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Cooperation and bacterial pathogenicity: an approach to social evolution

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Abstract

Kin selection could provide an explanation for social behavior in bacteria. The production of public goods such as extracellular molecules is metabolically costly for bacteria but could help them to exploit nutrients or invade a host. Some bacterial cells called social cheaters do not produce public goods; however, they take advantage of these extracellular molecules. In this review, the relationships between social behavior, cooperation, and evolution of bacterial pathogenicity are analyzed. This paper also examines the role of horizontal transfer of genes encoding for virulence factors and how the movement of mobile genetic elements would influence the pathogenicity and social relationships. Moreover, the link between ecological relationships and evolution in entomopathogenic bacteria, focusing on *Bacillus thuringiensis* is considered. Finally, the findings obtained with *B. thuringiensis* are extrapolated on *Bacillus pumilus* 15.1, an entomopathogenic strain whose pathogenicity is not understood yet.

Keywords: Cooperation; Evolution; Pathogenicity; Public goods; Sociability

Introduction

The social relationships of microorganisms are based on a wide range of extracellular actions produced by individual cells, which can affect the reproductive efficiency of other nearby cells. The production of molecules such as enzymes or chelants, which allow for the exploitation of nutrients that would not otherwise be accessible, or the formation of multicellular structures, are examples of this kind of action (Buckling and Rainey 2002; Griffin et al. 2004; West et al. 2006). Extracellular products and other resources are public goods that may be used by the individual that produces them or by all individuals of the group or population (West et al. 2006, 2007). For example, bacteria produce numerous factors that are considered to be public goods and are released into the environment, such as quorum-sensing molecules (Daniels et al. 2003; Diggle et al. 2007a; Williams et al. 2007), membrane vesicles (Schooling and Beveridge 2006), microbial repellents (Burgess et al. 2003), host manipulation factors (Brown 1999), adhesive polymers (Rainey and Rainey 2003), or siderophores (West and Buckling 2003), amongst others. This social behavior is, from a metabolic point of view, costly for the individual cells, although it is beneficial for the group. However, certain cells, known as 'cheaters' can decide not to produce these beneficial molecules for the group and instead take advantage of the rewards of social actions without paying any cost (West et al. 2006). Thus, in microorganisms, social cheaters that do not display cooperative behavior are considered mutants (West et al. 2007; Wakano et al. 2009; Foster 2010; Smith et al. 2014) and can evolve with relative speed if they are favorably selected (Velicer et al. 2000; Rainey and Rainey 2003; Foster et al. 2004; Griffin et al. 2004; Dugatkin et al. 2005; Harrison and Buckling 2005).

Evolution experiments conducted on a wide range of microorganisms have demonstrated that this kind of cooperative behavior is most prevalent when there is a high degree of genetic closeness between the cells (West et al. 2006; Mitri et al. 2011). For example, the opportunistic pathogen *Pseudomonas aeruginosa* (Schroeter, 1872) produces extracellular iron-chelating molecules, known as siderophores (West and Buckling 2003). These molecules are iron-scavenging agents that are released into the environment in response to a lack of this element (Ratledge and Dover 2000). The siderophores high affinity for iron

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enables them to capture the metal from compounds such as ferrous hydroxide and host organism proteins such as transferrin or ferritin (Neilands 1995; Drechsel and Jung 1998). In addition, the relationship between siderophore production and bacterial growth rate suggests that the production of siderophores contributes to bacterial virulence (West and Buckling 2003). Some cells have evolved as social cheaters since they produce small quantities of siderophores and yet they benefit from the action of siderophores produced by other cells (Ross-Gillespie et al. 2007; Mitri et al. 2011). Using this strategy, the cheats increase their frequency in comparison with the wild genotype (that does produce the molecule) (Mitri et al. 2011; Smith et al. 2014). However, in order for siderophore production to remain high, populations must be exposed to a strong genetic bottleneck, which ensures that the genetic relationship between the cells that produces the chelating agent and the one that is not is high. There are numerous examples of the role of kin selection in explaining the cooperation between microorganisms, such as the example of *Bacillus subtilis* (Ehrenberg, 1835), which forms biofilms in which closely related cells cooperate by becoming differentiated and sharing out functions (Foster 2010). This same kind of social behavior has been reported in various other species (Wakano et al. 2009).

