Necrotising pneumonia due to Panton Valentine Leukocidin Staphylococcus aureus

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Introduction
Necrotising pneumonia, with destruction of lung tissue and formation of abscesses, can be associated with Gram-negative infections, such as Klebsiella (aspiration pneumonia in alcoholics) or Pseudomonas aeruginosa (neutropenics), fungal infections (e.g. Aspergillus in neutropenics) or Gram-positives (Group A beta-haemolytic streptococci and Staphylococcus aureus). The recent increase in cases due to Panton-Valentine Leukocidin (PVL) producing S. aureus prompted this review, since there are implications for routine laboratories when samples are received from such patients. PVL-related disease may present in association with meticillin sensitive S. aureus (MSSA) and meticillin resistant S. aureus (MRSA). The classical presentation of PVL-associated infection is recurrent skin and soft tissue abscesses. These are the result of the compensatory, chronic over-production of polymorphs in response to the leukocidal action of the PVL toxin. Although this is the most common presentation, many hospitals are now seeing fulminating and devastating pneumonia.

PVL-associated staphylococcal pneumonia produces cavitation, lung haemorrhage (which may be massive and exsanguinating), massive tissue necrosis, abscess formation and a mortality approaching 75%.

PVL production is not a new phenomenon. The “dreaded staph” used as a control in laboratories is PVL positive, and PVL probably accounted for some of the mortality in the 1919 influenza epidemic. “Old fashioned” PVL-negative S. aureus pneumonia has a mortality of some 2%, whereas PVL positive S. aureus pneumonia has a mortality of 62-75%. In 1919, in Fort Jackson, USA, when hundreds of troops were dying, clinicians commented, “the treatment of S. aureus infection of the lung is extremely ineffectual”. Nowadays, even appropriate antibiotics have a limited capacity to alter the outcome of severe infections.

PVL was first described in 1932 by Panton (see Figure 1) and Valentine in The London Hospital. PVL is now known to be encoded by two genes, luk-S-PV and luk-F-PV, and transferred between strains of S. aureus by bacteriophage. A heterogeneous mix of MSSA and MRSA strains carrying the genes for PVL production is spreading throughout Europe. Sporadic cases and small outbreaks have been reported in the UK, some spreading within hospitals, as has the emergence of new strains of PVL producing community-associated MRSA (CA-MRSA) now appearing in animals. Less than 2% of strains of S. aureus from invasive infections submitted to the Health Protection Agency (HPA) produced PVL.

A bi-component toxin, structurally similar to gamma-haemolysin, PVL comprises two sub-units (F & S) that together are leukocidal and dermonecrotic. Of S. aureus from invasive infections submitted to HPA, 1.6% of strains produced PVL, of which 46% were MRSA.

Figure 1. Dr Philip Panton
Since staphylococci spread between those living closely together with a lot of physical contact, spread of PVL-related disease is most common in families, nurseries, childcare centres, in closed establishments such as prisons, and where skin trauma encourages colonising staphylococci to adhere and then invade – e.g. turf burns, military training and athletic teams.

Community-associated or acquired MRSA (CA-MRSA) has been defined as an isolate obtained from an out-patient (or inpatient within 48 hours of hospitalisation) in the absence of identified risk factors for acquiring MRSA. Molecularly, CA-MRSA strains contain Staphylococcal Cassette Chromosome (SCC) mec element types such as IV and V (IV-VI normally lack antibiotic resistance genes other than meca, hence strains are relatively sensitive to antibiotics other than beta lactams. Most CA-MRSA carry genes encoding for PVL, as well as up to 18 additional virulence factors, including toxin producing genes, (sea, seb, see, seh and sek). CA-MRSA is characterized by enhanced spreadability, adherence to damaged skin and a propensity for necrosis, e.g. the painful necrotic lesions leading many patients in the USA to conclude, erroneously, they have been bitten by spiders.

