after in vitro susceptibility testing, a combination of imipenem plus aminoglycoside was used more frequently (59%). Ceftazidime along with an aminoglycoside was used in 30% of cases. Ceftazidime combined with quinolone was used rarely (11%). In general, the recommended drugs in most of the studies have been either extended-spectrum penicillins, broad-spectrum cephalosporins, or imipenem, combined with an aminoglycoside.

CHAPTER 11

ACINETOBACTER SPP; Prevalence and Infections - Indian Scenario

11.1 Acinetobacter infections in India:

Several cases of Acinetobacter infections are reported from India. Most of these reports were referring to the organism using old nomenclature. Only after 1990 few studies from India being reported wherein, new taxonomic developments were acknowledged and used.

The first report dates back to 1963 where in Prakash et al. describes the strains resembling Bacterium anitratum isolated from patients in Delhi. Seventeen strains of Bacterium anitratum were isolated from blood, urine, sputum and conjunctivae and studied for their pathogenesis. Further studies on this by the same group elucidated a case of septicaemia in pediatric patient due to Bacterium anitratum (Prakash et al., 1963b). In 1964 Mukerji isolated and studied bacteria resembling Bacterium anitratum in specific group of patients.
In 1965 Sahadevan et al. reported three cases of meningitis due to *Mima polymorpha*, the non-acid producing strains of genus *Acinetobacter*. In 1968 Madhavan et al. studied strains of *Mima polymorpha* isolated from patients belonging different geographical area in India such as Pondicherry. In the same year Sood and Madhavan described an unusual case of ophthalmia neonatorum caused by *Mima polymorpha*. According to this report conjunctivitis infection caused by *Mimeae* arises very consistently from the fifth to the seventh day after birth and may persist for as long as two months. It was also observed in this study that a change from an unbuffered silver nitrate use to a buffered one used in the eyes at birth reduces the number of such cases of infections. Here treatment failure with penicillin was a noted difference from that of infections with Neisseria.

In 1969, first case of meningitis due to *Bacterium anitratum* in India was reported (Madhavan and Jayakrishna, 1969). Later a study from Pondicherry highlighted the important reservoirs of *Bacterium anitratum* and *Mima polymorpha* in patients (Venkataramani et al., 1972). Skin and mucous membrane were shown to be reservoirs of *Bacterium anitratum* and *Mima polymorpha*. Out of 35 patients 7 were positive for carriage on their hand web space, axilla, forearm, skin around abdomen and nose. Out of 70 normal subjects 6 were positive for the bacteria on their hand web space and axilla. Among 28 other cases even though the organism was not isolated from skin and mucous membrane, the organism was clearly implicated in the disease in at least 4 cases studied. The study concluded that these bacteria exists as commensals on the normal human skin and occasionally produce specific human
infection and certain criteria were also proposed to label these bacteria as definite causative agent in specific human infection.

After reclassification of group Mimeae to the generic name *Acinetobacter* including two species namely *Acinetobacter calcoaceticus* and *Acinetobacter lwaffi* (Henriksen 1976; Skerman et al., 1980), only few reports from India regarding *Acinetobacter* appeared in literature. In 1978 a case report from Bombay (now Mumbai) described postoperative urinary tract infection by *Acinetobacter calcoaceticus* (Dalal et al., 1978). In this study, *A. calcoaceticus* was repeatedly isolated in significant numbers on three occasions over a three month period in a female aged 46 years, who had been operated for a congenital stenosis at the right pelviureteric junction. However the patient's serum did not show antibodies to antigens prepared from the organism. Authors were skeptical about the role of organism in this infection.

In 1980, Sachdev et al. reported a case of fatal acinetobacter meningitis, septicemia and bilateral otitis media in a 10-year old male child. The authors also briefly reviewed the reported cases of *Acinetobacter* meningitis in the English literature. They came across a total of 47 cases of meningitis caused by *Bacterium anitrarum* and *Mima polymorpha*; both were previous species names of genus *Acinetobacter*.

In the above study they could isolate *A. calcoaceticus* from the blood and CSF which was resistant to many antibiotics. The presence of Gram-negative coccobacilli in aural swab also pointed out that ear was the portal of entry for the bacteria in this case. The usual CSF cytology in acinetobacter meningitis
predominantly polymorphonuclear in type, however case it was lymphocytic in this
case. Authors conclude that the *Acinetobacter* has potential for fatal pathogenicity and
it also stands out in its ability to mimic meningococcal meningitis, both clinically and
in pathogenesis, and hence the organism should not be dismissed as a 'harm less'
commensal by the microbiologist and the clinician.