This sacrificial phenomenon does not occur in multicellular organisms since the cells do not differ genetically from one another. No cell has an advantage over another, and if any cell should vary, it would be excluded from the cell line. For this reason, the deception of individual cells is restricted in the majority of multicellular organisms (Kessin 2000). However, there is another route to multicellularity that is adopted by organisms such as *Dictyostelium* or *Myxococus* (a group of social bacteria). When nutrients become limited and cell density is high, *Myxococcus xanthus* (Beebe, 1941) activates various genes that break up the aggregation of local groups that exchange intercellular signals, allowing the formation of spore-producing fruiting bodies (Kadam and Velicer 2006). These fruiting bodies contain approximately 50,000 cells, but only a fraction of the cells have differentiated latent myxospores (Curtis et al. 2007) which apparently sacrifice themselves for the common good (Fiegna and Velicer 2006; Fiegna et al. 2006; Kadam and Velicer 2006). *M. xanthus* cells also secrete toxins that can kill other cells within the fruiting body, or the species that feed on them; therefore, it is considered an example of group predation (Foster 2010). In addition, there are mutant social cheaters that make up part of the local group and contribute less to the production of the non-spore-producing parts of the fruiting body (Fiegna et al. 2006; West et al. 2007).

Cooperation can be found at all levels of biological organization: genes cooperate in genomes, organelles cooperate to form eukaryotic cells, cells cooperate to form multicellular organisms, pathogenic bacteria cooperate to overcome the defenses of their hosts, animals feed cooperatively, and humans and some species of insect cooperate to construct societies (West et al. 2006, 2007). With regard to the cooperation of pathogenic bacteria, reproduction within the host can be a cooperative activity that involves the secretion of virulent factors whose benefits can be shared by all of the cells, whether they secrete them or not (Smith 2001). In this way, cooperation and success in overcoming the host's defenses will depend on the nature of the virulence factor (public good) as well as the proportion of social cheaters present in a population. For example, this could explain why the enterotoxigenic *Escherichia coli* (Migula, 1895) (producer of LT and ST toxins) requires a greater population density to cause an infection when compared with the enterohemorrhagic *E. coli*, a producer of Shiga toxin (STX) (Smith and Halls 1968; O'brien et al. 1984), which requires a smaller number of cells. Enterohemorrhagic *E. coli* populations would therefore include a lower proportion of cheater cells.

Although there is no doubt about cooperative behavior, there is controversy on how this kind of behavior might be interpreted, via kin selection or via group selection (West et al. 2007; Wilson 2008). As mentioned above, there are various pieces of evidence that support the idea that this kind of behavior in bacteria could be explained by the kin selection theory. For example, in bacterial species that form biofilms, cells that are genetically related are found close to one another (Griffin et al. 2004; Diggle et al. 2007a,b). Nevertheless, the fact that all of the cells originate from a single mother cell in the majority of biofilms has been presented as an argument in favor of group selection (West et al. 2006; Boyle et al. 2013; Drescher et al. 2013).

Biofilms are communities of microbial cells, commonly composed of various lineages or species, which grow on surfaces surrounded by polymers (Kolenbrander 2000; Hall-Stoodley et al. 2004). In this kind of community, the cellular groups have limited space and influence each other depending on the distance between them. These spatial relationships are of the utmost importance for the compression of cellular community cooperation evolution (Durrett and Levin 1994; Drescher et al. 2013). When different cellular lineages are separated in space, cooperative phenotypes are more likely to benefit other cells belonging to their phenotype (Mitteldorf and Wilson 2000; Griffin and West 2002; Axelrod et al. 2004). However, when the different cellular lineages are intermingled, cells that exploit the resources of others are able to prosper more efficiently (Griffin et al. 2004; Diggle et al. 2007a,b; Sandoz et al. 2007; Chuang et al. 2009). If the cells of a genotype are mixed with other genotypes, it is more probable that competitive characteristics that cause potential harm to

