundles (vb).

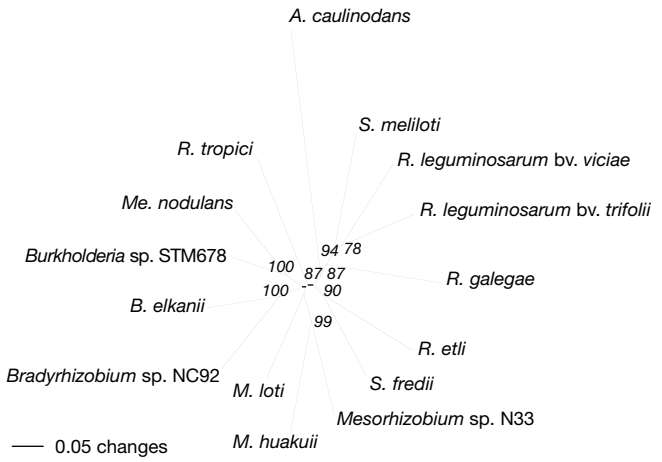

Figure 3 Unrooted NodA tree showing the close phylogenetic relationship between the NodA of strain STM678 and those of α-rhizobia. The tree is based on full-length sequences, and constructed by using the neighbour-joining method. Bootstrap values (% from 1,000 replications) are indicated. NodA sequences of published rhizobia are available in GenBank. NodA from *Burkholderia* sp. STM 678 is given in the text (AJ 302321). *A*, *Azorhizobium*, *B.*, *Bradyrhizobium*. *M*, *Mesorhizobium*. *Me*, Methylobacterium. *R*, *Rhizobium*. *S*, *Sinorhizobium*.

years ago[11]. Although rhizobia have been studied for more than 100 years, symbionts of less than 10% of the 750 legume genera have been fully characterized. Our work suggests that characterization of the symbionts of the yet unexplored legumes may reveal the rhizobial nature of additional members of the β-Proteobacteria and possibly other taxonomic classes. Such a study may contribute significantly to the understanding of the origin, and the evolution of, the legume–rhizobia symbioses, and may open new perspectives for engineering beneficial associations. □

Methods

Strains and culture conditions

Strain STM678 was provided by H. P. Spaink (Univ. Leiden). Cells were maintained and grown on yeast extract–mannitol medium[2].

DNA amplification, sequencing and analysis

Nearly full-length 16S rDNA was amplified and sequenced as previously described[2]. 16S rDNA PCR-RFLP analysis was performed as described[2] except that *Cfr*13I, *Hinf*I and *Msp*I were used. A 870-bp part of *dnaK* was amplified and sequenced using the primers 5′-GAMGTCAARCGBATCATCAA-3′ and 5′-TGTCYTTVGCBSMNACRTGCAG-3′. A 370-bp fragment of 23S rRNA gene was amplified and sequenced using the universal primers 5′-AGAGGCGATGAAGGACGT-3′ and 5′-ACCTTTCCCTCACGGTACT-3′. A 1,509-bp fragment containing partial *nifH* and *nifD* genes were amplified using the primers 5′-GCCWTCTAYGGNAARGGNGG-3′ and 5′-ATCAGGCCGATCGGGCATT-3′ and further sequenced. A 2.6-kb fragment containing the *nodAB* genes of strain STM678 was amplified using the primers 5′-CAGATCNAGDCCBTTGAARCGCA-3′ (located at the end of *nodD* in rhizobia) and 5′-CTNCGNGCCCARCGNAGTTG-3′ (located within *nodC* in rhizobia). This fragment was further sequenced using pairs of degenerate primers defined from conserved motifs of *nodA*, *nodB*, *nodC*, and *nod* box. Two 1-kb overlapping fragments containing part of the *nodC* and *nodI* genes of strain STM678 were amplified using the primer pairs 5′-TAYRTGGTYGAYGACGGWTC-3′/5′-CCATACGCACCGTGG TGCTCTTGC-3′ and 5′-GGTTATCGGACCGAGTACG/5′-TCTTCCATVAWRTGVGT NGTCA-3′ (forward primer located at the beginning of *nodC* in rhizobia, reverse primer located within *nodI* in rhizobia) and further sequenced. *nodA* PCR-RFLP analysis was performed on a 455-bp PCR product obtained with the primer pair 5′-TCACARCTC KGGCCCGTTCCG-3′/5′-TGGGCSGGNGCNAGRCCBGA-3′ and digested with *Cfr*13I, *Hinf*I and *Hae*III. Multiple alignments were performed with CLUSTALX[12]. Phylogenetic analyses used the neighbour-joining method and the programmes in PAUP version 4.0b5[13].

Construction of a *nodA* mutant

A 2.5-kb *Xho*I-*Xho*I fragment containing the *nodAB* genes of strain STM678 was obtained by PCR amplification using modified primers containing additional *Xho*I sites and cloned in the *Sal*I site of pJQ200mp18 suicide vector[14]. The 4.7-kb *Sal*I *lacZ*-kanamycin-resistance cassette of pKOK5[15] was inserted at the *Sal*I site of the *nodA* gene cloned in pJQ200mp18. The pJQ200 derivatives obtained, which encoded a counterselectable *sacB* marker, were transformed into *Escherichia coli* XLII, and further introduced by conjugation into a spontaneous chloramphenicol-resistant derivative of strain STM678. Transconjugant colonies grown on YM medium containing 50 μg ml^{-1} kanamycin and 100 μg ml^{-1} chloramphenicol were plated onto YM medium containing 7% sucrose and kanamycin. Sucrose-resistant colonies were screened by PCR to ensure replacement of the wild-type *nodA* gene by the *nodA::lacZ* mutated gene. The *Burkholderia* status of the mutated strain was assessed by 16S rDNA PCR-RFLP.

Plant tests

Seeds were surface-sterilized with concentrated sulphuric acid for 5 min. Plant cultivation and nodulation tests were carried out as described[2]. Effectiveness was estimated by visual observation of plant vigour and foliage colour of 30-day-old plants. Sections were made using a Leica VT1000S Vibratome, and examined after staining with 0.01% methylene blue.

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Spatial awareness is a function of the temporal not the posterior parietal lobe

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Our current understanding of spatial behaviour and parietal lobe function is largely based on the belief that spatial neglect in humans (a lack of awareness of space on the side of the body contralateral to a brain injury) is typically associated with lesions of the posterior parietal lobe. However, in monkeys, this disorder is observed after lesions of the superior temporal cortex[1], a puzzling discrepancy between the species. Here we show that, contrary to the widely accepted view, the superior temporal cortex is the neural substrate of spatial neglect in humans, as it is in monkeys. Unlike the monkey brain, spatial awareness in humans is a function largely confined to the right superior temporal cortex, a location topographically reminiscent of that for language on the left[2]. Hence, the decisive phylogenetic transition from monkey to human brain seems to be a restriction of a formerly bilateral function to the right side, rather than a shift from the temporal to the parietal lobe. One may speculate that this lateralization of spatial awareness parallels the emergence of an elaborate representation for language on the left side.

Spatial neglect is a characteristic failure to explore the side of space contralateral to a brain lesion. Patients with this disorder behave as if one side of the surrounding space had ceased to exist. Since the early post-mortem studies, we have believed that, in humans, lesions located predominantly in the posterior parietal lobe are critical for this disorder. Analyses of computerized tomography scans of right-hemispheric stroke patients with neglect found that superimposed lateral projections of these lesions centred on the inferior parietal lobule (IPL)[3,4] and the temporo-parieto-occipital (TPO) junction[4]. More recent studies have confirmed the validity of this conclusion although evidence for additional pathology leading