PVL seems to enhance the spreadability of staphylococci expressing it, possibly by conferring better adherence to damaged skin. The most successful PVL-associated clones of MRSA are USA-300 & USA-400, with USA-300 more “fit” for survival. There is a clonal shift from USA-400 to USA-300 predominance, especially in institutional settings. Finally, CA-MRSA grows faster than hospital acquired MRSA (HA-MRSA).

Professor Jemma EIkeime, a world authority on PVL, estimated the incidence of PVL-associated staphylococcal pneumonia to be 1 case per 10 million persons per year, but the true incidence is unknown. A recent review of the world literature found only 141 cases2, which must be a massive underestimate. Few cases are published, molecular testing for the PVL genes is not routine in hospital laboratories and the disease is not notifiable. In Exeter alone, since 2009, we have seen 6 cases due to unrelated strains of PVL positive MSSA, and one CA-MRSA strain.

Pathogenesis of PVL-associated staphylococcal pneumonia

Bacteria enter the lungs via the bloodstream or into the airways during inhalation. Post influenza staphylococcal pneumonia is well recognized. PVL producing strains bind preferentially to basement membranes and exposed type IV collagen, colonising denuded epithelium and producing sheets of staphylococci.

Haematogenous spread from skin or soft tissue infection (SSTI), such as an infected injection site in an intravenous drug user, is a common cause of right sided endocarditis, when septic pulmonary embolisation results, shunting staphylococci into the lung vessels. There, staphylococcal multiplication and tissue production ensues in the thrombus. PVL and other toxins, particularly alpha-haemolysin, act together to produce necrosis in adjacent tissues. Once established, rapid bacterial multiplication produces sheets of staphylococci, and further production of other toxins ensues, particularly as the cells enter their phase, during the late exponential phase of growth, induction of alpha toxin expression begins, maximal production occurring during early stationary phase. Quorum sensing, whereby cell to cell communication between bacteria via auto-inducer molecules, allows bacterial populations to coordinate gene expression. Induced gene expression at high cell density affects cell development and virulence factor expression. This mechanism may be responsible for expression of protase and alpha toxins, which are particularly cytotoxic.

PVL binding activates the approaching neutrophils, opening membrane calcium channels and liberating interleukins and other inflammatory mediators, generating a cascade of local vassodilatation, chemotaxis and additional neutrophil infiltration. Secretion of degradative enzymes and generation of superoxide ions promote necrosis. As infection spreads throughout the lung tissue, inflammatory changes result in a necrotising vasculitis with massive areas of infarction and haemorrhage.

Clinical presentation and diagnosis of PVL-associated staphylococcal pneumonia

Relatively few patients developing necrotising pneumonia have a history of skin sepsis, themselves, but often have family members or close contacts with SSTI.

The pathognomonic presentation is that of a previously fit, young patient with a recent flu-like illness, a fever of >39°C, respiratory rate >30, tachycardia of >140 beats per minute, significant haemoptysis and hypotension1. Marked leucopenia and very high C-reactive protein levels (>300-400g/l) may be found, reflecting the gross tissue destruction, thrombosis and sepsis. Multilobar, alveolar infiltrates are usual, and, unlike HA-MRSA pneumonia, frequently cavitate with effusions1,2.

What is presumed to have been a “viral illness” with shivers and shakes may be due to a virus or indeed secondary staphylococcal infection and bacteremia. Together with an initially non-productive cough and normal chest X-ray, this misleading presentation leads the unfortunate clinician reviewing the patient to be falsely reassured by the lack of radiographic changes and the “normal” neutrophil count. Hemo, patients may be discharged with a non-specific “viral illness” diagnosis, and often only given inhalers or ineffective low doses of oral antibiotics. Clinicians do not realise the massive wide blood cell (WBC) expense resulting from the staphylococcal infection has already been decimated by PVL, so what would have been a WBC count of 20-30 may instead be in the normal range. Only hours later, as necrosis and consumption of platelets and neutrophils reaches a zenith, does the classical leucocytosis occur, and now septic shock alerts the clinician to the gravity of the situation.

