Table 1 Appropriateness and free-text feedback

Was the questionnaire appropriate? (n)	Free-text comments made (n)	Comments (n with similar theme)
Yes (83)	16	Positive comments regarding the questionnaire, eg good questions, simple to complete, comprehensive (6). Given too early, not started treatment yet (5). Response options in communication and information issues not clear (2). Good that I did not have to answer questions I did not want to. Difficult to complete because of small print. Specific questions considered inappropriate; sexual function (spouse in nursing home); religious needs (atheist). Unspecified questions considered potentially upsetting to patient by spouse completing questionnaire.
Left blank (14)	3	Not started chemotherapy yet which may alter the response (2). I may have different answers on different days.
No (3)	1	Not had any treatment yet, or know the extent of my illness.

use in patients with a recent diagnosis of thoracic cancer, before adopting it locally, we have surveyed its acceptability.

Patients within 4 weeks of their diagnosis of thoracic cancer attending outpatient clinics were identified. After obtaining verbal consent, patients were asked to complete the SPARC questionnaire, if necessary with the help of a carer or the member of staff in attendance, with the instruction to leave any questions they were unsure of blank. On completion, they were asked to record on a separate feedback form if they felt the questionnaire was appropriate, had any comments about the questions asked or had any other comments. The results were collated anonymously and analysed using descriptive statistics, with comments relating to the questionnaire grouped into themes. The survey was registered with the Trust Governance and Health Audit department (no. 775).

Of those approached, 86% agreed to take part, with data from 100 patients analysed (63 male; mean (SD) age 68 (9) years; non-small cell lung cancer 70, small cell lung cancer 20, mesothelioma 10; all Caucasian, with English as the first language in 98). Questionnaires were completed by the patient alone, or with the aid of a member of staff or carer in 65, 22 and 8 instances, respectively. Of the maximum 56 responses, the median (IQR) number completed was 52 (47–54). The questionnaire was considered appropriate by 83 patients, not appropriate by 3, and 14 did not answer this question. Of 22 comments made, only one related to some questions being potentially upsetting (table 1). Patients had a median (IQR) of 2 (0–5), 4 (2–7) and 8 (5–12) symptoms or issues which distressed or bothered them 'very much', 'quite a bit' and 'a little', respectively. Most common were feeling tired, shortness of breath and problems sleeping at night.

Our results suggest that the SPARC questionnaire is acceptable to the majority of patients with a recent diagnosis of thoracic cancer. A clear explanation of the purpose of the questionnaire, the instruction to leave a question blank when unsure and the offer of help to complete it, taken up by about a fifth of patients, may all have contributed to its acceptability. On the basis of our results, we have introduced the SPARC into routine practice, having increased the text size to improve readability.

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An outbreak of H1N1 influenza in a respiratory unit

Pandemic influenza A (H1N1) is a major global public health concern.[1] We report an outbreak of H1N1 influenza involving clinical staff and patients in a teaching hospital in the North East of Scotland.

In October 2009, a teenage patient was admitted with an exacerbation of asthma. As there was no reason to suspect H1N1, the patient was not isolated. However, nose and throat swabs were in fact taken on the day of admission and were positive for H1N1 by PCR within 24 h. At this point, source isolation precautions were instituted. Seven otherwise healthy clinical staff in our department (6 doctors and 1 nurse; mean age 29, three males) subsequently developed typical symptoms within the next 9 days. Cases were confirmed by viral PCR from pooled nasal and throat samples. As H1N1 was common in the community at this time, it was not possible to determine whether the patient was the source of infection; however, five of the affected clinical staff had been in direct contact with the patient and the first symptoms reported by them were 48 h following his admission to the ward.

Affected staff were advised to remain off work for a week after the initial onset of their symptoms; 33 working days were subsequently lost due to illness. The cumulative number of staff members infected with H1N1 virus in our unit over a week from the time of first symptoms reported by a member of staff is shown (figure 1). During the same period, six patients on the ward also tested positive for H1N1 after developing flu-like symptoms. In two of these, H1N1 was likely to have been contracted while in hospital as they had been admitted several days earlier and were recovering from their presenting illnesses.

Our experience has demonstrated that within the hospital environment, H1N1 is readily and rapidly transmissible between individuals. This outbreak highlights the importance of rapidly identifying infected patients and instituting source isolation procedures. Moreover, it is imperative that members of hospital staff use appropriate protection equipment and receive H1N1 immunisation (as recommended by the Department of Health) at the earliest opportunity. This should reduce the risk not only of healthcare workers contracting the virus from infected patients, but also of cross-infection between other patients and other healthcare workers. It is important that hospital trusts recognise the potential impact of H1N1 on frontline staffing and ensure appropriate contingency plans are made.

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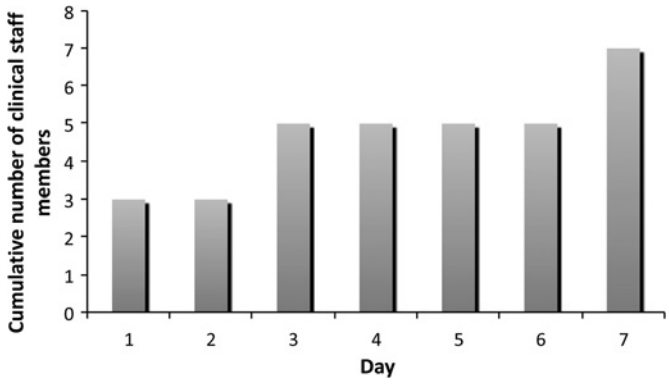

Figure 1 Cumulative number of clinical staff in our unit with serological H1N1 influenza over a 7-day period.

