Antonie van Leeuwenhoek **58**: 87–93, 1990.

Relationship between intestinal microecology and the translocation of intestinal bacteria

C.L. Wells
Departments of Laboratory Medicine and Pathology and Surgery, University of Minnesota, Minneapolis, MN 55455, USA

Received 2 January 1990; accepted 5 January 1990

Abstract

It is now well known that endogenous bacteria can translocate from the intestinal tract and cause many of the complicating infections seen in severely ill, hospitalized patients. Of the hundreds of bacterial species in the intestinal tract, relatively few aerobic/facultative species appear to translocate with any frequency. Van der Waaij and colleagues (1971, 1972a, 1972b) originally proposed that, by a process termed 'colonization resistance', strictly anaerobic bacteria prevented the intestinal overgrowth and subsequent translocation of these potentially pathogenic aerobic/facultative bacteria. Selective antimicrobial decontamination, designed to maintain colonization resistance, has been effective in reducing the incidence of infectious morbidity in high risk patients. However, the mechanisms controlling bacterial translocation remain unclear, but appear to depend on host factors, as well as on factors inherent in the microbe itself. There is both clinical and experimental evidence supporting the concept that strictly anaerobic bacteria do not readily translocate. Bacteria that are able to survive within macrophages (e.g., *Salmonella* species and *Listeria monocytogenes*) translocate easier than others, and there is recent experimental evidence that normal intestinal bacteria may translocate to the draining mesenteric lymph node within host phagocytes. There is also evidence that anaerobic bacteria translocate along with facultative species in situations associated with intestinal epithelial damage, i.e., burn trauma, oral ricinoleic acid, and acute mesenteric ischemia. In contrast, recent experimental evidence demonstrates that facultative bacteria can translocate across a histologically intact intestinal epithelium, and that the ileal absorptive cell may be at least one portal of entry prior to transport into deeper tissues. It is anticipated that further clarification of the routes and mechanisms involved in bacterial translocation will provide new insights into the treatment and prevention of a significant proportion of the infectious morbidity seen in severely ill, hospitalized patients.

Bacterial translocation and the concept of colonization resistance

It is now widely recognized that normal flora bacteria can leave the intestinal lumen and enter extraintestinal sites by a process called bacterial translocation. Severely ill patients typically considered to be at high risk for developing systemic disease from translocating intestinal bacteria include immunosuppressed patients, surgery patients, and trauma patients. In the early 1970s, Van der Waaij and colleagues (1971, 1972a, 1972b) proposed that translocation of intestinal bacteria might be controlled by a process termed 'colonization resist-

Antoni van Leeuwenhoek Lecture presented at the Annual Meeting of the Netherlands Society of Microbiology, Utrecht, 23 November, 1989.

ance'. According to this theory, the normal intestinal flora, primarily the strictly anaerobic microflora which do not readily translocate, function to control the intestinal colonization and translocation of potentially pathogenic species such as *Escherichia coli* and other Enterobacteriaceae, *Pseudomonas* species, and *Enterococcus* species. Based on this theory, many high risk patients have been treated with prophylactic antimicrobial agents designed to selectively eliminate potentially pathogenic intestinal bacteria, while maintaining the populations of strictly anaerobic species. There is considerable evidence that this approach has been extremely effective in reducing the number of complicating infections in high risk patients (Clasener et al. 1987; Guiot & Van Furth, 1984; Kerver et al. 1988).

Clinical evidence for bacterial translocation

Several clinical investigators have noted that surveillance stool cultures could be used, not only to identify patients at high risk for bacteremia, but also to predict the antibiogram of the organism subsequently isolated from systemic infection (reviewed in Wells et al. 1988c). Possibly the most extensive study of this type was that reported by Tancrede and Andremont (1985) who prospectively identified and quantitated bacteria in 4,347 stool specimens from 688 hospitalized cancer patients receiving no prophylatic antimicrobial therapy; 60 patients developed 64 cases of gram-negative bacteremia that appeared to be caused by a dominant fecal organism that translocated from the patients' intestinal tract during a period of severe granylocytopenia. There have also been many clinical studies documenting that selective antimicrobial decontamination of the intestinal tract (i.e., eliminating the potentially pathogenic aerobic and facultative bacteria while maintaining the strictly anaerobic population) decreased the incidence of infectious morbidity in severely ill patients. A recent example of a well-designed study in this area was that of Kerver et al. (1988) who prospectively studied 96 mechanically ventilated, intensive care patients randomized to receive either no prophylatic antibiotics or a regimen consisting of intravenous cefotaxime for five to seven days (until oropharyngeal and tracheal cultures revealed no microorganisms) followed by a mixture of three oral antibiotics (polymyxin E, tobramycin, and amphotericin B). In this study, the control patients had 107 nosocomial infections as opposed to 42 infections in the treated group. As a result of this and similar studies, it is now well-recognized that selective antimicrobial decontamination can be extremely effective in reducing the incidence of infectious morbidity. However, the effect of selective decontamination on the incidence of mortality is less clear (Clasenar et al. 1987; Guiot & Van Furth 1984) and further studies are needed to resolve this issue.