The rapid, extensive pneumonia evolves into an acute respiratory distress syndrome with haemorrhage, which may be massive, and usually, a profound leucopenia. Pulmonary manifestations associated with PVL positive MRSA may be diverse, including classical interstitial pneumonia, pneumonia with pneumatoceles, empyema, septice emboli and abscesses1,5.
The first recognised UK case of PVL-associated pneumonia was in London in 2002, although France, America and Australia had noted cases since 1999. Not all PVL-associated pneumonia cases exhibit the classical characteristics; depending on the time since infection, patients will exhibit some or none of these symptoms, especially if the progress of infection has been affected by antibiotic therapy prior to admission.

**Laboratory investigations**

Leucopenia and thrombocytopenia are common, and a C-reactive protein, usually >300 units, would be expected. Although patients presenting in the early stages of disease may have a normal WBC count, there is a high C-reactive protein. The case study of a patient with PVL MSSA invasive disease, has occurred. However in Exeter, ten days secondary transmission, particularly within families and producing nose/throat/wounds of contacts may occasionally be necessary as used, since secondary cases are known to have occurred. Screening patients need intensive care and aggressive antibiotics. In addition only 38%2.

**Radiology investigation**

Early on, there may be nothing to find on examination or chest X-ray. Later, the chest X-ray shows multiple areas of patchy consolidation, monolobular or multilobular infiltrates developing into cystic changes and cavitation, best visualised with a CT or MRI scan. Pleural effusions and empyema are common.

**Treatment of PVL-associated staphylococcal pneumonia**

For many years, GPs have known to cover for secondary staphylococcal pneumonia after influenza. Traditionally, post-viral anti-staphylococcal prophylaxis for bacterial pneumonia with co-association, erythromycin or simply adding flucloxacillin to the local anti-staphylococcal prophylaxis for bacterial pneumonia with co-staphylococcal pneumonia after influenza. Traditionally, post-viral infection. Haemoptysis in the setting of severe pneumonia justifies urgent Gram staining, and typical sheets of Gram-positive cocci in clusters with a paucity of neutrophils are seen (see Figure 2).

**Detection of PVL in clinical samples**

**Figure 2.** Gram stain of sputum from a patient admitted with PVL pneumonia. Note the paucity of leucocytes and massive numbers of staphylococci.

**Which antibiotics should be used?**

Even for MSSA, the bactericidal antibiotic, flucloxacillin, may not be ideal, since nafcillin increases PVL production in vitro at concentrations just above the MIC. Nafcillin upregulates mRNA expression in both MRSA and MSSA, resulting in prolonged and increased toxin production. Sub-inhibitory levels of meticillin lead to increased alpha toxin levels via increased exo-protein synthesis and in vitro, low concentrations of nafcillin increase the concentration of PVL up to 3-fold.

Instead, since the necrotising process is primarily driven by toxin production, antimicrobials acting at the ribosomal level of protein synthesis seem preferable. Empirical therapy should block toxin production, penetrate dead and dying tissue, preferably be bactericidal and cover the possibility of CA-MRSA until sensitivity test results are available. Antibiotics suitable for SSTI treatment (co-trimoxazole, daptomycin) are not appropriate for fulminant pneumonia.

Unsuspected CA-MRSA infection in five Minnesotan children admitted to hospital in 1999 was the reason for failure of the empirical cephalosporin antibiotics. All four died. Predicting the sensitivity of staphylococci seen in clinical specimens is increasingly difficult, depending largely on the geographical location and clonality. Most USA-300 strains of CA-MRSA are resistant only to beta-lactams and macrolides. However, recently a strain resistant to tetracycline, fusidic acid, clindamycin and fluoroquinolones has been reported, as have strains with increasing vancomycin resistance. A PVL positive CA-MRSA strain (probably USA-300), causing necrotising pneumonia in a young Italian, had an MIC to vancomycin of 2-4. It responded to a combination of imipenem, tetracycline and rifampin and necessitated 6 weeks in hospital.

