

Amazingly we found only rare evidence in the families for a toilet respectively kitchen community of the coliflora with regard to the plasmid and the colitype content. Plasmid and coliform transfer were observed between newborn and mother, between newborn and sisters and brothers but only once between newborn and father. Without influence of antibiotics the plasmid status of each member of the families was astonishingly stable for long periods.

The susceptibility of *Pseudomonas pseudomallei* to six β -lactams and five other antibiotics in vitro

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We determined the minimal inhibitory concentrations (MIC) and the minimal bactericidal concentrations (MBC) of 11 compounds against 20 clinical isolates of *Pseudomonas pseudomallei* from Thailand. We worked with Mueller-Hinton broth (micro-dilution technique, NCCLS) and Mueller-Hinton agar. The most active compounds were weight-for-weight ($\mu\text{g/ml}$) in terms of MIC₉₀, MBC₉₀. Range (MIC): Ro 17-2301 (4, 2, 1-128), Cefazidime (CAZ) (64, 2, 0.5-128), Ciprofloxacin (CIP) (8, 8, 2-8), Co-trimoxazole (SXT) (2, 4, 1-4). Although most strains were resistant to both sulfamethoxazole (S) and trimethoprim (T), the combination SXT was highly active against all isolates tested. Ro 17-2301 and CAZ were as a rule fourfold more active than ceftriaxone, latamoxef and aztreonam. We conclude that structurally related β -lactams may have different potency in vitro against *P. pseudomallei* and that Ro 17-2301 and ceftazidime were the most active of those tested.

Antibiotic susceptibility of anaerobic bacteria determined by agar dilution

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250 anaerobic bacteria, being probably of clinical significance, were identified by gram stain of culture smears, gas liquid chromatography (Holdeman et al., Anaerobe laboratory manual, 1977) and with API 20 A. Susceptibility testing was performed according to the recommendations of the National Committee for Clinical Laboratory Standards (NCCLS) (M 11-T 1982) using Wilkins-Chalgren agar containing 5% sheep blood and Adatab antimicrobial tablets (Mast). The break-points were chosen as recommended by the NCCLS M 7-T 1983: Penicillin G 0.125 mg/l, Amoxycillin/Clavulanate 8/4 mg/l, Carbenicillin 32 mg/l, Cefoxitin 8 mg/l, Metronidazole 16 mg/l, Chloramphenicol 8 mg/l, Clindamycin 0.5 mg/l, Tetracyclin 1 mg/l. In agreement with other publications, Amoxycillin/Clavulanate and Chloramphenicol proved to be very efficient antimicrobials against anaerobic germs in vitro. Furthermore, it is noteworthy that 30% *Peptococcus* and 70% *Peptostreptococcus* species were resistant to Metronidazole. Therefore, Metronidazole should not be considered as universal agent against infections caused by anaerobic bacteria.

OXA-2 and TEM-1 beta-lactamase DNA probes for epidemiological studies

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DNA probes seem promising for molecular epidemiology of antibiotic resistance. Two probes have been produced as markers of beta-lactamase (Bla) producing strains. TEM-1 Bla probe consists of λ bb DNA carrying Tn 2301 originated from plasmid R 111. OXA-2 Bla probe was constructed with plasmid

R 46 as donor and pBR 327 as vector. pBR 327 was digested by *Bam* HI and *Pst* I, leading to isolation of a fragment carrying the origin of replication, without the AMP gene. Plasmid R 46 was digested by *Bam* HI, and the fragment carrying the *Bla* gene was purified and digested by *Pst* I. Vector and donor *Bam* HI-*Pst* I fragments were ligated and introduced into *E. coli* C600 by transformation. One clone producing OXA-2 Bla has been then isolated. These TEM and OXA₂ Bla DNA probes have been tested against a collection of strains producing known Bla, and will be used as epidemiological markers.

Beta-lactamases produced by *Campylobacter jejuni*

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Out of 20 sporadic isolates of *C. jejuni* (6 resistant and 14 susceptible to ampicillin (A), 18 had Betalactamase (Bla) activity (0.01 to 1.5 IU per ml of undiluted sonic extract). Bla have been characterized after partial purification by gel filtration: profil of activity by a bio assay, relative rates of hydrolysis, inhibition profile and immunological specificity at the spectro, IEF in polyacrylamide gel and mol. wt in column. Bla type A (18 out of a collection of 23 Bla producing strains) is active against peni (P), oxa (O) and to a lesser extend carbenicillin (C), pI: 8.3, mol. wt: 30 K; neutralized by homologous rabbit antiserum. Bla type B (1/23 strain) is similar to Bla type A, but also hydrolyzes cephaloridine and cefuroxim; IEF: 1 major band at 8.6, accessory band at 8.3 and 8.1. Bla type C (1/23) is active against P, A, C, O, cephalothin, cephaloridine and cefotaxime, but not cefuroxim. pI: 8.3, mol. wt: 45 K, neutralization by anti Bla type A antiserum. Bla type D (1/23) is mainly active P, less against A, O and C, pI: 7.4, mol. wt: 45 K, no neutralization by anti Bla type A. Bla may be involved.

Immunology

Suppressive mechanism of cyclosporin A: blocked IL-2 secretion of Con A activated T-cell hybridomas

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The fungal metabolite cyclosporin A (Cy A) is a powerful immunosuppressant with apparent selective action on lymphocytes (mainly T-cells). The mechanism of suppression is still controversial. The examination of the mechanism, by which Cy A suppresses cellular immune responses, is complicated by the fact that T-cell activation is dependent on the presence of antigen presenting cells, lymphokines, expression of lymphokine receptors and immune-response related cell proliferation. In order to further define the suppressive mechanism of Cy A, we searched for an assay system which is free of these complications. The activation of T-cell hybridomas with Con A, in the absence of accessory cells, provides the desired experimental conditions. We found that pharmacologic levels of Cy A (25 ng/ml) strongly inhibited IL-2 secretion, but had no effect on DNA synthesis of T-cell hybridomas. This observation suggests, that the primary mechanism of Cy A mediated suppression is due to the blocking of the T-lymphocyte activation pathway, leading to IL-2 secretion.