A rare report of *A. calcoaceticus* causing subacute bacterial endocarditis was
reported in one of the study in 1981 (Pal et al., 1981). Kulachandra Singh et al.,
(1983a&b) conducted a detail study on prevalence of *Acinetobacter calcoaceticus*
infections in coastal Karnataka (South India). A total of 106 isolates obtained from
both patients and hospital environment were studied. *A. calcoaceticus* was isolated
from 75 patients with diverse clinical manifestations such as meningitis, pancreatitis,
urinary tract infections (UTI), fever, bronchopneumonia, bronchitis, bronchiectasis,
ocorneal ulcer, middle ear infections and carbuncles. However, majority of the isolates
(45) were recovered from urine samples of patients with urinary tract infections.
Serum samples of UTI patients were tested for antibodies. Precipitation test was
found to be more sensitive than agglutination test in the serological diagnosis wherein
they used antigens prepared from the isolates obtained from the respective patients.
Multiple drug resistance was seen in about 75% of the isolates. The isolates from the
hospital environment (31) showed a higher percentage of resistance than the strains
recovered from patients. In the study it was stressed for the need of performing in
vitro susceptibility testing whenever feasible and suggested the use of the antigen
prepared from patient's own isolate for serological diagnosis. Major drawback of this
study was, only in 5 cases they were able to isolate the organism repeatedly and
despite of some deviations in biochemical properties few strains were grossly placed in genus Acinetobacter.

In another study from Mishra et al. (1986), nonfermenters other than Pseudomonas aeruginosa were identified from clinical material for over a period of one year. Out of 124 strains of nonfermenters, the commonest isolate in 75(60.4%) cases was Acinetobacter anitratus followed by Acinetobacter lwoffii comprising another 26 (20.9%) strains. Majority, that is 46.7 and 27.37 percent of Acinetobacter anitratus were isolated from pus and blood respectively, where as maximum number A. lwoffii isolates were from purulent fluid (47.8%) and blood (23.1%). Around 21-80 percent of the nonfermenters were resistant to commonly used antibiotics such as ampicillin, chloramphenicol, cloxacillin, cotrimoxazole, cephalaxin, tetracycline, streptomycin and kanamycin. In 28 i.e 70% of the 40 repeat samples, the same organism was reisolated. However, the sensitivity patterns of repeat isolates were similar only in five instances. This study also asserts that acinetobacters have an etiological role to play in infections.

Zaer and Deodhar (1989) studied 54 isolates of A. calcoaceticus over a period of 6 months. Maximum isolates were from burns cases and environmental sampling from burns ward also yielded the same organism. The authors assert that there is a need for proper identification since the organism always displays multiple drug resistance.

A particularly interesting outbreak of meningitis caused by Acinetobacter spp was reported in a group of children with leukaemia (Kelkar et al., 1989) from Mumbai following the administration of intrathecal methotrexate. Of the twenty
children who received intrathecal methotrexate, 8 returned within 2 to 19 h of treatment with signs and symptoms of acute meningeal irritation. *Acinetobacter* was isolated from the CSF of 5 of these patients, as well as from the methotrexate solution. Three of the children died as a result of meningitis and five recovered. The outbreak was caused by the use of inappropriately sterilized needles.

Most of the infections reported recently are in high risk group patients. A case of acute tracheitis caused by *Acinetobacter* was reported (Sarkar et al., 1991) from Chandigarh, where in hypomagnesemic hypocalcemia was the cause of persistent upper airway obstruction.

One comprehensive study from Ludhiana (Pearce et al., 1993) reports 5.6% incidence of acinetobacter meningitis. A total of 10,468 CSF samples from cases of meningitis in different age groups were cultured during 1988-1991. *Acinetobacter calcoaceticus* was identified in 12, of 211 positive bacterial cultures. All strains were 100% resistant to ampicillin, cotrimoxazole and tetracycline. 50% were resistant to cephazolin, gentamicin and kanamycin. However, all were susceptible to chloramphenicol.