The prevalence of inducible clindamycin resistance shows striking differences between geographical areas. A report from Dallas showed a dramatic fall in resistance over 3 years, associated with a clinical shift. Laboratories should routinely “D test” all S. aureus isolates appearing to be erythromycin resistant and clindamycin sensitive to exclude inducible clindamycin resistance. Clindamycin resistance is particularly prevalent in CA-MRSA strains in Taiwan.
Proteomic studies showed that linezolid, one of a new class of antimicrobials, the oxazolidinones, reduced the tumour necrotising factor (TNF) inducing activity of S. aureus in a dose-dependent manner, reduced expression of virulence factors alpha and beta haemolysins, and decreased protein A. Clindamycin suppresses protein synthesis, including alpha and delta haemolysins, exotoxin production in vitro and exo-protein expression, including alpha toxin.

The combination of clindamycin and linezolid has proven synergistic in vitro and is our recommended initial therapy pending antibiotic sensitivity results. Combinations of anti-staphylococcal antimicrobials, such as rifampicin, fucidinaxic, and cephalothin, have also been used with varying degrees of success. In Exeter, we use 1-2-1 in IV intravenous clindamycin, 6-hourly, and linezolid 600mg, 12-hourly. Latterly, addition of a third antibiotic, rifampicin, coincided with significant improvement, and so now we would use all three initially, since the rifampicin is active against intracellular staphylococci.

Of the quinolones, in vitro studies show that moxifloxacin is far superior to ciprofloxacin in treating CA-MRSA strains, and of the newer antimicrobials, daptomycin, being inactivated in surfactant, is superior to ciprofloxacin in treating CA-MRSA strains, and of the newer antimicrobials, daptomycin, being inactivated in surfactant, is superior to ciprofloxacin in treating CA-MRSA strains. Combinations of anti-staphylococcal antimicrobials, such as rifampicin, fucidinaxic, and cephalothin, have also been used with varying degrees of success. In Exeter, we use 1-2-1 in IV intravenous clindamycin, 6-hourly, and linezolid 600mg, 12-hourly. Latterly, addition of a third antibiotic, rifampicin, coincided with significant improvement, and so now we would use all three initially, since the rifampicin is active against intracellular staphylococci.

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Adjuvant therapy for PVL-associated infections

(i) Intravenous immunoglobulin (IVIG)

We routinely add intravenous immunoglobulin, 2g/kg, for its beneficial immunomodulatory action on patients in toxic shock, as it neutralises superantigens and circulating toxins. This is repeated at 48 hours if there is still evidence of sepsis or failure to respond. IVIG neutralises PVL pore formation and the cytopathic effect of PVL in vitro, inhibition being concentration dependent.

(ii) Protein C

Whilst anecdotal reports suggest a possible role for activated Protein C, it is contraindicated in active bleeding, which may be occurring before frank haemoptysis develops.

Extra-corporeal membrane oxygenation (ECMO)

This process, where the lungs are bypassed and the blood is oxygenated outside the body, is available in only one or two centres in the UK. ECMO has been used in Sweden, the USA and, recently, in the UK for PVL pneumonia, although with few successes to date.

Future therapies

Inactivation of toxins that have already been produced and continue to drive the response may be the key to improving survival. Potential areas for research include re-directed phage therapy, and perhaps re-visiting old treatments such as leukocidin toxoid and phage therapy.