Microbial factors that influence the incidence of bacterial translocation

It is evident that some microbes translocate easier than others. Of the hundreds of microbial species in the normal intestinal flora, relatively few have been noted to translocate with any frequency in experimental animals. These species include *E. coli, Klebsiella pneumoniae, Proteus mirabilis,* other Enterobacteriaceae, *Enterococcus* species, *Streptococcus* species, *Lactobacillus* species, *Candida albicans,* and possibly *Staphylococcus* species (Wells et al. 1988c). Curiously, these species are those most frequently associated with complicating infections in severely ill, hospitalized patients.

Bacterial species that translocate most readily are those considered to be facultative intracellular pathogens such as *Salmonella* species and *Listeria monocytogenes* (reviewed in Wells et al. 1988c). These intestinal bacteria have been shown to translocate after simple oral inoculation into normal animals. These species are considered facultative intracellular pathogens because they are able to replicate outside of host cells, but are also able to survive and replicate within host white blood cells. It may not be coincidental that there is evidence that host white blood cells, particularly the tissue macrophage, can play a role in transporting intestinal particles to extraintestinal sites (Wells et al. 1987a, 1988d). We instilled two different colors of

fluorescent latex beads into different isolated jejunal segments of individual dogs. Subsequent microscopic examination of mesenteric lymph node cells showed mononuclear phagocytes containing multiple beads of one or the other color, but not both colors, indicating that the phagocyte ingested the beads in the intestinal segment prior to transporting them to the draining mesenteric lymph node. (Similar work by Harmsen et al. [1985] also showed that alveolar macrophages could transport latex beads to the draining tracheobronchial lymph node.) If intestinal particles can be transported to the draining mesenteric lymph node within tissue phagocytes, it seems reasonable that bacteria considered to be facultative intracellular pathogens can translocate easier than other species.

Although intestinal anaerobic bacteria outnumber aerobic and facultative bacteria by 100 : 1 or 1000 : 1 (Moore & Holdeman 1975), anaerobes rarely translocate and rarely cause complicating infections in immunosuppressed/trauma patients. In 1971, Van der Waaij and colleagues studied gnotobiotic mice that were colonized exclusively with species of strictly anaerobic bacteria; the intestinal flora of these mice had a high degree of 'colonizing resistance' which led to the speculation that anaerobic bacteria might function to control the colonization and translocation of facultative bacteria. Direct comparisons of the relative translocation incidences of aerobic, facultative, and anaerobic bacteria have been reported by several investigators. Steffen et al. (1988) studied 13 different groups of mice monoassociated with similarly high cecal population levels of a single aerobic, facultatively anaerobic, or obligately anaerobic species of bacteria; the anaerobic species had the lowest incidences of translocation. Our group (1988a) studied defined-flora mice colonized with from two to seven species of various mixtures of facultative and anaerobic species; although anaerobes and facultatives colonized the ceca at similarly high concentrations, the incidence of translocation of anaerobic bacteria was significantly less than that of facultative species. Thus, there is both direct experimental evidence and indirect clinical evidence that anaerobic bacteria do not translocate as readily as aerobic and facultative species. The mechanism responsible for the preferential translocation of aerobic and facultative bacteria is unclear but appears to be independent of the concentration of intestinal bacteria.

