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## Genetic and evolutionary aspects of methanogenesis

Jhp Hackstein

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**Extended abstract****Genetic and evolutionary aspects of methanogenesis\*****JHP Hackstein**

*Department of Microbiology and Evolutionary Biology, Faculty of Science, Catholic University of Nijmegen, Toernooiveld 1, NL-6525 ED Nijmegen, The Netherlands.*

It has been assumed that the feeding habits of vertebrates predispose the variety of intestinal differentiations and the composition of the microbial biota living in their intestinal tracts [1-8]. Consequently, the presence of methanogenic bacteria in the various differentiations of the large intestine and the foregut of herbivorous vertebrates, and the hindgut of xylophagous insects had been attributed to the existence of anaerobic habitats and the availability of methanogenic substrates such as carbon dioxide and hydrogen that are generated during the fermentative microbial digestion of plant-based diets [4,9,10].

More than 160 species of arthropods and 253 species of vertebrates were screened systematically for methane emissions to test the hypothesis that both vegetarian feeding habits and the presence of intestinal, "fermentative" differentiations of the host are the necessary prerequisites for the presence of symbiotic methanogens [11-15]. The results of these screens, however, failed to reveal a consistent positive correlation between methane production and vegetarian feeding habits: there are herbivorous animals

that lack intestinal methanogens and carnivorous species that host methanogens. For example, the vegetarian giant panda and all of its (predominantly vegetarian) relatives analyzed do not produce methane whereas carnivorous crocodiles and giant snakes do [13]. Unexpectedly, also the various ant- or termite-eating animals such as echidna, aardvark, giant ant-eater, tamandua, and armadillo produce methane; only pangolins (*Manis tricuspis*) meet the expectation since they fail to produce methane using a high-protein diet. Most of these non-vegetarian, but methanogenic animals possess relatively simple intestinal tracts: they lack elaborate intestinal differentiations that are believed to be essential for the presence of fermenting microbiota [5-7].

On the other hand, ostriches possess a rather complex digestive system [16]. The analysis of faeces from the various species of ostriches, however, reveals that only the African and South American species emit methane. The ostriches living in Australia or New Zealand do not; they release large amounts of hydrogen [13]. Since the absence of significant methane

emissions cannot be matched with different feeding preferences or with substantial differences in the anatomy of the digestive system, the reasons for emitting hydrogen instead of methane remain elusive. The intensity of the hydrogen emissions by faeces of non-methanogenic hosts argues against the presence of a significant alternative intestinal hydrogen sink. Therefore, it seems unlikely that methanogens were out-competed by other hydrogen-consuming microbiota such as sulfate-reducers or acetogens [c.f. 17]. A certain background of methane release from faeces also excludes the possibility of a failure of a post-partum infection with methanogens [12-14]. However, the phylogeny of ostriches provides the clue for the understanding of this phenomenon. A phylogenetic analysis of the mitochondrial rDNA genes confirmed the assumption that the non-methanogenic species emu, cassowary, and kiwi (living in Australia and/or New Zealand) share a common ancestry and the absence of methane emissions [18]. On the other hand, their African and South American relatives ostrich and nandu share a common ancestry and intestinal methanogens. Since African ostriches and nandus occupy a basal position in the phylogenetic tree, it is likely that the last common ancestor of kiwi, emu, and cassowary lost the property to host methanogens secondarily: its descendants do not emit methane notwithstanding unchanged feeding strategies and their highly differentiated digestive tracts. Only the kiwi changed its feeding behaviour - most likely while becoming smaller and occupying a new ecological niche [19,20].

Methanogenesis in mammals follows the same rules: if the methane status is included into phylogenetic trees, it becomes evident that also among mammals the

phylogenetic position of the host is more important for intestinal methanogenesis than the feeding behaviour or the presence of elaborated fermenting devices of the digestive tract [13]. However, ruminants (and other fermenting foregut differentiations) as well as caeca evolved exclusively in methanogenic taxa. Non-methanogenic hosts possessing such structures obviously lost their intestinal methanogens secondarily during their evolution from methanogenic ancestors [15]. Thus, instead of the anticipated dependence on plant derived diets and intestinal differentiation, our studies disclosed stringent taxonomic constraints of the association between methanogens and their hosts. Moreover, the analysis of the hosts's phylogeny showed that the character "methane production" obeys Dollo's law without any exception, i.e. methanogenesis does not reappear in those branches of the phylogenetic tree that are characterized by a previous loss of this trait.

Therefore, it is likely that a hereditary predisposition is essential for intestinal methanogenesis. Studies among primates and humans argue for a genetic control of the symbiosis between methanogens and their hosts. "Mutant" hosts that lack significant numbers of intestinal methanogens (and above-background levels of methane in their breath) constitute a certain fraction of the various local populations [21-24]. Pedigree analysis of a number of European families showed that the trait "methanogenesis" segregates as an autosomal dominant Mendelian factor [12,14]. These observations strongly suggest that the ability to host methanogens primarily depends on a heritable character of the

hosts, and not only on the availability of suitable intestinal redox-potentials and pH values [c.f. 25,26]. Moreover, at least in insect hindguts, methanogens occur at sites that are clearly not anoxic [27]. Intestinal methanogens are absent in young children and new-born milk-fed mammals: they appear in the course of the weaning period that last more than 30 days in most of the methanogenic species [14,27]. During the weaning period, a cross-talk between intestinal microbes and their host triggers modifications of the intestinal tract that facilitate the colonization of the gut by complex microbiota [29,30]. In methanogenic hosts this process seems to provide the basis for the persistence of methanogens in the intestinal tract [31].

\*Dedicated to the memory of Rudolf A. Prins

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