References

Erysipelothrix rhusiopathiae: forgotten but not gone

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**Erysipelothrix rhusiopathiae infection**

Erysipelothrix rhusiopathiae is a facultative, non-sporing, non-acid fast, small, Gram-positive bacillus. The name means literally ‘erysipelas thread of red disease’. The organism has had a long history and many nomenclature changes. The earliest member of the genus *Erysipelothrix* was termed *Erysipelothrix muriseptica* when first isolated by Koch in 1876 from the blood of mice with septicemia. In 1892, a bacillus was observed, in pigs dying of infection, by Pasteur and Dumas. Later, Loeffler reported a similar bacillus in the skin blood vessels of a pig that had died of swine erysipelas. This was the first description of this organism as an infectious agent causing disease in swine.

*E. rhusiopathiae* was first described as a cause of human disease in 1870, and further cases were documented in 1873 as erythema necroticans. *Erysipelothrix* was not established as a human pathogen until 1884, when Rosenbach isolated an organism from a patient with localised cutaneous lesions. He used the term ‘erysipeloid’ to differentiate between the human streptococcal disease, erysipelas, and the condition he had observed. Subsequently, *Erysipelothrix* has been identified as the cause of infection in many animal species. Rosenbach distinguished three separate species of the organism, *E. muriseptica*, *E. proci* and *E. erysipeloid*, based on their isolation from mice, pigs and humans, respectively. It was realised later that these three organisms were nearly identical strains of the same species. The name *E. insidiosa* was proposed for them originally by Trevisan in 1885. This and all 36 other documented names for the organism were rejected in 1896 in favour of *E. rhusiopathiae*, a combination that originated in 1876.

Three forms of human disease have been recognised, including a localised cutaneous lesion form, *erysipeloid*, which is so-called to distinguish it from the human streptococcal disease erysipelas, a generalised cutaneous form and a septicaemic form often associated with endocarditis. *E. rhusiopathiae* is ubiquitous and able to persist for a long period of time in the environment, including marine locations. It is a pathogen or a commensal in a wide variety of wild and domestic animals, birds and fish. Swine *erysipeloid* is the disease of greatest prevalence and economic importance. Diseases in other animals include *erysipeloid* of farmed turkeys, chickens, ducks and musk, and polyarthritis in sheep and lambs. The organism causes no known disease in fish but can survive for long periods of time on the mucoid exterior slime of fish. Infection due to *E. rhusiopathiae* in humans is occupationally related, principally occurring as a result of contact with contaminated animals, their products or waste, or soil. *Erysipelothrix* is the most common form of infection in humans. Some other names have been used to describe this infection, including whale finger, seal finger, spock finger, blubber finger, fish poisoning, fish handler’s disease, and pork finger. These reflect the occupational attributes of the disease. While it has been suggested that the incidence of human infection could be declining due to technological advances in animal industries, infection still occurs in specific environments. Additionally, infection by the organism is possibly under-diagnosed due to the resemblance of *Erysipelothrix* infections to other infections, and problems encountered in isolation and identification.

Diagnosis of *erysipeloid* can be difficult if not recognised clinically, as culture is lengthy and the organism resides deep in the skin. *E. tonsillarum* and other unnamed species were classified in the genus of *Erysipelothrix* 20 years ago. *E. tonsillarum* was considered morphologically and biochemically identical to *E. rhusiopathiae*. However, *E. tonsillarum* could ferment sucrose while *E. rhusiopathiae* could not. The pathogenicity of other *Erysipelothrix* species is not well understood. *E. tonsillarum* is frequently isolated from tonsils of healthy pigs, cattle and broiler chickens. It was reported that *E. tonsillarum* was avirulent for swine and mice. However, some studies have identified *E. tonsillarum* as a cause of endocarditis in dogs, indicating that some strains of *E. tonsillarum* may be canine pathogens.

Various virulence factors may be involved in the pathogenicity of *E. rhusiopathiae*. The presence of a hyaluronidase and neuraminidase has been recognised, and neuraminidase plays a significant role in bacterial attachment and subsequent invasion into host cells. The role of hyaluronidase in the disease process is understood, however, the capsule is an important virulence factor.