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First independent replication study confirms the strong genetic association of *ANXA11* with sarcoidosis

Sarcoidosis is an inflammatory disease characterised by the presence of granulomas that can affect the skin, lungs, heart, brain and nervous system, eyes and various other tissues and organs.[1] The disease can present in an acute or subacute form and is often self-limiting, but in many cases it is chronic with variable disease activity over many years.[2] A genetic association between sarcoidosis and a truncating splice-site mutation in the gene *BTNL2* (butyrophilin-like 2, a member of the immunoglobulin gene family) has been confirmed in different studies and populations.[3–5]

Very recently the first whole-genome association study (WGAS) reported a strong association between sarcoidosis and *ANXA11* (annexin A11) on chromosome 10q22.3, a member of the annexin family of calcium-dependent phospholipid-binding proteins involved in structural organisation of the cell, growth control, calcium signalling, cell division, vesicle trafficking and apoptosis.[6 7] Different single nucleotide polymorphisms (SNPs) within and downstream of the *ANXA11* gene were strongly associated with sarcoidosis, including one non-synonymous SNP rs1049550 (c.688T→C, p.C230R) and several intronic or intergenic SNPs (rs1953600, rs2573346, rs2784773, rs2789679). The associated SNPs were in strong linkage disequilibrium.

To replicate the association in an independent cohort, we performed a case–control association study in 325 patients (mean age 52.1 years) and 364 healthy matched controls (healthy white German subjects, mean age 49.7 years). The diagnosis of sarcoidosis was based on evidence of non-caseating epitheloid cell granuloma in biopsy specimens and chest radiographic abnormalities. A chronic course was defined as disease over at least 2 years or at least two episodes in a lifetime. Acute sarcoidosis was defined as one episode of acute sarcoidosis which had totally resolved at the date of the examination. None of the individuals in the control group had a history of lung disease or showed any symptoms of lung or other disease by chest radiography or laboratory blood tests.

The C allele frequency of rs1049550 was significantly increased in the patients with sarcoidosis (C=0.6504, T=0.3496 in cases; C=0.5479, T=0.4521 in controls; p=0.00014, table 1). It was significantly associated with an increased risk of sarcoidosis in the individuals carrying the CC genotype (OR 2.18, 95% CI 1.39 to 3.43; p=0.00065). The increased risk was present in both dominant and recessive models (p=0.017 and p=0.0004, respectively). The allelic and genotypic risk of rs2573346 with sarcoidosis was even stronger, as described previously (allelic p=0.00008 and genotypic p=0.00022). The calculated population attributable risk (PAR) for rs2573346 CC homozygotes and CT heterozygotes was 21%. These data independently confirm the strong association between variations in *ANXA11* and sarcoidosis and support the hypothesis that ANXA11 represents a strong genetic risk factor for sarcoidosis.

In contrast to the previously reported difference in association to *BTNL2* in our patient cohort,[5] there was no statistical difference between acute and chronic forms of sarcoidosis and rs1049550 or rs2573346 alleles, and both groups showed a significant allelic and genotypic association for both SNPs (table 1). However, this effect was slightly more pronounced in the chronic form, which was the larger subgroup with higher statistical power. All p values obtained in our study withstand a conservative Bonferroni correction for multiple testing.

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Table 1 Statistical analysis of the case–control study

SNPs			Controls	Cases	OR(95% CI)	p Value	Acute*	p Value	Chronic*	p Value
rs1049550	Allele	T	283 (45%)	244 (35%)	1		82 (35%)		119 (34%)	
		C	343 (55%)	454 (65%)	**1.54 (1.23 to 1.92)**	**0.00014**	152 (65%)	**0.0073**	233 (66%)	**0.0005**
	Genotype	TT	65 (21%)	48 (14%)	1		18 (16%)		24 (14%)	
		CT	153 (49%)	148 (42%)	1.31 (0.85 to 2.03)	0.2245	46 (39%)		71 (40%)	
		CC	95 (30%)	153 (44%)	**2.18 (1.39 to 3.43)**	**0.00065**	53 (45%)	**0.0143**	81 (46%)	**0.0018**
rs2573346	Allele	T	303 (47%)	260 (37%)	1		86 (36%)		129 (35%)	
		C	341 (53%)	452 (63%)	**1.55 (1.24 to 1.92)**	**0.00008**	150 (64%)	**0.0050**	239 (65%)	**0.00074**
	Genotype	TT	71 (22%)	50 (14%)	1		18 (16%)		26 (15%)	
		CT	161 (50%)	160 (45%)	1.41 (0.93 to 2.15)	0.1097	50 (42%)		77 (43%)	
		CC	90 (28%)	146 (41%)	**2.30 (1.47 to 3.60)**	**0.00022**	50 (42%)	**0.0131**	76 (42%)	**0.0027**

Significant associations are shown in bold.
*Only patients with unequivocal classification were included.

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