neighboring cells will evolve than when the cells are surrounded by clones (Mitri et al. 2011). Therefore, it has been suggested that the spatial structure of biofilms is a critical factor in microbial interactions (Boyle et al. 2013). Intra-species cooperation could be favored by spatial segregation that keeps secreting cells away from those that do not secrete, while inter-species cooperation is favored by the presence of various species (Sandoz et al. 2007; Nadell et al. 2009). Cooperative phenotypes are subject to exploitation by non-cooperative cell lineages since they could be influencing the interaction between cooperative and non-cooperative cells (Nadell et al. 2009, 2010). It is difficult for cooperative cells to be successful in competition against non-cooperative cells, which exploit public goods without paying the costs. However, if the cooperative cells are spatially segregated and prefer to interact amongst themselves, they can prevail. The spatial distribution of genetic lineages is related to physical and biological parameters, such as the availability and capacity for nutrient diffusion, cellular metabolic efficiency, cell growth rate, or biomass density. Drastic changes in these parameters can change the spatial layout of microbial populations within a community and determine if cells with cooperative phenotypes could compete locally or globally with social cheaters. The result would be the segregation of cellular lines that favor the evolution of cooperative phenotypes (Nadell et al. 2009).

There is also abundant evidence that demonstrates the fact that the presence of different genotypes in biofilms limits cooperation across a wide range of bacterial characteristics (Greig and Travisano 2004; Gore et al. 2009), such as the secretion of enzymes (Griffin et al. 2004); iron capture (Diggle et al. 2007a), the secretion of quorum-sensing molecules and the formation of fruiting bodies (Foster et al. 2002; Buttery et al. 2009). Accordingly, it has been suggested that interactions between species can have a profound influence on intra-species cooperative behavior in spatially structured microbial groups like biofilms. It has been demonstrated that ecological competition with other species preferentially prejudices secreting cells more than non-secreting cells or social cheaters. This may be due to the fact that investing in secreting extracellular molecules can delay the growth of cellular lineages at critical stages. This initial investment leaves secreting cells vulnerable to competition with other lineages, particularly in nutrient-poor conditions where resources are limiting and the majority of lineages are eliminated through a strong genetic bottleneck. The potential for bottlenecks in growing microbial groups has been well documented (Gage 2002; Hallatschek et al. 2007; Boyle et al. 2013). Bottlenecks have been interpreted as favorable for the evolution of cooperation because they promote genetic identity in emerging clonal groups (Brockhurst 2007; Nadell et al. 2010). However, Mitri et al. (2011) suggest that bottlenecks can also be indicative of the fact that strong ecological competition can eliminate cells with cooperative behavior before they have had the chance to establish themselves. For example, it has been demonstrated that *P. aeruginosa* lineages that secrete siderophores that capture iron are vulnerable to competition from lineages that do not secrete (but that use the siderophores without producing them) when *Staphylococcus aureus* (Rosenbach, 1884) is added (Harrison et al. 2008). Population bottlenecks fulfill a fundamental role in the maintenance of social characteristics in microorganisms. Some ecological parameters such as colonization or disturbance may favor cooperation, causing population bottlenecks that increase genetic kinship. Moreover, the size of the population bottleneck fulfills a fundamental role in the success of cooperation. In this way, kinship increases as the size of the bottleneck decreases, favoring the evolution of cooperation. Brockhurst (2007) used *Pseudomomas fluorescens* (Migula, 1985) SBW25 to experimentally prove that the quantity of social cheaters increases as the size of the bottleneck increases, which suggests that the reduction in kinship caused by large genetic bottlenecks works against cooperation.

Other experiments have underlined the importance of ecological competition in favoring cooperation between species. The model developed by Rankin et al. (2007) demonstrated that intra-species competition can increase or decrease the ability to compete with other species. It has also been proposed that interactions with other species can promote the evolution of secreting genotypes (Mitri et al. 2011; Drescher et al. 2013). Social isolation allows the secreting cells to form patches in which they preferentially help their own genotype; therefore, the spatial structure of a biofilm may promote the evolution of cooperation (Nowak and May 1992; Xavier et al. 2011). The importance of the effects of social isolation in natural communities has not yet been clarified. However, it has been suggested that it could be more significant under conditions where nutrients are abundant in which various species can coexist. An interesting case study is that of the human microbiome, particularly that of the intestine, where the cells can form dense biofilms containing various species (Macfarlane and Dillon 2007).

