Virulence Factors of *Helicobacter pylori* – Ultrastructural Features

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The detection of *Helicobacter pylori* in gastric biopsy specimens of patients with gastritis, and gastric or duodenal ulcers has provided the basis for a new concept in the etiology of gastroduodenal diseases [1–4].

Little is known concerning the factors that allow the colonization of the gastric mucosa and the induction of gastritis. *H. pylori* entering the human digestive system encounter a battery of nonspecific and specific chemical and cellular defense mechanisms. The role of these mechanisms is to inactivate and eliminate foreign materials. By overruling the various defenses *H. pylori* is able to establish itself within the mucosa. As this occurs, the bacteria start to exert their pathogenic potential.

The phenomenon of mucosal infection by *H. pylori* is complex and multifactorial. Several biochemical properties may act individually or together to produce infection and disease. After identification of microbial factors in various strains of *H. pylori* exhibiting virulence-associated properties, it is necessary to evaluate their actual contribution to bacterial pathogenicity.

This paper deals in detail with the virulence factors of *H. pylori* and their influence on ultrastructural alterations of surface mucus cells. Additionally an attempt is made to identify strains of high and low virulence on the basis of the morphological features. The investigated virulence factors, their proposed function, and their relation to ultrastructural alterations are summarized in Table 1.

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**Table 1.** Virulence factors of *H. pylori* and their relation to pathological alterations

<table>
<thead>
<tr>
<th>Virulence factor</th>
<th>Reference</th>
<th>Pathological alterations</th>
</tr>
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<tbody>
<tr>
<td>Motility (shape, flagella)</td>
<td>[5, 7, 8]</td>
<td>Favors colonization</td>
</tr>
<tr>
<td>Urease</td>
<td>[12, 13]</td>
<td>Neutralizes gastric acid</td>
</tr>
<tr>
<td>Protease</td>
<td>[20, 23]</td>
<td>Breakdown of mucus network</td>
</tr>
<tr>
<td>Lipolytic activity</td>
<td>[29, 30]</td>
<td>Generation of lysolecithin</td>
</tr>
<tr>
<td>Cytotoxins</td>
<td>[34, 35]</td>
<td>Intraplasmatic vacuolization</td>
</tr>
<tr>
<td>Adhesins</td>
<td>[37, 39]</td>
<td>Binding to the receptor</td>
</tr>
</tbody>
</table>

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Shape – Flagella – Motility

*H. pylori* appears to be specifically adapted to the viscous milieu of the gastric mucus. The spiral shape and the active flagella enable the bacteria to move easily in the viscous environment [5]. Motility is an established virulence factor for bacterial pathogens [6, 7] and it was shown by Eaton et al. [8] that the most motile strain of *H. pylori* is the most virulent with 100% infection rate in gnotobiotic piglets. In recent studies it was shown that flagella do not only provide motility, but they bear adhesins and are thus more directly involved in the actual adhesion event [9]. In addition, flagella of *H. pylori* have bulbs on their ends (Fig. 1) similar to those seen in Vibrio species. These bulbs may essentially favor the adhesion.

In several isolated strains of *H. pylori* we were able to demonstrate a so-called polar organelle (Fig. 2). This organelle presumably is part of the flagellar

Fig. 1a–c. The flagellar characteristics of *H. pylori*. a 4–6 flagella originate from one pole. Arrows point at the typical "terminal bulb." Negative staining; ×20000. b Negative staining; ×160000. c Ultrathin section; ×160000

Fig. 2a, b. The "polar" organelle of *H. pylori*. a This organelle is occasionally found along the membrane; ×35000. b Ultrastructural characteristics; ×170000