Bacterial translocation in mice with selective elimination or retention of intestinal anaerobic bacteria

To challenge the hypothesis that anaerobic bacteria have a protective function and actually prevent the intestinal overgrowth and translocation of facultative species, we (1987b, 1988a) studied mice that had been treated with parenteral metronidazole. After three days of metronidazole treatment, the anaerobic cecal flora was selectively eliminated resulting in a concomitant overgrowth of cecal facultative bacteria; these facultative species (primarily *E. coli* and enterococci) translocated to the mesenteric lymph nodes of the majority of mice. Thus, the selective elimination of anaerobic bacteria facilitated the intestinal overgrowth and translocation of facultative bacteria.

In a subsequent experiment, mice were also treated with neomycin in an attempt to induce intestinal overgrowth and translocation of strictly anaerobic bacteria. Separate groups of female 18 to 22 g Swiss Webster mice were treated for three days with either 4 mg/ml neomycin sulfate (Upjohn Co., Kalamazoo, MI, USA) in the drinking water or 4 mg/0.2 ml/mouse intramuscular B.I.D. metronidazole (Searle Phatmaceuticals, Inc., Chicago, IL, USA) diluted in phosphate buffered saline. Mice were then killed and cecal and mesenteric lymph node bacteria were quantitatively cultured both anaerobically and aerobically as described (Wells et al. 1987b, 1988b). The limits of detection were 500 bacteria for the cecum and 10 bacteria for the MLN. The numbers of cecal bacteria were analyzed by the Student t-test and the numbers of mice with translocating bacteria were analyzed by the Chi square test.

As seen in Table 1, occasional control mice had translocating *E. coli* recovered from mesenteric lymph nodes. This was not an unusual finding. The literature contains repeated reports of the recovery

of viable intestinal bacteria from the mesenteric lymph nodes of a small percentage of normal mice, most likely as part of the normal antigen processing function of the gut-associated lymphoid tissue (Wells et al. 1988c). No anaerobic bacteria were recovered from the mesenteric lymph nodes of control or metronidazole-treated mice. Metronidazole therapy selectively eliminated all cecal anaerobic bacteria with a 10 to 100-fold increase in the numbers of facultative gram-negative and gram-positive bacteria; translocating facultative bacteria were recovered from the majority of these mice and were identified primarily as *E. coli* and *Enterococcus* species (Table 1). Neomycin therapy eliminated all cecal aerobic and facultative bacteria (except relatively low numbers of gram-positive sporeformers), and the numbers of strictly anaerobic bacteria increased approximately 100-fold; however, only low numbers of anaerobic bacteria were recovered from the mesenteric lymph nodes of a small percentage of these mice (Table 1). Thus, even after intestinal anaerobic species were induced to overgrow to artificially high population levels in response to neymycin therapy, anaerobes translocated in low numbers in only a small percentage of mice.

Bacterial translocation across an intact and a damaged intestinal mucosa

It is likely that the mechanism of bacterial translocation across the intestinal tract differs across an intact and a damaged intestinal mucosa. Indirect evidence for this is provided by the repeated observation that although strictly anaerobic bacteria outnumber facultative species by 100 : 1 or 1000 : 1, anaerobes typically translocate only if the integrity of the intestinal epithelium has been compromised. Animal models of bacterial translocation that involve histological alterations to the intestinal epithelium include lethal irradiation (Brook et al. 1984), burn trauma (Maejima et al. 1984), oral ricinoleic acid (Morehouse et al. 1986), and mesen-

Table 1. Cecal and mesenteric lymph node bacteria in mice with a selective retention or elimination of cecal anaerobic bacteria in response to three days of neomycin or metronidazole therapy.

Drug	Avg. log ± SE viable bacteria/g cecum[a]			No. of mice with viable mesenteric lymph node bacteria Total no. of mice[b]	Identity of translocating mesenteric lymph node bacteria
	Aerobic and facultative gram-negative bacilli	Aerobic and facultative gram-positive bacteria	Strict anaerobes		
Control	6.7 ± 0.6	6.9 ± 0.3	9.2 ± 0.7	5/30 (17%)	*E. coli*[c]
Neomycin	ND[d]	3.6 ± 1.4[e]	11.5 ± 0.4[f]	6/30 (20%)[g]	*Bacteroides* species[h]
Metronidazole	9.2 ± 0.3	9.7 ± 0.5	ND	25/30 (83%)[i]	facultative species[j]

[a] Results represent one of three similar experiments with four mice per group.

[b] Results represent pooled data from three separate experiments with ten mice per group.

[c] The numbers of translocating *E. coli* ranged from 10 to 150 per mesenteric lymph node.