In one more case, disseminated carcinoma stomach was the predisposing factor for the spontaneous bacterial peritonitis caused by *Acinetobacter* species. The patient was only thirteen years old from Lucknow (Agarwal et al., 1993).

*Acinetobacter* sepsis in neonates as well as their incidence rate was studied for four years (1986-1990) by a group of workers (Christo et al., 1993) in southern part of India, Manipal (Karnataka). Twenty-six neonates were diagnosed to have acinetobacter sepsis, representing 6.5% of all cases of bacteriologically proven sepsis
during four years period. The male and female ratio was 9:17. The major predisposing factor was low birth weight (19 neonates). All infants had clinical evidence of multi system involvement. The case fatality rate was 42.3% as 11 babies died. Except for gentamicin most of the isolates showed resistance to commonly used antibiotics. *Acinetobacter* was cultured from other sites like eye swabs, skin pustules and umbilical catheter tips. Environmental samples also yielded *Acinetobacter*, however they were not isolated simultaneously. The above study did not attempt to speciate and type the isolates, which is desirable for epidemiological purposes.

Another study from same place highlighted the role of *Acinetobacter* in bacteraemia in immunocompromised patients (Sugandhi Rao et al., 1993). *A. calcoaceticus* (62) was the second commonest nonfermenter isolated from 308 blood cultures. *Acinetobacter* was prevalent in patients with burns and terminal cancers. Most isolates were resistant to commonly used antibiotics. Sensitivity to ciprofloxacin and netilmicin ranged from 93-100% in different strains. In this *P. aeruginosa* bacteraemia was seen in 45.14 percent of patients and 30.09 percent was due to *A. calcoaceticus*. Even though maximum number of *A. calcoaceticus* was isolated from blood cultures of malignant patients, it was equally prevalent in patients with burns and septicaemia.

A detail study using recent rational taxonomic developments was reported only recently (Chopade et al., 1994 a&b). One hundred and seventy six strains of *Acinetobacter* isolated from various clinical sources. Baumann's medium was modified and used for enrichment of *Acinetobacter*. Five different genospecies of *Acinetobacter* were identified, and majority of them were multiple resistant to the
most of the antibiotics tested. *A. baumannii* was the most predominant genospecies obtained followed by *A. junii*, Genospecies 3, Genospecies 6 and Genospecies 12 (Table-11.1). Eighty five percent of strains were belonging to *A. baumannii* and further biotyping of *A. baumannii* revealed that biotype 7 was most prevalent followed by biotype 2. Overall 8 biotypes were obtained.

Table 11.1 Source and species of *Acinetobacter* from the study of Chopade et al., 1994.

<table>
<thead>
<tr>
<th>Clinical</th>
<th>Total no. of samples collected</th>
<th>Total no. <em>Acinetobacter</em> isolates</th>
<th><em>A. baumannii</em></th>
<th><em>A. junii</em></th>
<th>Genospecies 3</th>
<th>Genospecies 6</th>
<th>Genospecies 12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood</td>
<td>126</td>
<td>34</td>
<td>31</td>
<td>1</td>
<td>2</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Urine</td>
<td>157</td>
<td>72</td>
<td>58</td>
<td>10</td>
<td>4</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Sputum</td>
<td>26</td>
<td>13</td>
<td>13</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Pus</td>
<td>51</td>
<td>24</td>
<td>22</td>
<td>-</td>
<td>2</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>CSF</td>
<td>102</td>
<td>27</td>
<td>21</td>
<td>3</td>
<td>-</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Stool</td>
<td>36</td>
<td>3</td>
<td>3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>TS*</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>ICU</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

* Throat Swab

They also studied multiple antibiotics and metal resistance in strains of *Acinetobacter* and their elimination through curing the plasmids responsible for resistance. Conjugative antibiotic resistant plasmids were studied in 11 strains of *Acinetobacter*. Non-conjugative antibiotic and metal resistant plasmids were mobilised from 6 *Acinetobacter* species, using plasmids RP4 and R388. Plasmids thus obtained were used to transform *A. calcoaceticus* BD413, and *Escherichia coli* HB101. Molecular weights of plasmids were found to be in the range of 15-56Md.
Plumbagin was the most effective agent to cure antibiotic and metal resistant plasmids. Forty five percent of strains showed detectable beta lactamases. Plasmid pUPI 100 exhibited a high level of cephalosporinase in transconjugants as well as transformants.

