Increase in Endemic *Neisseria meningitidis* Capsular Group W Sequence Type 11 Complex Associated With Severe Invasive Disease in England and Wales

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**Background.** In England and Wales, the incidence of invasive meningococcal disease has been declining for more than a decade, but meningococcal group W (MenW) cases have been increasing since 2009.

**Methods.** Public Health England conducts enhanced national surveillance of invasive meningococcal disease in England and Wales. Detailed clinical information was obtained for all laboratory-confirmed MenW cases diagnosed during 3 epidemiologic years (2010–2011 to 2012–2013), alongside whole-genome sequencing analysis of the clinical isolates.

**Results.** The year-on-year increase in invasive MenW disease across all age groups since 2009–2010 was due to rapid endemic expansion of a single clone belonging to the sequence type 11 complex (cc11). In 2013–2014, MenW was responsible for 15% of all invasive meningococcal disease. All but 1 of the recent MenW:cc11 isolates were very closely related, consistent with recent clonal expansion. Clinical follow-up of all 129 MenW cases diagnosed during 2010–2011 to 2012–2013 revealed that most patients were previously healthy (n = 105 [81%]), had not travelled abroad prior to illness and the majority presented with septicemia (n = 63 [49%]), meningitis (n = 16 [12%]) or both (n = 21 [16%]); however, one-quarter had atypical presentations including pneumonia (n = 15 [12%]), septic arthritis (n = 9 [7%]), and epiglottitis/supraglottitis (n = 5 [4%]). Forty-eight (37%) required intensive care and 15 (12%) died. There was no association between infecting strain, clinical disease, or outcome.

**Conclusions.** The recent increase in invasive MenW disease in England and Wales is due to rapid endemic expansion of a single clone belonging to cc11 and is associated with severe disease with unusual clinical presentations. This increase will require careful monitoring in the coming years.

**Keywords.** meningococcal W disease; meningococcal pneumonia; ST-11 clonal complex; travel; epidemiology.

*Neisseria meningitidis* is a major infectious cause of morbidity and mortality worldwide [1]. In the United Kingdom, routine vaccination against meningococcal capsular group C (MenC) since 1999 has resulted in capsular group B (MenB) being responsible for >85% of invasive meningococcal disease (IMD) cases across all age groups [2]. The meningococcal quadrivalent conjugate vaccine (MenACWY) is only used for high-risk individuals and travelers to endemic regions and for controlling outbreaks. Recently, much attention has been focused on MenB prevention following the licensure of a novel, protein-based, multicomponent vaccine in Europe [3]. At present, however, England and Wales are experiencing an upsurge in invasive capsular group W (MenW) disease. Historically, MenW incidence has been low, accounting for only 1%–2% of IMD cases annually. An increase during 2000–2002 was associated with travel to the Hajj [4], but following mandatory meningococcal vaccination for pilgrims, MenW cases...
declined to pre-2000 levels. Since 2009–2010, however, MenW cases have increased year-on-year. In response to this increase, we followed up all laboratory-confirmed MenW cases diagnosed over 3 epidemiological years and investigated the genomic profile of clinical MenW isolates over the same time period by using publicly available whole-genome sequencing data. This study describes the epidemiology of invasive MenW disease in England and Wales since 2000–2001, clinical characteristics of case-patients with laboratory-confirmed MenW disease during 2010–2011 to 2012–2013, and the molecular characteristics of clinical MenW isolates. In particular, the study aimed to identify differences in risk factors, clinical characteristics, or outcomes of IMD caused by the emerging MenW strain compared with previously circulating strains.

**METHODS**

Public Health England (PHE) routinely conducts national surveillance of IMD using a combination of clinical and laboratory reporting schemes [5]. At the same time, PHE’s Meningococcal Reference Unit (MRU) provides a national service for species confirmation, grouping, typing, subtyping, and antimicrobial susceptibility testing of all invasive *Neisseria meningitidis* isolates. The MRU also provides free nonculture polymerase chain reaction (PCR) confirmation of meningococcal diagnosis (including genogroup and genosubtype analysis) for clinical specimens that are routinely submitted by National Health Service (NHS) hospitals in England and Wales [5].

From 2012, after the observed increase in MenW cases, laboratory-confirmed (culture and/or PCR) MenW cases diagnosed in England and Wales were identified retrospectively and prospectively and followed up for 3 epidemiological years (July 2010 to June 2013) by requesting that the case-patient’s general practitioner or, alternatively, the hospital physician or public health consultant complete a short questionnaire. Case-patients reported to have died were cross-checked with the national electronic Personal Demographic Service database.

