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conditions exhibit immunological evidence of a contact of *Listeria* antigens with immunological tissues but the carrier state appears as transient.

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***In vitro* infection of intestinal finite and continuous cell lines by *L monocytogenes*.** P Velge, B Kaeffer, E Bottreau, P Pardon (INRA, Centre de Recherche de Tours-Nouzilly, Laboratoire de Pathologie Infectieuse et Immunologie, 37380 Nouzilly, France)

Listeria-contaminated foods have been associated with 6 recent human listeriosis outbreaks in Europe and North America indicating that the gastro-intestinal tract is the most probable site of entry into the host (Jones, 1990). Unfortunately, we are still uncertain about the precise nature of the cells that are the natural target for the intestinal translocation of *L monocytogenes*. Indeed, controversial results were obtained after experimental *in vivo* infections (Racz *et al*, 1972; MacDonald *et al*, 1980). Numerous studies reported that *L monocytogenes* is able to enter several spontaneous continuous cell lines and to multiply within. However, these *in vitro* invasions were not performed with finite cell lines, another model to study cell-bacteria interactions (finite line refers to a finite life span whereas continuous line refers to an infinite life span). In this context, we established a collection of fibroblastoid and epithelioid finite cell lines from the ileum of an adult histocompatible minia-

ture boar (Kaeffer *et al*, 1993). All the virulent *Listeria* strains tested exhibited a weak penetration and a lack of intracellular multiplication inside these finite cell lines compared with the continuous cell lines. To demonstrate that the cell immortalization modified *Listeria* invasion, we transfected the finite cell lines with the plasmid pSV3-neo containing the SV40 large T oncogene to obtain continuous cell lines (Kaeffer *et al*, 1993). Our immortalized cell lines became permissive to all the virulent *L monocytogenes* strains used, as observed with CaCo-2 cells and contrary to their untransformed counterparts. These data support the hypothesis that *Listeria* penetrates an enterocyte subset at a defined state of differentiation or expressing particular receptors (Velge *et al*, submitted for publication). This is in agreement with the *in vivo* cell differentiation where the progressive crypt to villus migration of the epithelial cells occurs by the interplay of expression/loss of functions or synthesis of specific receptors (Louvard *et al*, 1992).

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