

# Preface

Microbes colonize nearly every biotic and abiotic niche on our planet. This includes also our human body which is densely populated with microbes, the majority of which interacts with us in a commensal, sometimes even mutualistic, relationship. Only a minority of our microbiota represents pathogenic organisms with the ability to cause infection.

Traditionally, the microbiota colonizing a niche or the entire body is usually distinguished into non-pathogenic and pathogenic organisms based on their potential to cause disease. Based on Jakob Henle's assumption that microorganisms are the causative agents of infectious diseases (Henle 1840), his student Robert Koch and Friedrich Loeffler formulated already in 1884 four criteria, to establish a causal relationship between a causative microbe and a disease (Koch 1884; Loeffler 1884). According to the so-called Henle–Koch postulates, bacteria isolated from disease cases have been compared with isolates from healthy individuals with regard to the presence of virulence-associated traits that contribute to the establishment of an infection in healthy hosts and that distinguish pathogenic from non-pathogenic variants.

The term commensalism has been introduced in the second half of the nineteenth century by Pierre-Joseph van Beneden, defining a relation between individuals of two species where one species obtains benefits from the other without harming or benefiting the latter. The commensal, which benefits from the association, may obtain nutrients, shelter, or locomotion from the host species, which is substantially unaffected (Boucher 1985).

Whereas the Henle–Koch postulates and the definition of commensalism allow(ed) to categorize obligate pathogens, this is often difficult for opportunistic pathogens which may belong to the normal microbiota of healthy individuals. Successful infection by such facultative pathogens does often not only rely on virulence-associated bacterial traits, but also requires susceptible hosts with an impaired mucosal barrier and/or immune response defects. Identifying individual host susceptibility factors as well as the characterization of compositional changes

of the microbiota, their gene expression and metabolic profiles in patient subsets will thus be essential for an improved treatment of infectious diseases in the future.

The vast majority of microbes colonizing a healthy host, can probably be considered commensals, although for most of them we still lack information regarding the nature of their relationship with the host (whether they hurt or help...). Estimated 500–1000 bacterial species belong to the resident human intestinal flora and the same holds true for the microbial consortium colonizing the skin (Grice et al. 2009; Sears 2005). The microorganisms living inside or on “us” outnumber “our” somatic and germ cells by a factor of ten (Turnbaugh et al. 2007), but so far only a minor fraction of this microbiota has been characterized and identified (Marcy et al. 2007). The human microbiome project will help us to further increase our knowledge on the microbial diversity associated with our healthy body and the contribution of microbiota to disease and infection. Although we already know that, e.g., the individual composition of the microbial flora differs from person to person, and that different diseases are associated with deviations in the composition of the microbial community in the diseased niche, we are far from understanding the commensals’ exact role for human development, physiology, protection against opportunistic pathogens, immunity, and nutrition.

This book covers various aspects of the interplay between commensal or pathogenic bacteria with their hosts. The chapters summarize the recent knowledge on geno- and phenotypic traits of opportunistic bacterial pathogens, such as *Escherichia coli*, staphylococci or *Pseudomonas aeruginosa*, as well as the impact of commensal and probiotic bacteria on intestinal physiology and health. The differential interaction of pathogenic, commensal, or probiotic bacteria with their host is reviewed from the bacterial and from the host perspective to complete this compilation of articles on differences and similarities of pathogenic and commensal microorganisms.

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## References

- Boucher DH (1985) The idea of mutualism, past and future. In: Boucher DH (ed) The biology of mutualism: ecology and evolution, Oxford University Press, New York, pp. 1–28
- Grice EA et al (2009) Topographical and temporal diversity of the human skin microbiome. *Science* 324:1190–1192
- Henle J (1840) Von den Contagien und Miasmen und den contagiös-miasmatischen Krankheiten. Berlin
- Koch R (1884) Die Aetiologie der Tuberkulose. *Mittheilungen aus dem kaiserlichen Gesundheitsamte* 2:1–81
- Loeffler F (1884) Untersuchung über die Bedeutung der Mikroorganismen für die Entstehung der Diphtherie beim Menschen, bei der Taube und beim Kalbe. *Mittheilungen aus dem kaiserlichen Gesundheitsamte* 2:421–499

- Marcy Y et al (2007). Dissecting biological “dark matter” with single-cell genetic analysis of rare and uncultivated TM7 microbes from the human mouth. *Proc Natl Acad Sci USA* 104:11889–11894
- Sears CL (2005) A dynamic partnership: celebrating our gut flora. *Anaerobe* 11:247–251
- Turnbaugh PJ, Ley RE, Hamady M, Fraser-Liggett CM, Knight R, Gordon JI (2007) The human microbiome project. *Nature* 449:804–810

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