As mentioned above, the secretion of public goods in biofilms is affected by the availability of nutrients. When competition for nutrients is strong, the addition of new species can inhibit cooperation by eradicating secreting lineages before they can establish themselves. When nutrients are abundant and various species are found in the same habitat, the secreting lineages of any species are surrounded by other species. This 'social isolation' protects those cells that produce public goods from competition by those from the same species that do not produce public goods, and this can improve cooperation

between species (Hol et al. 2013). In addition, limitations in the interactions between species have been observed since it is difficult to find conditions that encourage cooperation between cells of the same species and of other species (Mitri et al. 2011). On the other hand, it has been suggested that species with different metabolic needs show a greater tendency toward mutualistic behavior (Little et al. 2008). In general, cooperation in microbial communities will be favored when there is a high level of competition between communities (Mitri et al. 2011).

In conclusion, the appearance of biofilm organization could have arisen without active coordination. This implies that certain properties like phenotypic differentiation, species stratification, and the formation of channels do not necessarily require cells to communicate amongst themselves using specialized signal molecules. Also, although local cooperation between bacteria occurs frequently, the evolution of cooperation between cells belonging to a biofilm may be unlikely. Strong conflicts may arise between species and lineages in a biofilm, and spontaneous mutations may cause conflicts even if the communities were initiated by cells that were genetically identical (Nadell et al. 2009; Hol et al. 2013).

On the other hand, the appearance of cooperation via kin selection has also been described in more complex organisms such as vertebrates (Griffin et al. 2004). Without a doubt, the best way to clarify what kind of selection is affecting the evolution of cooperative behavior is by means of experimentation. The evidence provided by experimental studies of evolution (Griffin et al. 2004; Kummerli et al. 2009) has been a fundamental pillar in the understanding of microorganism sociability.

Review

Horizontal gene transfer and the evolution of bacterial pathogenicity

Bacteria transfer their genes vertically, from a mother cell to a daughter cell, similarly to what happens in more complex organisms, although they can also interchange their genes horizontally. Genes move quickly between genomes via mobile genetic elements such as plasmids, transposons, bacteriophages, and self-splicing molecular parasites (Nogueira et al. 2009; Siefert 2009). A vertically transmitted genome encodes for fundamental cellular processes, while a horizontally transmitted genome encodes for genes that allow for the exploitation of specialized niches or genes that provide resistance to toxic molecules (Hacker and Carniel 2001). Many of the genes responsible for pathogenic bacterial virulence are found in mobile genetic elements that can be transmitted horizontally between different bacterial lineages. The horizontal transfer of virulence factor genes has played a fundamental role in the evolution of pathogenic bacteria. However, it is poorly understood why these genes are so mobile (Griffin et al. 2004; West et al. 2006; Nogueira et al. 2009).

While plasmids benefit from the horizontal gene transfer as selfish DNA, this mobility implies a range of costs on host bacteria, especially in terms of the resources invested in conjugation (Nogueira et al. 2009). On the other hand, bacteriophages are major contributors to the process of horizontal gene transfer given their environmental ubiquity, their great abundance in nature, and the functional effects that they cause in hosts (Hanage et al. 2005). In this way, phages make a significant contribution to the diversity and evolution of bacteria since, in addition to acting as vectors for gene transfer, they promote a high coevolution rate and a high level of specialization between bacterial genotypes in natural microbial communities (Siefert 2009). Lysogenic phages integrate their genome within the host genome until the prophage is excised from the host genome and cell lysis results. During this process, various segments of the host DNA can be introduced into the viral genome, and when lysis of the cell occurs, the host DNA is dispersed in the phages (Siefert 2009). In this way, during lysogenic conversion, prophages can provide benefits to their hosts while they remain latent through the addition of new functions in the bacterial genome. For example, it is through this process that the harmless *Vibrio cholerae* (Pacini, 1854) lineage is transformed into the highly virulent lineage that causes cholera (Hanage et al. 2006). Additionally, the insertion sequences (which can be autonomous or part of composite transposons) are efficient at moving themselves between bacterial genomes as they reshuffle and shape them (Chandler and Mahillon 2002; Siguier et al. 2006). It has been demonstrated that the massive expansion of insertion sequences has a positive correlation with the appearance of some pathogenic bacterial species (Siefert 2009).

Numerous theories could explain why bacteria invest in gene mobility but none has been experimentally tested, which means that the evolutionary bases of the fundamental genome organization of bacteria remain unclear. The horizontal gene transfer is particularly important in the evolution of infectious diseases, in the spread of virulence genes, and in antibiotic resistance (Holden et al. 2004; Frost et al. 2005; Gogarten and Townsend 2005; Murphy and Boyd 2008). Key virulence genes, essential for infecting host organisms, are carried on mobile genetic elements in various clinically significant species such as *S. aureus*, *Bacillus cereus* (Frankland and Frankland, 1887), *E. coli*, and the agents that cause anthrax, cholera, and diphtheria (Smith 2001).

