are the producers of antibiotics because they should have protection mechanisms against the antibiotics they synthetize? It is indeed a popular view that the main contributors of antibiotic resistance genes for dissemination are the producers of antibiotics, which protect themselves this way from the lethal action of antibiotics produced (Wright 2012). To respond this question, we performed several phylogenetic analyses with antibiotic resistance genes including those from antibiotic producers. Our phylogenetic analyses of genes conferring tetracycline resistance via the ribosomal protection mechanism, for example, consistently suggested their ancient evolutionary history (Aminov et al. 2001; Aminov and Mackie 2007). They belong to the genes encoding translation elongation factors (EF-Tu, EF-Ts, EF-G, and EF-P) but diverged from the group very early in evolution forming a monophyletic branch. The next bifurcation, which was also a fairly early event, led to the separation of genes residing in antibiotic producers such as *Streptomyces lividans* and *Streptomyces rimosus* and in the rest of nonproducing microbiota including environmental, commensal, and pathogenic. According to these analyses, there are no indications of recent horizontal transfer of the *tet* genes from antibiotic producers to other microbiota following the widespread use of tetracyclines in humans and agriculture.

Some earlier DNA–DNA hybridization-based studies, however, have implied that there is a certain level of homology between the *tet* genes in mycobacteria and tetracycline-producing strains of *Streptomyces* (Pang et al. 1994; Roberts 1996). What could be detected in tetracycline-resistant mycobacteria, however, is the *tet*(M) gene (Rossi-Fedele et al. 2006), which is not present in the streptomycetes. Besides, when a large collection of environmental and clinical mycobacterial strains was checked by PCR for the presence of *otr*(A) (which is present in *S. rimosus*), no positive signal was detectable (Kyselková et al. 2012). According to this and some earlier reports (Aínsa et al. 1998; De Rossi et al. 1998), tetracycline resistance in mycobacteria is predominantly encoded by the efflux pump genes, *tet*(V) and *tap*. Thus, there is no indication of acquisition of tetracycline resistance genes from antibiotic producers by pathogens due to the recent extensive tetracycline use. What is shared between *M. tuberculosis* and *Streptomyces*, though, is a putative transcriptional activator, *whiB7*, which confers multidrug resistance, including resistance to tetracycline (Morris et al. 2005). The gene, however, is not related phylogenetically to the *tet* gene family discussed here, and it was presumably acquired by the mycobacteria long time ago, when their ancestors shared the soil ecological niche with the streptomycetes.

Our other phylogenetic analyses of genes and gene clusters conferring resistance against tetracyclines, macrolides, glycopeptides, and quinolones (Aminov et al. 2001; Aminov et al. 2002; Aminov and Mackie 2007; Koike et al. 2010) essentially supported the view that there is no indication of antibiotic resistance genes from antibiotic producers being mobilized into other