<table>
<thead>
<tr>
<th>Age Group, y</th>
<th>MenW Type</th>
<th>Epidemiological Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;5</td>
<td>2a</td>
<td>2008–2009</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>2008–2009</td>
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<td></td>
<td>Nonculture</td>
<td>2008–2009</td>
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<tr>
<td>&lt;5 total</td>
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<tr>
<td>5–19</td>
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<td></td>
<td>Other</td>
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<td></td>
<td>Nonculture</td>
<td>2008–2009</td>
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<td>5–19 total</td>
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<tr>
<td>20–44</td>
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<td></td>
<td>Nonculture</td>
<td>2008–2009</td>
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<td>20–44 total</td>
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<tr>
<td>45–64</td>
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<td>45–64 total</td>
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<td>≥65</td>
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<td>≥65 total</td>
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<tr>
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<td>Nonculture</td>
<td>2008–2009</td>
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<tr>
<td>All ages total</td>
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</tbody>
</table>

Data are presented as No. of cases (No. of deaths).

Abbreviation: MenW, meningococcal capsular group W.
demographics/pds), and the cause of death was ascertained from death registration records provided to PHE by the Office for National Statistics (www.statistics.gov.uk).

Genomic data for all English and Welsh MenW isolates collected over the corresponding 3-year period were obtained from the Meningitis Research Foundation Meningococcus Genome Library (www.meningitis.org/genome-library). The population structure of the MenW sequence type (ST) 11 clonal complex (cc11) isolates was characterized at high resolution by performing a partial meningococcal genome comparison using the genome comparator tool in www.pubmlst.org. In brief, the isolates were compared in terms of a total of 1546 of 1592 core genes employing a “gene by gene” approach that uses arbitrary, sequentially assigned, preindexed allele identifiers at each gene, akin to those of standard 7-locus multilocus sequence typing [6]. A Neighbor-net phylogenetic network was then generated by applying SplitsTree4 (version 4.12.8) to the resulting distance matrix [7]. The remaining 46 core genes were excluded from the analysis, having previously generated false differences due to paralogy (eg, the Elongation Factor Tu genes neis0116 and neis0128) or the presence of hypermutable homopolymeric tracts (eg, the phase variable lacto-N-neotetraose biosynthesis glycosyl transferase gene, lgtA, or neis1902). The included/excluded loci are listed in Supplementary Table 1. To place the English/Welsh MenW:cc11 population in a broader context, other available MenW:cc11 genomes in www.pubmlst.org (identifiers 26899, 26914, 26898, 21587, 27087, 19957, 21578, 21573, 21584, 21588, 21582, 21583, 21581) were also included. Clinical and laboratory data were merged into a single Microsoft Access 2010 database (Microsoft Corporation, Redmond, Washington), cleaned, and de-duplicated before exporting to Stata version 13.0 (StataCorp LP, College Station, Texas) for analysis. Data are mainly descriptive. Proportions were compared using the $\chi^2$ test or Fisher exact test, as appropriate.

PHE has approval under Section 60 of the Health and Social Care Act 2001 (now subsumed into the National Information Governance Board for Health and Social Care with Section 60, now Section 251 of the NHS Act 2006) to process confidential patient information for public health surveillance (see http://www.legislation.hmso.gov.uk/si/si2002-20021438.htm).

RESULTS

Epidemiology (July 2000–June 2014)
The total number of laboratory-confirmed IMD cases in England and Wales declined from 2448 during the 2000–2001 epidemiologic year to 664 during 2013–2014 mainly because of routine MenC vaccination since 1999–2000, alongside a secular decline in MenB cases after 2001–2002 (Figure 1). The Hajj-associated MenW outbreak peaked in 2000–2001 and ended in 2003–2004, after which time none of the MenW cases were epidemiologically linked to Hajj travel [4]. MenW accounted for 127 of 2448 cases (5.2%) in 2000–2001 and 21 of 1164 cases

![Figure 1](cid201506015feb131864550)

**Figure 1.** Laboratory-confirmed cases of invasive meningococcal disease by capsular group in England and Wales during the 2000–2001 to 2013–2014 epidemiological years. Abbreviations: MenB, meningococcal capsular group B; MenC, meningococcal capsular group C; MenW, meningococcal capsular group W; MenY, meningococcal capsular group Y.

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In 2008–2009, MenW cases increased year-on-year, and 98 cases were confirmed in 2013–2014, accounting for 15% of 664 IMD cases (Table 1). Phenotypic characterization of the MenW isolates revealed that the increase was nearly all caused by MenW strains phenotypically expressing PorB serotype 2a (Figure 2). During 2009–2010, 4 of the 5 MenW:2a cases were diagnosed in adults aged ≥45 years but, by 2011–2012, MenW:2a cases were observed in all age groups. By contrast, non-2a MenW cases remained relatively constant across the age groups.