Virulence, secretion, and horizontal mobility are closely related in bacterial genomes. Virulence factors are commonly secreted with the aim of reaching specialized tissue within the host (Smith 2001). There is evidence that reveals

that the *E. coli* genes responsible for secreted products tend to be more mobile than genes coding for intracellular products (Nogueira et al. 2009). Secreted products are used as public goods by neighboring bacteria, independently of whether the neighbor secrets products for itself. In cooperative secretion, the individual bacteria that invest in secretion pay a cost in terms of growth, although the group benefits from the presence of virulence factors, for instance allowing an infection to become established in a host organism (West et al. 2006; Raymond et al. 2010).

Virulence factor genes would have evolved to increase the pathogenicity of bacteria, causing positive effects such as an increase in their transmission rate, which would help them to colonize new hosts, obtain new resources, evade the host defenses or disperse themselves to colonize new individuals (Ochman et al. 2000; Nogueira et al. 2009). However, extracellular virulence factors are potentially available to members of the pathogen population that do not produce the effector molecule. Thus, a social cheater cell could avoid the metabolic cost of producing the virulence factor, but it would increase its frequency during an infection due to the action of molecules produced by other cells in the population (Ross-Gillespie et al. 2007, 2009).

If pathogens compete for resources within a host, the increase in cheats will reduce the transmission of the genotype that produces the virulence factor (Buckling and Rainey 2002; Mitri et al. 2011; Smith et al. 2014). Nevertheless, it would be possible to reestablish the infectiveness by reducing the density of the cheater cells. The pathogenicity could also be restored by reintroducing functional versions of the genes coding for virulence factors in the cheats. As a result, it has been proposed that virulence factors are maintained in horizontally transmissible mobile genetic elements so that they can be reintroduced and prevent a high frequency of cheats (Smith 2001; Wakano et al. 2009). Accordingly, the existence of the tragedy of the commons in antibiotic-resistant plasmids was recently stated (Smith 2011). Mobile genetic elements would be superinfecting bacteria that had already been previously infected, increasing the fitness of the plasmid. However, they would become victims of their own success since they reduce the density of their bacterial hosts (Smith 2011).

The sociability of entomopathogenic bacteria and its ecology

Relatively few species of bacteria have been described as insect pathogens, but they have received a lot of attention as a result of their potential for pest control in agriculture and for the control of vector-transmitted diseases (van Emden and Service 2004). Amongst these, most interest has been concentrated on *Bacillus thuringiensis* (Berliner, 1915), due to its specificity and its applicability as a biological control agent. *B. thuringiensis* is a strict aerobic, Gram-positive, flagellate, ubiquitous, sporulating bacteria that is morphologically and genetically related to *B. cereus* and *B. anthracis* (Cohn, 1872) and is widely used as a biological insecticide for insect control (Schnepf et al. 1998; Crickmore et al. 2013). *B. thuringiensis* produces protein crystals with insecticidal properties (Helgason et al. 2000). The proteins of which they are composed are called δ-endotoxins and there are two main types: Cry toxins and Cyt toxins. δ-endotoxins are pore-forming proteins specifically active toward membranes from insects (Lepidoptera, Coleoptera, and Diptera), acari, nematodes, flat worms, and protozoa (Schnepf et al. 1998; Lightwood et al. 2000; Li et al. 2001).

The mechanism of action of Cry toxins is a complex process that develops in various stages; however, the symptoms caused in the insect subjected to ingestion of the toxins can be summed up in the following steps: (1) ingestion ceases, (2) gut paralysis, (3) excretion of residues (vomiting and diarrhea), (4) total paralysis, and (5) death. A larva intoxicated with *B. thuringiensis* shows a characteristic black color due to tissue necrosis (Bravo et al. 1992).