[d] ND, none detected

[e] These microbes were identified as sporeformers, i.e., *Bacillus* species and fungi.

[f] Significantly increased compared to control group, $P < 0.01$.

[g] Significantly increased compared to the numbers of control mice (0/30) with strictly anaerobic bacteria recovered from mesenteric lymph node, $P < 0.05$.

[h] Each of six mice had 10 bacteria recovered from mesenteric lymph node. These bacteria were identified as either *B. vulgatus, B. fragilis, B. fragilis* group, or *Bacteroides* species.

[i] Significantly increased compared to control group, $P < 0.01$.

[j] Individual mice had from 10 to 3000 translocating bacteria per mesenteric lymph node. These bacteria were identified as *E. coli, Enterococcus* species, *Proteus mirabilis, K. pneumoniae, E. coli* A–D, *Citrobacter freundii,* and alpha-streptococci, in decreasing order of frequency. From one to four different species were recovered from a single mouse mesenteric lymph node.

teric ischemia (Bennion et al. 1984). In each of these animal models, all bacterial species appear to translocate, and aerobic and facultative species translocate along with strict anaerobes. In general, the greater the degree of epithelial and mucosal damage, the more frequently anaerobic bacteria are detected as translocating agents. Thus, it appears that situations that permit the translocation of anaerobic bacteria are those in which the intestinal tract is mechanically damaged, possibly resulting in the destruction of the mechanism that controls the selective translocation of aerobic and facultative species.

Although most of the studies involving bacterial translocation do not include a study of intestinal histology, there are several reports documenting that bacteria can translocate across an intact intestinal mucosa. Berg et al. (1988) recently reported that intestinal bacterial overgrowth coupled with immunosuppression (cyclophosphamide or prednisone) synergistically promoted increased levels of bacterial translocation in mice with a histologically normal intestinal tract. Several years ago, we (1986) reported that normal intestinal bacteria (primarily *E. coli* and enterococci) appeared to translocate from a histologically normal intestinal tract to an experimental intraabdominal abscess that had been initiated with defined bacterial species. (Several other investigators have also reported that experimental intraabdominal abscesses became unexplainably contaminated with normal intestinal bacteria [McConville et al. 1981; Onderdonk et al. 1974]). We have also recently studied two different mouse models involving antibiotic manipulation of the mouse intestinal flora coupled with oral inoculation, intestinal overgrowth, and translocation of an antibiotic resistant strain of bacteria. One model involved treatment with either streptomycin, bacitracin-streptomycin, or metronidazole plus a streptomycin-resistant strain of *E. coli* (1987b, 1988e); the other model involved treatment with either metronidazole-streptomycin or clindamycin-streptomycin plus an antibiotic resistant strain of enterococcus (1988b). These studies documented increased incidences of translocation of *E. coli* and enterococcus in mice with a histologically intact intestinal epithelium.

The route of bacterial translocation

There are multiple possible routes by which a microbe can translocate out of the intestinal tract to extraintestinal sites – retrograde migration into the lung, direct transmural migration across the bowed wall, and migration into the mesenteric lymph nodes and/or liver by way of lymphatic and/or vascular channels. In 1950, Sweinburg et al. noted that oral (but not intravenous) *E. coli* was attracted to a sterile irritant (e.g., inflammatory focus) in the peritoneal cavity by an undefined mechanism termed 'transmural migration'; presumably, bacteria pass through the intestinal mucosa into the abdominal cavity by a route that has not received further study, but may be involved in the translocation of intestinal bacteria into intraabdominal abscesses. There is also substantial evidence (Pingleton et al. 1986; Van Uffelen et al. 1987) that upper intestinal overgrowth facilitates aspiration and retrograde migration of intestinal bacteria into the lung of severely ill trauma patients, primarily those on mechanical ventilation. In 1979, Berg and Garlington defined bacterial translocation as the passage of viable bacteria from the gastrointestinal tract to the mesenteric lymph nodes and possibly other organs. Consistent with this definition, it has been repeatedly shown that the mesenteric lymph node is the most sensitive organ to culture in order to monitor bacterial translocation in many animal models. However, in studying the route of bacterial translocation, investigators should be aware that one or more routes of bacterial translocation may be operating at the same time in a particular patient or in a particular animal model.