**Isolation and identification of *E. rhusiopathiae***

*E. rhusiopathiae* is most likely to be confused with other Gram-positive, non-sporing, rod shaped bacteria, such as members of the genera *Brochothrix*, *Corynebacterium*, *Lactobacillus*, *Listeria*, *Kurthia* and *Streptococcus*. Identification of *Erysipelothrix* species is based on Gram stain (see Figure 1), cultural morphology, motility, haemolytic characteristics and biochemical properties, particularly hydrogen sulphide production. *E. rhusiopathiae* can be distinguished from other Gram-positive bacteria by the following: non-motility, absence of β-haemolysis; inhibition of growth by...
Culture

E. rhusiopathiae is ubiquitous in nature, being found wherever nitrogenous substances decompose. E. rhusiopathiae and infections caused by this organism are worldwide in distribution and affect a wide variety of vertebrate and invertebrate species. Human disease can originate from an animal or environmental source.

Swine erysipelas caused by E. rhusiopathiae is the disease of greatest prevalence and economic importance. It is economically detrimental to the pig industries of North America, Europe, Asia and Australia. Polyarthritis of sheep and lambs and erysipelas in calves, ducks and domestic turkeys are also economically significant diseases caused by E. rhusiopathiae. In Australia, E. rhusiopathiae causes polyarthritis in pigs, and it is an emerging problem in farmed emus.

The domestic pig is the most important reservoir of E. rhusiopathiae. It is estimated that 30-50% of healthy swine harbour the organism in their tonsils and other lymphoid tissues. Carriers can discharge the organism in their bacons, urine, saliva and nasal secretions, creating an important source of infection. Soil, bedding, food and water can be contaminated by infected pigs, leading to the indirect transmission of the organism. Over 30 species of wild birds and at least 50 species of wild mammals are known to harbor E. rhusiopathiae, providing an extensive reservoir.

The organism can survive for long periods in marine environments. The slime on fish appears to be an important source of infection for man. The organism has been isolated from the environment, but this may be secondary in importance to animal reservoirs as a source of E. rhusiopathiae. Although E. rhusiopathiae is killed by moist heat at 55°C for 15 min, it is resistant to many food preservation methods, such as salting, pickling and smoking.

E. rhusiopathiae infection in humans is occupationally related. It occurs mostly in those people whose jobs are closely related with contaminated animals, their products or wastes, or soil. People with the highest risk of exposure include butchers, abattoir workers, veterinarians, farmers, fishermen, fishmongers and housewives. The infection has also been associated with a wide variety of occupations, including meat cutters, meat-processing workers,
Although antibiotic susceptibility data for Erysipelothrix are still concentrated, penicillin and ceftriaxone, with low minimum inhibitory concentrations of 0.03 mg/l and 0.125 mg/l, respectively, remained the antibiotics of choice for Erysipelothrix endocarditis. A more recent study showed that penicillin and ceftriaxone are active against Erysipelothrix and it was suggested that they should continue to be recommended for treatment.

Oral penicillin will resolve a case of erysipelas in around 48h, but intravenous penicillin is recommended for more serious Erysipelothrix infections. Although the mortality rate for endocarditis has been reduced from the 100% seen in the pre-antibiotic era, it is still 38% for Erysipelothrix endocarditis in spite of available treatment. This rate may partly be explained by the use of vancomycin (to which the organism is resistant) in the empirical therapy of endocarditis. No recent data are available on mortality in humans. Early diagnosis of all forms of Erysipelothrix infection is therefore essential. In those allergic to penicillin, cephalosporins have been described as the most appropriate alternative, since clindamycin and erythromycin are only bacteriostatic for Erysipelothrix.