Cry insecticide toxins are the main virulence factors of *B. thuringiensis* and can be considered a public good. The sociability of these effector molecules has been studied under almost natural conditions using the larva of the diamondback moth, *Plutella xyllostella* (Linnaeus, 1758), as a host (Raymond et al. 2012). As is the case with other bacteria that possess social behavior, certain *B. thuringiensis* genotypes do not produce Cry toxins. However, they are able to benefit from the production of the insecticidal proteins synthesized by other individuals in the population when infecting the host. Thus, non-toxin-producing cells can take advantage of the formation of pores in the epithelial cells of the insect gut to infect it. This would demonstrate that the reproductive efficiency of microorganisms can depend on the cooperation between cells, both in controlled conditions and in nature (Griffin et al. 2004; Raymond et al. 2012). Interestingly, the high densities reached by social cheaters could explain why *B. thuringiensis* does not cause epidemics on nature (Raymond et al. 2012). This connection between cooperative virulence and epidemiology could also be relevant for species of bacteria that do not produce toxins, including some human pathogens.

The entomopathogenicity of *Bacillus pumilus* (Meyer and Gottheil, 1901) could also be explained by the social evolution of virulence factors. *B. pumilus* is a ubiquitous bacteria with a wide range of significant activities from a biotechnological point of view. Some strains of *B. pumilus* have fungicidal properties and have been used as biological control agents against phytopathogenic fungi (Bottone and Peluso 2000; Lehman et al. 2001;

De-Bashan et al. 2010), while others have shown powerful antibacterial activity (Aunpad and Na-Bangchang 2007). In addition, *B. pumilus* has been reported as entomopathogenic bacteria. The first study that demonstrated the entomopathogenicity of *B. pumilus* is detailed in a patent that describes a strain active against the corn rootworm (e.g., *Diabrotica undecimpunctata* (Mannerheim, 1843), *Diabotrica longicornis* (Say, 1824)), the armyworm (*Spodoptera exigua* (Hubner, 1808)), and some species of nematodes (Heins et al. 1999). The entomopathogenic activity of *B. pumilus* was confirmed in a study that described the isolation of the strain 15.1, a highly toxic strain against Mediterranean fruit fly larvae, *Ceratitis capitata* (Wiedemann, 1824) (Molina et al. 2010). Since *B. pumilus* has never been considered as a classic entomopathogenic bacteria, the origin and the possible evolutionary route of the insecticidal activity of the *B. pumilus* 15.1 strain is a fairly interesting topic that would help to explain the process of pathogenicity. The specific case of *B. thuringiensis*, whose ecology is widely known, could represent the best analogous example for the discussion of this topic. Therefore, we will concentrate on *B. thuringiensis* strains active against leaf-eating insects (since it is the best case study), with the aim of explaining the possible origin of *B. pumilus* 15.1 pathogenicity.

The role of *B. thuringiensis* in the environment remains unclear due to, among other reasons, a lack of manipulative field experiments (Travers et al. 1987; Raymond et al. 2010). One of the many hypotheses that attempt to explain the role of *B. thuringiensis* suggests that the bacteria has evolved in order to provide symbiotic protection to plants since it forms part of the normal phylloplane microbiota (Smith and Couche 1991; Elliot et al. 2000). Another hypothesis proposes that *B. thuringiensis* is a natural soil inhabitant with incidental insecticidal activity (Martin and Travers 1989). This hypothesis states that except from the fact that *B. thuringiensis* has previously been found in association with insects, there is no reason to believe that this association is vital. The ubiquity of *B. thuringiensis* in soil is consistent with this idea and further supports the hypothesis of Dulmage and Aizawa (1982), which proposes that the soil is the normal environment of *B. thuringiensis*. However, this bacteria is not usually toxic against insect larvae that live in the soil, but it is against insects with aerial or water-borne larvae.

On the other hand, Jensen et al. (2003) suggested that *B. thuringiensis* could be part of the commensal gut microbiota of various insect species without causing obvious disease or death. However, Raymond et al. (2010) demonstrated that *B. thuringiensis* behaves as a specialized insect pathogen in the field. This study suggests that the normal habitat of *B. thuringiensis* is the soil, and that movement from the soil (which acts as a reservoir) toward the aerial parts of the plant, where susceptible hosts are present, is a key feature of its ecology.

Similarly to *B. thuringiensis, B. pumilus* is an ubiquitous bacteria whose typical habitat is the soil. The *B. pumilus* 15.1 strain was isolated from a partially decomposed reed plant (Molina et al. 2010), suggesting that plants can also form part of the natural habitat of this bacterium. Besides, as it has been mentioned above, other *B. pumilus* strains have been reported playing many other roles: first, as a growth-promoting rhizobacteria; second, providing protection to the plants as a result of its antifungal properties against phytopathogenic fungi; and finally playing a role in the plant defense reactions (Benhamou et al. 1996).

