postulated that such differences may be attributed to social behaviour and food hygiene practices in the two populations and/or the occurrence of less virulent *Campylobacter* strains and subspecies types in the Northern Ireland (NI) populations than in Britain.

The most recent and unpublished phagetyping data available in Northern Ireland is from 2002, which demonstrated the presence of 29 phagetypes among 219 faecal *Campylobacter* isolates, with 14/219 (6.4%) isolates being non-typable (Table 1). When compared with phagetypes from Wales,[5] Northern Ireland isolates shared similarities with the Welsh isolates. The most common phagetype in both regions was phagetype 1, with the second most common being phagetype 2 in both regions. The third and fourth most common phagetypes in Northern Ireland were 5 and 33, respectively, which were inverted in Wales. The four most common phagetypes in Northern Ireland accounted for 60.2% of total isolates examined, whereas in Wales this value was lower at 42.3% of total isolates examined. However, it must be borne in mind that these two data sets were not matched contemporaneously, as there were no data available to match over a similar period of time.

Overall, this comparison of a limited number of Northern Ireland isolates with a larger proportion of isolates from Wales demonstrates that there are no marked differences in the frequency of phagetypes of clinically significant campylobacters found in Northern Ireland and Wales, when employing the Colindale phagetyping scheme as an epidemiological marker. This would suggest that phenotypic differences in isolates do not account for the differences in attack rates between England and Wales and Northern Ireland and that the latter shares a similar pool of phenotypes with England and Wales.

Previously, LaFong and Bamford[6] suggested that differences in attack rates between Britain and Northern Ireland may be due to (a) relatively low consumption of unpasteurised milk in Northern Ireland compared to Britain, (b) a higher ratio of red meat to white meat consumption in Northern Ireland, (c) climatic factors and (d) a social likeness for food to be 'well done' in Northern Ireland.

The data presented here would support the hypothesis that such differences are probably due to social aspects of human behaviour (e.g., food preparation), as well as the four points suggested by LaFong and Bamford,[6] rather than to any differences in isolate type.

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Do equine strains of *Pseudomonas aeruginosa* carry the Liverpool epidemic strain markers relevant to patients with cystic fibrosis?

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Recently, in the UK, there have been several reports describing the emergence of the Liverpool epidemic strain (LES) of *Pseudomonas aeruginosa*, isolated from the sputum of cystic fibrosis (CF) patients.[1,2] What remains unclear is whether or not strains of *P. aeruginosa* carrying the LES genetic markers[3] are important in terms of their virulence and/or transmissibility, or are these markers purely of epidemiological interest?

Thus, it is important to have a full understanding of the epidemiology of where such epidemic strains originate, their persistence and transmissibility, particularly in relation to their risk to patients with CF. In order to address this and help to determine whether LES strains exist in *P. aeruginosa* solely within the CF community and not outside CF, we examined the presence of LES in a population of clinically significant *P. aeruginosa* isolates originating in infections from a comprehensive population of *P. aeruginosa*-infected horses.

P. aeruginosa isolates (n=86) were obtained from the bacteriological culture archive of the Irish Equine Centre, Johnstown, Naas, Co. Kildare, Ireland, over a five-year period (2003–2007). These strains were originally isolated from sites in infected and symptomatic equines, including abscess, ear, eye, faeces, genitourinary, guttural pouch, lung, nose, pharynx, semen, skin scrapings, throat, tissue, urine

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and wound, in 2007 (n=20 isolates), 2006 (n=20 isolates), 2005 (n=20 isolates), 2004 (n=20 isolates) and 2003 (n=6 isolates).

All isolates were revived from frozen storage and were identified phenotypically using a combination of conventional identification methods (e.g., oxidase) as well as the API identification scheme (API 20NE; bioMérieux, Les Halles, France). The presence of LES markers (LES-PS21 and LESF9) was determined by molecular methods, as recently described.[3]

Results indicated that none of the equine isolates were LES strains, as defined by Fothergill *et al*.[3] Recently, there have been several reports describing the emergence of the LES of *P. aeruginosa* in CF patients.[1] This LES strain has been reported as the most frequently isolated clone obtained from CF patients in England and Wales.[2] This epidemic strain has also been reported to cause superinfection[4] and is associated with greater morbidity in patients than is the case with other non-LES *P. aeruginosa* strains.[5] In addition, it has been shown to be highly transmissible from a CF patient to non-CF parents,[4] and from a CF patient to a cat,[6] which resulted in increased morbidity for recipients of LES PA in both reports. Hence, it is a priority to define the carriage of these markers in *P. aeruginosa*, both within and outside the CF community.

This study was not able to demonstrate the presence of LES strains in a comprehensive collection of clinically significant *P. aeruginosa* from a non-CF source, suggesting that LES is a CF-related phenomenon. However, further studies are required to determine the carriage of LES markers in non-CF-associated *P. aeruginosa* in other clinical and non-clinical isolates in order to determine the uniqueness of LES within the CF population.

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Molecular characterisation and diagnosis of Hb J-Taichung (129[H7]Ala→Asp) in a Taiwanese family subject

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Thalassaemia and haemoglobin variants are the most common genetic disorders in Taiwan. The frequency of α-thalassaemia is 3–5% and that of β-thalassaemia is 1–3% in the Taiwanese population. Haemoglobin (Hb) J-Taichung is a rare β-chain variant first describe in a Chinese female by Blackwell *et al*.[1] in 1969, and not found in other ethnic populations.

Hb J-Taichung is a non-pathological (stable) β-chain variant characterised by mutation at codon 129 of the β-globin gene exon 3 that changes alanine (GCC) to aspartic acid (GAC). Several other Hb variants depend on mutations at this region and include Hb Crete (β129 [H7] Ala→Pro) and Hb La Desirade (β129 [H7] Ala→Val). However, these mutations would not create a *Tse*I site.

In the present case study the proband was a 14-year-old Taiwanese girl admitted to the Children's Medical Centre, China Medical University Hospital, Taichung, following a four-month history of pallor. Informed consent to all investigations was obtained.

Peripheral blood samples, anticoagulated with

Table 1. Results of haematological indices and serum iron-related tests.

Parameter	Father	Mother	Brother	Proband
RBC (x10^6/μL)	4.98	4.70	5.82	4.87
Hb (g/dL)	16.1	12.9	15.6	10.0
Hct (%)	47.6	39.8	47.2	32.3
RDW	13.0	14.0	14.5	19.3
MCV (fL)	95.6	84.7	81.1	66.7
MCH (pg)	32.3	27.4	26.8	19.1
MCHC (g/dL)	33.8	32.4	33.1	30.7
HbA (%)	97.3	64.8	65.5	75.0
HbA2 (%)	2.7	3.3	3.3	3.5
HbF (%)	<0.1	<0.1	<0.1	<0.1
HbX (%)	–	31.8	31.1	21.4
Transferrin (mg/dL)	239.6	290.5	232.8	293.2
TIBC (μg/dL)	342.6	415.4	332.9	419.3
Fe (μg/dL)	82	80	143	19
Ferritin (ng/mL)	120.2	135.1	210.6	5.9

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