The study group also felt the need of following approaches required in India to enhance the knowledge regarding Acinetobacter.

a) Survey of Acinetobacter infections in India.

b) Need for diagnostic kits and rapid identification methods

c) Monitoring studies on multiple antibiotic and metal resistance.

d) Genetic studies on pathogenicity of Acinetobacter

e) Studies on survival of Acinetobacter in nosocomial environment

f) Genetic and molecular basis of resistance as well as possibility of transfer of resistance genes from Acinetobacter to other pathogenic organisms and

g) Naturally occurring plasmids and transposons in Acinetobacter.

Bacterial meningitis caused by two organisms at the same time was reported from Madyapradesh (Sharma and Mohanthy, 1995). This mixed meningitis is in a young man caused by Acinetobacter calcoaceticus var lwoffii and Streptococcus faecium. Such an etiology has not been reported previously. The patient recovered completely without neurologic sequelae when treated with chloramphenicol and penicillin. Here old nomenclature was used to describe Acinetobacter genus.

An outbreak in the medical oncology ward was reported (Kapil et al., 1998) from Delhi, where nine patients suspected of bacteraemia were blood culture positive.
repeatedly, where in one specimen collected through the i v cannula while another through the peripheral venous puncture. The bacterium that was isolated from the environment was similar with that of clinical isolate as confirmed by cell protein profiles on SDS-PAGE. The source of infection can be from the environment, as use of disposable heparine ampoules and reinforcement of proper barrier nursing resulted in control of outbreak.

A case of acinetobacter meningitis following head injury in a patient, who later developed cerebrospinal fluid otorrhea, and did not have any neurosurgical procedure, was reported from Vellore (Venkataraman et al., 1999). In this case pefloxacin monotherapy was associated with a poor clinical response.

Community acquired ocular infections due to Acinetobacter spp is rare. Prashanth and Madhavaranga et al. (2000) from Pondicherry reported a case of corneal perforation due to Acinetobacter junii for which a therapeutic penetrating keratoplasty was done and patient eventually recovered. Most recently a case of ocular infection was described by Gopal et al. (2000) wherein postoperative endophthalmitis caused by sequestered A. calcoaceticus. sub sps lwoffii. Unfortunately they used old nomenclature to classify the Acinetobacter spp, thus it was not possible to accurately implicate a particular species within lwoffii phenotype (DNA groups 8/9,15) for the disease.

Above are few reports on Acinetobacter infections documented in India. Unfortunately, most of the studies used either old nomenclature to describe the organism or used invalid names like A. anitratus for reporting their cases. So there is very little knowledge about different species of Acinetobacter circulating in India,
which might be responsible for severe nosocomial infections. And most infections due to *Acinetobacter* are not being documented because of common belief lying in physicians regarding role of the bacteria.

**11.2 Antimicrobial susceptibility testing:**

Studies on resistance levels to antibiotics and metals were done and evaluated in few centres across the country (Despande et al., 1994). Despande and Chopade (1994) have reported an environmental isolate showing multiple drug resistance as well as metal resistance. *Acinetobacter baumannii* BL88 was resistant to more than 13 metals and 10 antibiotics. They detected a 54-kb plasmid (pUPI199) in *A. baumannii* BL88 conferring the above resistance. But the plasmid was stable only when there is a selection pressure. An earlier study from the same authors (Despande et al., 1993) found that biotype *A. baumannii* was most resistant to metals and resistance to mercury was prevalent only in beta-lactamase producing *A. baumannii* biotype.

In another study from the same place, correlation of metal resistance with that of antibiotic resistance was attempted (Dhakephalkar and Chopade, 1994). Forty strains isolated from different environmental samples comprised of strains belonging to four genospecies viz Thirty three isolates were of *A. baumannii*, 3 belonging to *A. calcoaceticus*, 3 *A. junii* and only one *Acinetobacter* genospecies 3. All isolates were resistant to multiple metal ions, while all but one of the strains was resistant to multiple antibiotics (minimum four antibiotics). Maximum number around 60% were
sensitive to mercury, while all were found resistant to copper, lead, boron and tungsten even at 10mM concentration. Ninety-four and 89.5% of strains were susceptible to rifampicin and nalidixic acid respectively. *Acinetobacter* genospecies 3 was found to be most resistant species showing resistance to almost to all metal ions and antibiotics tested. An inhibitory concentration of 10mM of Ni (2+) and Zn (2+) was observed to inhibit the growth of all of the clinical isolates but allowed the growth of the environmental isolates, thus facilitating the differentiation between pathogenic and nonpathogenic acinetobacters. This group also proposed a selective media based on the above findings.