It is a fact that *B. pumilus* interacts with plants and there is evidence to suggest that this ecological relationship is beneficial to both organisms. Smith and Couche (1991) proposed an explanation for this type of interaction between a plant and an entomopathogenic bacteria. This study proposed that *B. thuringiensis* populations found in the phylloplanes of plants could inhibit the feeding of insect larvae. This situation could be a symbiotic relationship in which *B. thuringiensis* also benefits from being a phylloplane epiphyte. In such a way, the plant could provide nutrients from leaf exudates and associated microflora and also provide a niche free from competition with other soil-borne spore-forming bacteria. On the phylloplane, *B. thuringiensis* is accessible to leaf-feeding insect larvae. If the larvae ingest sufficient toxin spore and crystals to inhibit their feeding, defoliation would be reduced, and as a result, the plant would be protected. This proposed hypothesis for plant-entomopathogenic bacteria association could also explain the relationship between *B. pumilus* and plants. Furthermore, this hypothesis does not exclude other concepts about the ecological role of *B. pumilus*, for instance, that it could act as an antifungal or an antibacterial agent in the soil microcosm (Elliot et al. 2000).

Elliot et al. (2000) extended the plant hypothesis (originally described as predators and parasitoids) to entomopathogens and essentially agrees with Smith and Couche (1991). In order for a plant to employ an entomopathogen as a bodyguard, this relationship must represent a good return on investment (the benefits of the relationship must outweigh the costs) (Price et al. 1980, Elliot et al. 2000). The 'bodyguard' hypothesis in which a bacteria acts mutualistically to a plant could explain why *B. thuringiensis* is maintained in the phylloplane and on the ground. The plant benefits from the entomopathogenic activity of the bacteria before its population reaches high densities (Elliot et al. 2000; Buckling and Rainey 2002).

Based on the aforementioned case of *B. thuringiensis*, on the fact that *B. pumilus* is a ubiquitous bacteria with the ground as its typical habitat, and on its ecological relationship with plants, it is suggested that the bodyguard

hypothesis could also explain the entomopathogenicity of *B. pumilus* 15.1. *C. capitata* larvae are not leaf-feeding, but they are considered as phytophages. For this reason, and because of the relationship that *B. pumilus* maintains with plants, the application of the bodyguard theory could be feasible. This hypothesis may represent the best explanation for *B. pumilus* insecticidal activity. On the other hand, in spite of the fact that *B. pumilus* is a different strain, it has been reported as a pathogen for *S. exigua* (Heins et al. 1999), a species with defoliating larvae.

In addition, the presence of crystals has been described in sporulating cultures of *B. pumilus* 15.1 that are similar to the Cry proteins of *B. thuringiensis* (Molina et al. 2009). The role of these structures has not yet been elucidated. However, it could be suggested that they are involved in the entomopathogenicity of *B. pumilus* 15.1 due to their structural similarity with the insecticidal toxins of *B. thuringiensis* (Molina et al. 2009). The molecules synthesized and excreted by *B. pumilus* 15.1 would be considered as the bacteria public goods; therefore, it is possible that they would have the same social behavior as that which has been widely reported. If so, the presence of social cheater cells could also explain why *B. pumilus* 15.1 does not cause epidemics in nature, from where it was isolated.

Conclusions

The production of public goods such as protein crystals that are toxic to insects by entomopathogenic bacteria could constitute an excellent model for the study of the relationship between the sociability of microorganisms and the evolution of their pathogenicity. In addition, the model would be robust due to the ability of sporulating microorganisms to resist adverse conditions and to continue producing public goods for the next generation. Similarly, it would be expected that due to the selective pressure applied by insects on bacteria with insecticidal activity, their virulence would increase through the production of a greater quantity of protein crystals after each pass through the insect. For all the forgoing reasons, understanding the evolution of pathogenicity and its relationship to sociability should focus on the study of bacteria that are pathogenic for insects.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

CAM reviewed the literature and wrote the manuscript. SV helped to draft the manuscript and provided comments on it. Both authors read and approved the final manuscript.

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