Between July 2010 and June 2013 (3 epidemiologic years), there were 129 laboratory-confirmed MenW cases, including 106 (82%) confirmed by culture, which were phenotypically characterized as MenW:2a (n = 64 [60%]) or nontypeable (n = 42 [40%]). Whole-genome sequencing data were available for all 64 MenW:2a isolates, and all were confirmed as cc11, as were 7 additional MenW isolates that had been phenotypically characterized as MenW:2a (n = 64 [60%]) or nontypeable (n = 42 [40%]). The remaining MenW isolates belonged to ST-22 (n = 32), ST-174 (n = 2), and ST-23 (n = 1) clonal complexes. All but 1 of the recent MenW:cc11 isolates from cases in England and Wales, along with 3 Irish isolates from 2013, formed a tight cluster on the phylogenetic network (mean distance, 33 [standard deviation (SD), 16] loci; Figure 3). The remaining isolate was relatively distant from these (mean distance, 200 [SD, 8] loci) forming a cluster (mean distance, 29 [SD, 17] loci) with 6 South African isolates (2004–2011) and an English isolate from 2000. This cluster was also relatively close to an isolate from Burkina Faso in 2002 (mean distance, 84 [SD, 15] loci). Within the network were also 2 relatively intermediate isolates from South Africa. The predominant genotype among the recent English/Welsh cc11 isolates (including the PorB nt types) was P1.5,2: F1–1: ST-11 (cc11) (58/71 [81.7%]). The remainder differed by just 1 of either PorA, FetA, or ST: P1.5–1,10–4: F1–1: ST-11 (n = 1), P1.7–2,14: F1–1: ST-11 (n = 1), P1.5,2: F1–146: ST-11 (n = 4), P1.5,2: F1–5: ST-11 (n = 1), P1.5,2: F1–1: ST-10651 (n = 4), P1.5,2: F1–1: ST-1860 (n = 1), and P1.5,2: F1–1: ST-10284 (n = 1). In addition, all but 1 of the tightly clustered recent isolates possessed alleles for factor H binding protein (fHbp) peptide 22, whereas the more distant isolate (along with the closely related South African and historical English isolates) possessed an allele for fHbp peptide 9.


Clinical information was collected for all 129 laboratory-confirmed MenW cases during the 3 epidemiological years. A quarter of cases occurred in children aged <5 years, and half the cases in adults aged ≥45 years. The demographics, clinical characteristics, risk factors, infecting strain, and outcome by infecting strain are summarized in Supplementary Table 2. Of note, only 2 case patients had a history of travel—both were overseas residents visiting the United Kingdom when they...
became unwell. None of the case-patients were contacts of another MenW case. Predisposing risk factors (12/129 [9%]) and other comorbidities (12/120 [9%]) were uncommon, mainly malignancy and chronic renal disease, respectively, and occurred mostly in those aged ≥65 years. None had asplenia, splenic deficiency, or previously known complement deficiency. Half of the case-patients presented with septicemia, which was relatively consistent across the age groups. Bacteremic pneumonia (10 cc11, 5 non-cc11) was more common in those aged ≥45 years (13/65 [20%] vs 2/64 [3%] in those aged <45 years; P = .003). Of note, 9 case-patients developed septic arthritis with MenW isolated from joint fluid (4 cc11, 3 non-cc11, 2 unknown), mainly at extremes of age. In addition, 5 cases presented with severe upper respiratory tract infection (epiglottitis or supraglottitis; 3 cc11, 1 non-cc11, 1 unknown). Overall, intensive care admission was high (37%), especially in older children and young adults. Two-thirds of those with meningitis and septicemia required intensive care unit (ICU) admission (14/21 [67%]), 56% with meningitis (9/16), 40% pneumonia (6/15), and 30% septicemia (19/63). In total, 19 patients died and 15 deaths were meningocccal-related (case-fatality ratio [CFR], 13%), with nearly all deaths occurring within 72 hours of diagnosis (13/15 [87%]). The 4 nonmeningococcal deaths occurred in adults aged ≥65 years who recovered from their infection but died ≥6 weeks later. CFR was similar for all clinical presentations apart from those with severe upper respiratory tract infection (none required intensive care or died).

There was no association between the infecting strain (cc11 vs non-cc11 vs not typed or type 2a vs nontypeable vs not typed) and age at disease, risk factors, clinical presentation, ICU admission, or case fatality. However, 14 of the 19 deaths among cases with typed isolates during 2010–2011 to 2012–2013 were associated with MenW:cc11. In contrast to non-cc11 strains, which were only fatal in older adults, MenW:2a was also associated with death in children and young adults.

**DISCUSSION**

The increase in invasive MenW disease in England and Wales since 2009–2010 is almost entirely due to endemic expansion of a single clone belonging to cc11 and, although the number of cases remains low nationally, this expansion appears to be continuing, with no evidence of plateauing after 5 years.