Antibiotic resistance patterns and R-plasmids of *Acinetobacter calcoaceticus* were analyzed (De et al., 1995). A total of 169 strains isolated from diverse samples and studied for antibiotic resistance pattern. Ceftazidime, netilmicin, cefotaxime and norfloxacin showed maximum activity having percentage resistance ranging from 8.17% to 30.82%. All the strains were resistant to chloramphenicol and tetracycline. This study also revealed that antibiotic resistance could be transferred from *A. calcoaceticus* to *E. coli* K12F’ Lac’Nx’. The incidence of R-plasmids was 81.25%.

Yasodhara and Shyamala (1997) identified and characterized 100 nonfermenters. Among these 15 isolates belonging to *Acinetobacter* species, majority being isolated from urine and were resistant to commonly used antibiotics. Sisomycin was most effective antibiotic used.

In vitro susceptibility patterns for newer beta-lactamase - inhibiting antibiotics such as ampicillin-sulbactum (A/S) and amoxicillin -clavulanic acid (A/C) were
tested with 100 consecutive isolates of *Acinetobacter baumannii* obtained from various clinical samples (Pandey et al., 1998). The study recorded A/C MIC for 86% of the strains was more than 16/8 μg/ml, whereas as there was an A/S MIC of more than 16/8 μg/ml for only 38% of the strains. Although both above antibiotics should show similar activity theoretically, this study showed A/S had superior activity as compared to A/C against *A. baumannii*.

Goel et al (1998) reported the influence of iron on growth and extracellular products of *Acinetobacter baumannii*. *A. baumannii* growth was restricted in iron depleted medium (chemically defined medium CDM - Fe) when compared with iron containing medium. In CDM - Fe the bacterium is known to produce high molecular weight outer membrane proteins, which are designated as IROMPS, which were absent in CDM + Fe. They demonstrated that the supernatants of CDM - Fe having siderophores (catechol type) secreted by the organism extracellularly into the medium, which acts as iron chelators. Their conclusion was *A. baumannii* under iron restricted conditions express IROMPS along with production of catechol type siderophore in order to acquire iron from the external milieu.

One of the major outer membrane proteins OmpAb, 40 kDa from *A. baumannii* has been identified and purified and the role of this protein in the diffusion properties of the outer membrane and their importance was discussed (Jyothisri et al., 1999).
11.3 Biotyping of Acinetobacter species:

As mentioned earlier only in 1994, a detail study using recent rational
taxonomic developments was reported (Chopade et al., 1994 a&b). One hundred and
seventy six strains of Acinetobacter were isolated from various clinical sources.
Baumann's medium was modified and used for enrichment of Acinetobacter. Five
different genospecies of acinetobacters were isolated, and a majority of them showed
multiple resistant to the most of the antibiotics tested. A. baumannii was the most
predominant genospecies obtained followed by A. junii, Genospecies 3, Genospecies
6 and Genospecies 12 (Table-11.1). A total of 85.7% strains were belonging to A.
baumannii and further biotyping of A. baumannii revealed that biotype 7 was the
most prevalent followed by biotype 2. Overall 8 biotypes were obtained.