Containment and control of Erysipelothrix are effective in preventing the spread of infection in humans and animals. For individuals working in at-risk occupations, an awareness of the infection is essential. Suggested preventative measures include the wearing of gloves or other protective handwear, good hygiene, especially frequent hand washing with disinfectant soap and the use of antiseptic hand sanitizer. Alcoholism, may predispose to more serious forms of infection. The removal or regular disinfecting of contaminated sources is an important method of limiting the spread of the organism throughout a work environment. Erysipelothrix can be killed by commonly available disinfectants. A recent study found that several commercially available home disinfectants were effective in killing Erysipelothrix. Many investigators have noted, however, that structurally complex equipment is difficult to clean and, because the organism is able to survive in organic matter, disinfecting without cleaning is useless. If disinfection is impractical, other control measures become more important.

Control of reservoir populations of Erysipelothrix is impractical or impossible because of the widespread distribution of the organism, the large variety of animal hosts and its ability to persist in the environment.

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**Table 2. Distribution of MICs for 63 E. rhusiopathiae strains**

<table>
<thead>
<tr>
<th>Antimicrobial agent</th>
<th>No. of strains with the following MIC (µg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.03 0.05 0.1 0.2 0.39 0.78 1.56 3.13 6.25 12.5 25 50 100 &gt;100</td>
</tr>
<tr>
<td>Penicillin G</td>
<td>8 28 27</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>11 23 29</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>11 26 26</td>
</tr>
<tr>
<td>Oxacillin</td>
<td>1 4 38 20</td>
</tr>
<tr>
<td>Chloramphenicol</td>
<td>38 21 2</td>
</tr>
<tr>
<td>Dihydrostreptomycin</td>
<td>1 13 44 1</td>
</tr>
<tr>
<td>Kanamycin</td>
<td>63</td>
</tr>
<tr>
<td>Sulfadimethoxine</td>
<td>63</td>
</tr>
</tbody>
</table>

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Control of animal disease by sound husbandry, herd management, good sanitation and immunisation procedures is recommended. Erysipelas infection causes substantial economic losses in animal industries. Appropriate prophylactic measures are taken for the treatment and control of this disease. Antibiotic therapy is effective, and penicillin is usually the drug of choice. Vaccination is considered a useful procedure for controlling the problem in animal farms. Most commercially available vaccines are attenuated, live E. rhusiopathiae strains or bacterins. These vaccines offered protection to pigs and turkeys. The duration of immunity of these vaccines varies between 6 and 12 months, and the efficacy is variable, depending on the strains used and the use of the appropriate vaccine in different species of animals. Vaccination of humans is not considered to be a viable option because clinical erysipelas appears to convey little or no immunity as shown by relapse of infection.

References

7. Takahashi, T., Fujisawa, T., Yamada, Y. et al. (1985) Erysipelothrix rhusiopathiae isolation and use of the appropriate vaccine in different species of animals. Vaccination of humans is not considered to be a viable option because clinical erysipeloid appears to convey little or no immunity as shown by relapse of infection.

Eric Bridson Bows Out

Dr Eric Bridson has stepped down from the Editorial Board of Culture after 27 years service. At the time of the inception of Culture in 1978, Eric was Technical Director of Oxoid Limited, part of the Brooke Bond Group. The publication was the brainchild of Eric and George Fleming (then Marketing Director of Oxoid Ltd), conceived as a way for Oxoid to provide a service to microbiology by publishing articles covering a wide and eclectic range of topics from within the field of microbiology. Topics covered have been as diverse as from Antarctic micro-organisms: coming in from the cold to Mycobacteria and HIV – a deadly combination from Emporiatrics and jet fighter to the rise of microbial slimers in biodeterioration.

Eric retired from the Oxoid Board of Directors in 1989 but maintained his position on the Culture Editorial Board until November 2007, when he finally decided to step down. A presentation was made to mark the occasion.

The Culture Editorial Board and all of us at Oxoid would like to express our most sincere gratitude for all that Eric has contributed to both Oxoid and Culture. We send him our very best wishes for the future.

Ali Ball
Vice President, Marketing & New Product Development

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Our thanks go to the Culture editorial board: Professor Grahame W Gould, Dr David Petts, Mr David E Post and Dr Peter Stephens.

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