Possible cell types involved in regulating bacterial translocation across an intact intestinal epithelium

We recently proposed (1988c) that the intestinal macrophage may play a key role in the translocation of intestinal bacteria. If this is true, then bacteria that translocate easier than others should be those that are either ingested more readily by macrophages or those that are better equipped for intracellular survival within macrophages. Consis-

tent with this hypothesis is the observation that facultative intracellular pathogens, such as *Salmonella* species or *Listeria monocytogenes*, appear to translocate with relative ease. As described above, it is evident that facultative species, such as *E. coli* and *Enterococcus* species, translocate more readily than strictly anaerobic species. The mechanism responsible for this phenomenon is unknown. Perhaps anaerobic bacteria are either less readily ingested by tissue macrophages or are more susceptible to intracellular killing within these phagocytes. Unfortunately, the comparative interactions of facultative and anaerobic bacteria with tissue macrophages are essentially unknown.

As an alternative hypothesis, it is possible that the intestinal epithelial cell may play a pivotal role in controlling the incidence of bacterial translocation. There is evidence that enterocytes can act as fixed phagocytes. Various investigators have observed inert particles (ferritin), *Salmonella* species, and *Listeria monocytogenes* within intestinal epithelial cells (reviewed in Wells et al. 1988c). We recently studied enterococcal translocation induced by metronidazole-streptomycin manipulation of the intestinal flora; using immunofluorescence and transmission electron microscopy, translocating *E. faecalis* was localized within ileal enterocytes (Wells et al. 1990). The incidence of translocation may be controlled by the ability of the translocating microbe be taken up by intestinal epithelial cells. Perhaps strictly anaerobic bacteria are not readily taken up by enterocytes, or perhaps strictly anaerobic bacteria lack the receptor sites necessary to attach to the epithelial surface. This hypothesis is consistent with the observation that translocation of anaerobic bacteria is facilitated in those situations involving histological damage to the intestinal epithelium.

Our current working hypothesis for the route of bacterial translocation across an intact intestinal epithelium is as follows: Bacteria are taken up by absorptive epithelial cells, enter the lamina propria, and then either enter lymphatic channels that drain into the mesenteric lymph nodes or enter vascular channels that first go to the liver and then other organs. Any or all of this journey may taken place within tissue phagocytes. As described above, evidence from our laboratory indicates that this might be at least one route of bacterial translocation across an intact intestinal epithelium. However, other anatomical routes have not been ruled out. A clarification of these routes and mechanisms should lead to new treatment regimens designed to decrease the costly morbidity and mortality associated with systemic infections caused by translocating intestinal bacteria.

Acknowledgement

This work was supported in part by Public Health Service Grant AI 23484 from the National Institutes of Health, USA.

References

Berg RD & Garlington AW (1979) Translocation of certain indigenous bacteria from the gastrointestinal tract to the mesenteric lymph nodes and other organs in a gnotobiotic mouse model. Infect. Immun. 23: 403–411

Berg RD, Wommack E & Deitch RA (1988) Immunosuppression and intestinal bacterial overgrowth synergistically promote bacterial translocation. Arch. Surg 123: 1359–1364

Bennion RS, Wilson SE, Serota AI & Williams RA (1974) The role of gastrointestinal microflora in the pathogenesis of complications of mesenteric ischemia. J. Infect. Dis. 6 (Suppl): S132–S138

Brook L, MacVittie TJ & Walker RI (1984) Recovery of aerobic and anaerobic bacteria from irradiated mice. Infect. Immun. 46: 270–271

Clasener HAL, Vollard EJ & Van Saene HKF (1987) Long-term prophylaxis of infection by selective decontamination in leukopenia and in mechanical ventilation. Rev. Infect. Dis. 9: 295–328

Guiot, HFL & Van Furth R (1984) Selective antimicrobial modulation of the intestinal flora. Prophylaxis against infection in neutropenic patients. Infection. 12: 1–4

Harmsen AG, Muggenburg BA, Snipes MB & Bice DE (1985) The role of the macrophage in particle translocation from lungs to lymph nodes. Science 230: 1277–1280

Kerver AJH, Rommes JH, Verhage EAE, Hulstaert PF, Vos A, Verhoef J & Wittebol P (1988) Prevention of colonization and infection in critically ill patients: A prospective randomized study. Crit. Care Med. 16: 1087–1093