Gulati et al (1999) used the biotyping scheme of Bouvet and Grimont for
typing 100 isolates of Acinetobacter spp obtained from blood and CSF of sixty-one
patients admitted in the postoperative neurosurgery ICU. Among these patients, 40
had clinically diagnosed infections like bacteriemia or meningitis (Group A) while in
21 patients the isolation was regarded as contaminants (Group B). A. baumannii was
predominantly associated with clinical infections. The distribution of common species
in this study is given in the table 11.2.
In a most recent study Prashanth and Badrinath (2000) used simplified phenotypic scheme for identification of clinical isolates of *Acinetobacter* spp. Here only 7 carbon assimilation tests were used. During a 16-month period, 22 patients hospitalised mainly in Respiratory Intensive Care Unit (RICU), Paediatric and other medical wards were investigated either for infection or colonization by *Acinetobacter*. Forty-five isolates of *Acinetobacter* were further tested among the total number of 425 nonfermenters encountered. Twenty-four representative isolates from 45 *Acinetobacter* isolates were further selected for extended phenotypic identification. Four environmental isolates of the same were included in the study. Total of 28 isolates were typed using two methods, biotyping and antibiotyping, which helped in delineating *Acinetobacter* spp. into 12 phenotypes and two distinct antibiotypes respectively. A sudden increase of cases of a *Acinetobacter* infection suggested 3 outbreaks during the study period and was due to phenotypes 1 & 2 of Acb-complex. Strains of Acb-complex showed multiple drug resistance and were sensitive only to netilmicin. Comparatively high level of resistance to amikacin (48%) was also noted among these strains by agar dilution method. The ICU environment was recognised as an important reservoir for the

<table>
<thead>
<tr>
<th>No. of Patients</th>
<th><em>P. aeruginosa</em></th>
<th><em>K. pneumonia</em></th>
<th><em>A. calcoaceticus</em></th>
<th><em>A. baumannii</em></th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A (n=40)</td>
<td>33</td>
<td>4</td>
<td>2</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Group B (n=21)</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>6</td>
<td>5</td>
</tr>
</tbody>
</table>
resistant outbreak strain (Acb-1), which was probably leading to persistent colonisation and recurrent infections. This scheme was able to delineate all the isolates into 12 phenotypes (Table 11.3). However, the scheme was unable to speciate within Acb-complex.

11.4 The role of Acinetobacter in biodegradation:

As Acinetobacter is known for its versatility in utilizing numerous organic compounds, this character has been exploited for some biodegradation of waste. Few studies have been carried out in India regarding this aspect. Hanson et al. (1997) demonstrated that Acinetobacter spp. A3 was able to extensively degrade Bombay high crude oil and utilize it as the sole source of carbon. There was reduction in phytotoxicity of the crude oil owing to its degradation, which was evident when they witnessed the plant growth in soil containing this oil amended with Acinetobacter spp. A3.

Two types of Indian crude oil (Bombay High and Gujarat) were tested for their biodegradability by Acinetobacter calcoaceticus and Alcaligenes odorans. Acinetobacter showed high degradability (50% and 45%) of both oils when compared with Alcaligenes odorans (29% and 37%). This crude oil degradative capability of Acinetobacter species A3 could be exploited for bioremediation purposes.
Table 11. Distribution of different phenotypes among *Acinetobacter* strains from the study of Prashanth and Badrinath (2000).

<table>
<thead>
<tr>
<th>Phenotype number</th>
<th>Glucose</th>
<th>Gelatin</th>
<th>Haemolysis</th>
<th>Growth at 37°C</th>
<th>Growth at 44°C</th>
<th>L-Ascorbate</th>
<th>L-Histidine</th>
<th>Malonate</th>
<th>Histamine</th>
<th>Citrate</th>
<th>Penicillin</th>
<th>Chloramphenicol</th>
<th>DL-Aminoobutyrate</th>
<th>L-Phenylalanine</th>
<th>ICU</th>
<th>PAED</th>
<th>MED</th>
<th>ENV</th>
<th>PHENON</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (1)*</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>Acb</td>
</tr>
<tr>
<td>2 (2)</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>8</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>Acb</td>
<td></td>
</tr>
<tr>
<td>3 (3)</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>Acb</td>
<td></td>
</tr>
<tr>
<td>4 (4)</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>Acb</td>
<td></td>
</tr>
<tr>
<td>5 (5)</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>Acb</td>
<td></td>
</tr>
<tr>
<td>6 (-) NP</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>Acb</td>
<td></td>
</tr>
<tr>
<td>7 (7)</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>Acb</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 (25)</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>-</td>
<td>A. haeomolyticus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 (9)</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>n.t.A. haeomolyticus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 (13)</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>n.t.A. haeoffii</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11 (-) NP</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>n.t.A. haeoffii</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12 (-) NP</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>n.t.A. haeoffii</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* The number indicated in parenthesis are the phenotypic number assigned to different isolates in the Gerner-Smidt's study (1993).
* NP—New phenotype other than that of earlier studies, ICU—Intensive Care Unit, PAED—Paediatrics, MED—Medical, ENV—Environment.