specimens showing chronic atrophic gastritis. Further studies to investigate this relationship in more detail are required. □

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Molecular conservation within LES9F and PS21 Liverpool epidemic strain (LES) markers in wild-type clinical *Pseudomonas aeruginosa* isolated from the sputum of adult patients with cystic fibrosis

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The most common complication of cystic fibrosis (CF) is the recurrence of chronic chest infections usually caused by bacterial pathogens. Cystic fibrosis patients continue to suffer from recurrent and chronic respiratory tract infections and most of their morbidity and mortality is due to such infections, which are usually dominated by Gram-negative organisms, especially *Pseudomonas aeruginosa*, as well as members of the *Burkholderia cepacia* complex, *Stenotrophomonas maltophilia* and several emerging Gram-negative organisms.[1] One main clinical aim in their management is the prevention of acquisition and subsequent progression of bacterial respiratory pathogens to the chronic stage, as such events lead to a declining cascade of infection and inflammation, resulting in decreasing lung function and premature death.

Recently, several reports have described the emergence of the Liverpool epidemic strain (LES) of *P. aeruginosa* in CF patients.[2] This LES strain has been reported to be the most frequently isolated clone obtained from CF patients in England and Wales.[3] This epidemic strain has also been reported to cause superinfection[4] and is associated with greater morbidity in patients than seen with other non-LES *P. aeruginosa* strains.[5] In addition, it has been demonstrated to be highly transmissible from a CF patient to non-CF parents,[4] while there has also been a recent report of transmission from a CF patient to a cat,[6] which resulted in increased morbidity for recipients of LES *P. aeruginosa*.

As a result of this, attention has been directed to the development of reliable molecular diagnostic assays to differentiate LES *P. aeruginosa* from non-LES *P. aeruginosa* and several methods have been published, including the LES9F and PS21 markers as target amplicons for the presence of LES.[7] The recent publication of the entire genome of an LES-positive strain (*P. aeruginosa* LESB58; GenBank accession number: FM209186) has allowed comparison of clinical wild-type isolates to LESB58.

However, what remains unclear is the amount of variation that exists within these target LES amplicons and how conserved they are in wild-type *P. aeruginosa* isolates from CF patients, as presently there is only one gene sequence of these loci in GenBank (submitted in early January 2009). Hence, this short sequencing study aims to estimate the degree of genetic hypervariability that exists within wild-type LES *P. aeruginosa* organisms obtained from the sputum of adult CF patients and LESB58.

Examples of LES *P. aeruginosa* were isolated from the sputum of six randomly selected adult patients attending the Northern Ireland Regional Adult Cystic Fibrosis Centre, Belfast City Hospital, who were known to be LES-positive patients. The LES status was checked through the development of a multiplex assay of three targets (PS21,[7] LES9F[7] and FpvAIII pyoverdine[8]). Only *P. aeruginosa* isolates that were concurrently positive for all three amplicons were selected for subsequent dideoxy sequencing, as described previously.[9] Within each target, resulting sequences from all isolates were aligned with each other, as well as with LESB58 (Table 1). From the current study, a representative sequence of LES PS21 and LES 9F has been submitted to GenBank with the respective accession numbers FJ710791 and FJ710792. Sequencing results from the *P. aeruginosa* isolates were totally conserved within the entire PS21 amplicon, as well as having total similarity with this region in LESB58, despite several mutations being detected within the FpvAIII pyoverdine with these isolates (data not shown). In addition, all the isolates

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Table 1. Comparison of LES PS21 and LES 9F in wild-type *Pseudomonas aeruginosa* isolated from CF patients and the reference strain LESB58 (GenBank Accession No: FM209186).

Target	Submitted GenBank Accession Number	Closest BLAST sequence match	Similarity	Position (in relation to FM209186)
LES PS21	FJ710791	*Pseudomonas aeruginosa* FM209186; PLES_26321	100%	np 2833095–2832811
LES 9F	FJ710792	*Pseudomonas aeruginosa* FM209186; PLES_23591	99%	np 2524896–2524524

demonstrated a single nucleotide deletion of an adenine base at position 2524879 (FM209186) of the LES 9F gene locus.

This small study demonstrates that LES PS21 and LES 9F are highly conserved in the wild-type CF *P. aeruginosa* isolates examined, even in the presence of several mutations in the pyoverdine gene locus. BLAST analysis of these sequences demonstrates the uniqueness of these sequences in nature, whereby only one match was obtained (GenBank accession number: FM209186). At this stage, the significance of this is unclear, but it is important to be able to note variation within these amplicons and possible variations in clinical disease states. Further analysis is now required of isolates from around the world, in order to examine potential geographical diversity. □

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PRF1 gene mutation in a Saudi patient with haemophagocytic lymphohistiocytosis

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Haemophagocytic lymphohistiocytosis (HLH) is a rare autosomal inherited disease associated with activated macrophages that engulf erythrocytes, leucocytes, platelets and their precursor cells in bone marrow, lymph node, spleen and other tissues.[1,2] Patients with HLH manifest cellular immunological dysfunction of regulatory pathways that normally terminate in effector immune responses.[3]

Hereditary and sporadic cases of HLH have been reported mainly in children,[5] although the condition can affect other age groups.[6–9] The incidence of HLH is estimated to be 1.2 per 1,000,000.[4-6] Clinical manifestations include decreased fetal activity, neonatal hypotonia, neonatal feeding difficulties, hyperphagia with obesity, hypogonadism, short stature, small hands and feet, characteristic facial features, and mild to moderate mental retardation.

Haemophagocytic lymphohistiocytosis may be familial, or associated with a number of different infections, autoimmune disorders, or may occur together with malignancy.

The case present here is of an 18-month-old boy born at 34 weeks' gestation to a young couple (first cousins). Written informed consent was obtained from the parents of the patient for publication of this case report. The pregnancy suffered premature rupture of the membrane and neonatal polycythemia that required partial exchange of blood.

At the age of seven weeks the child was admitted to the paediatric intensive care unit with suspected septic shock syndrome. All microbiological tests were negative. Physical examination revealed hepatosplenomegaly and blood tests showed neutropenia (absolute neutrophil count: 400 cells/μL), thrombocytopenia (platelet count: 23,000/μL) and anaemia (haemoglobin [Hb]: 55 g/L). The patient recovered and his blood counts showed partial improvement.

At the age of three months he was admitted to the paediatric ward with fever and low blood counts that required frequent blood and platelet transfusion. Bone

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