Maejima K, Deitch EA & Berg RD (1984) Bacterial translocation from the gastrointestinal tracts of rats receiving thermal injury. Infect. Immun. 43: 6–10

McConville JH, Snyder MJ, Calia FM & Hornick RB (1981)

Model of intraabdominal abscess in mice. Infect. Immun. 31: 507–509

Moore WEC & Holdeman LV (1975) Discussion of current bacteriological investigations between intestinal flora, diet, and colon cancer. Cancer. Res. 35: 3418–3412

Morehouse JL, Specian RD, Stewart JJ & Berg RD (1986) Translocation of indigenous bacteria from the gastrointestinal tract of mice after oral ricinoleic acid. Gastroenterol. 91: 673–682

Onderdonk AB, Weinstein WM, Sullivan NM, Bartlett JG & Gorbach SL (1974) Experimental intra-abdominal abscesses in rats: Quantitative bacteriology of infected animals. Infect. Immun. 10: 1256–1259

Pingleton SK, Hinthorn DR & Liu C (1986) Enteral nutrition in patients receiving mechanical ventilation. Multiple sources of tracheal colonization include the stomach. Am. J. Med. 80: 827–832

Schweinberg FB, Seligman AM & Fine J (1950) Transmural migration of intestinal bacteria: A study based on the use of radioactive *Escherichia coli*. N. Engl. J. Med. 242: 747–751

Steffen EK, Berg RD & Deitch EA (1988) Comparison of the translocation rates of various indigenous bacteria from the gastrointestinal tract to the mesenteric lymph node. J. Infect. Dis. 157: 1032–1038

Tancrede CH & Andremont A (1985) Bacterial translocation and gram-negative bacteremia in patients with hematological malignancies. J. Infect. Dis. 152: 99–103

Van der Waaij D, Berghuis-De Vries JM & Lekkerkerk-Van der Wees (1971) Colinization resistance of the digestive tract in conventional and antibiotic-treated mice. J. Hyg. 69: 405–411

Van der Waaij D, Berghuis-de Vries JM & Lekkerkerk-Van der Wees (1972a) Colonization resistance of the digestive tract and the spread of bacteria to the lymphatic organs in mice. J. Hyg. 70: 335–342

Van der Waaij D, Berghuis JM & Lekkerkerk JEC (1972b) Colinization resistance of the digestive tract of mice during systemic antibiotic treatment. J. Hyg. 70: 605–609

Van Uffelen R, Rommes JH & Van Saene HKF (1987) Preventing lower airway colonization and infection in mechanically ventilated patients. Crit. Care Med. 15: 99–102

Wells CL, Rotstein OD, Pruett TL & Simmons RL (1986) Intestinal bacteria translocate into experimental intra-abdominal abscesses. Arch. Surg. 121: 102–107

Wells CL, Maddaus MA & Simmons RL (1987a) Role of the macrophage in the translocation of intestinal bacteria. Arch. Surg. 122: 48–53

Wells CL, Maddaus MA, Reynolds CM, Jechorek RP & Simmons RL (1987b) The role of the anaerobic flora in the translocation of aerobic and facultatively anaerobic intestinal bacteria. Infect. Immun. 55: 2689–2694

Wells CL, Jechorek RP & Maddaus MA (1988a) The translocation of intestinal facultative and anaerobic bacteria in defined flora mice. Microb. Ecol. Health Dis. 1: 227–235

Wells CL, Jechorek RP, Maddaus MA & Simmons RL (1988b) The effects of clindamycin and metronidazole on the intestinal colonization and translocation of enterococcus in mice. Antimicrob. Agents Chemother. 32: 1769–1775

Wells CL, Maddaus MA & Simmons RL (1988c) Proposed mechanisms for the translocation of intestinal bacteria. Rev. Infect. Dis. 10: 958–979

Wells CL, Maddaus MA, Erlandsen SL & Simmons RL (1988d) Evidence for the phagocytic transport of intestinal particles in dogs and rats. Infect. Immun. 56: 278–282

Wells CL, Maddaus MA, Jechorek RP & Simmons RL (1988e) Role of intestinal anaerobic bacteria in colonization resistance. Eur. J. Clin. Microbiol. Infect. Dis. 7: 107–113

Wells CL, Jechorek RP & Erlandsen SL (1990) Evidence for the translocation of *Enterococcus faecalis* across the mouse intestinal tract. J. Infect. Dis. (in press)