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**Figure 3.** Phylogenetic network analysis of meningococcal capsular group W (MenW) clonal complex 11 (cc11) isolates. The network (including all English and Welsh MenW:cc11 isolates from July 2010 to June 2013 along with other MenW:cc11 isolates, available on pubmlst.org; accessed 5 October 14) shows a complex population structure for English and Welsh MenW:cc11. All but 1 of the recent English and Welsh isolates formed a tight cluster alongside the recent Irish isolates, whereas the remaining isolate segregated with relatively historical isolates from England, South Africa, and Burkina Faso. Label color: none/green (England and Wales, July 2010–June 2013); blue (England and Wales, 2000); gray (Ireland, 2013); red (South Africa, years marked individually); black (Burkina Faso, 2002).
In 2013–2014, most of the MenW isolates that underwent molecular typing belonged to the 2a phenotype, and it is likely that this strain contributes to a similar high proportion of the untyped, PCR-positive cases. Genomic analysis of MenW isolates over 3 epidemiological years confirmed all PorB 2a isolates as belonging to cc11, in addition to 7 of the PorB nontypeable isolates. PorA P1.5,2 has also proved to be an imperfect marker for MenW:cc11 isolates, thus highlighting the usefulness of extended sequence analyses over serological characterization.

The emergence of cc11 cases occurred initially in adults but soon extended across all age groups such that, by 2013–2014, cc11 accounted for nearly all MenW cases in individuals aged 5–64 years and two-thirds of cases in those aged <5 and ≥65 years. Although there were no discernible differences in age distribution, clinical characteristics, or outcome in disease caused by cc11 strains compared with other strains or PCR-positive cases, the continuing increase in overall MenW cases is concerning. Unlike previously observed temporary increases in meningococcal group Y disease, for example, which occurred primarily in older adults with underlying comorbidities [8], the MenW cases have occurred mainly in healthy individuals of all ages, who developed severe clinical disease often requiring intensive care support. Moreover, the clinical presentation in a significant proportion of MenW cases was not typical of meningococcal disease, with septic arthritis and severe respiratory tract infections being overrepresented. Such atypical presentations have previously been reported in MenW case-patients [9]. Among 119 pediatric MenW cases diagnosed in France during 2001–2008, for example, 10 (8%) presented with septic arthritis [10]. Similarly, an older US study also reported a higher prevalence of arthritis with MenW (3.3%) than MenB (0.6%) or MenC (1.1%) disease [11]. Meningococcal epiglottitis and supraglottitis are even rarer presentations of meningococcal disease, and, to our knowledge, have never been reported for MenW:cc11 isolates, thus highlighting the usefulness of extended sequence analyses over serological characterization.

The MenW-associated CFR in our cohort (12%) was not as high as the 20% reported during the Hajj-associated outbreaks [4, 14]. It has been postulated that cc11 is more virulent and this is why it was associated with higher CFR for the Hajj-associated MenW (20%) compared with other culture-confirmed meningococcal disease during 1995–2000 (9%; risk ratio [RR], 2.0; 95% confidence interval [CI], 1.3–3.1) or other MenW serotypes (9%; RR, 2.6; 95% CI, 1.4–4.8), but similar to the CFR for culture-confirmed MenC:2a cases, also associated with cc11 (15%; RR, 1.3; 95% CI, 0.8–2.1) [4]. We did not observe any difference in CFR among cases caused by MenW:2a and other meningococcal strains. However, the recent increase in MenW:2a cases has led to more MenW-associated deaths attributed to this strain and, more concerning, MenW-related deaths were, for the first time in the past decade, observed in children and young adults.

Unlike the international MenW outbreak following Hajj pilgrimages in 2000 and 2001 [4], our follow-up of cases did not identify any association with travel or recent entry into the United Kingdom, nor was there any evidence of clustering of cases within households. A number of countries in Latin America, Africa, and eastern Asia have already reported increases in endemic MenW disease caused by strains belonging to cc11. In Brazil, MenW was uncommon until 2003, when MenW:cc11 emerged and started causing sporadic cases, outbreaks, and field clusters [15, 16]. A similar increase in MenW cases has been reported in Argentina since 2008, where, like Brazil, most reported cases were diagnosed in young children [17]. In Chile, MenW:2a belonging to cc11 emerged at the end of 2011 and, during 2012, 58% of laboratory-confirmed cases were caused by MenW, with 47% of cases occurring in children <5 years [18]. This increase was also associated with higher case fatality for IMD cases at all ages, from 15% in 2011 to 21% in 2012.

In South Africa, MenW incidence in Gauteng province increased across all age groups from 0.06 per 100 000 in 2000 to 2.99 per 100 000 in 2005, but particularly among infants [14]. When compared to other capsular groups, MenW was more likely to affect children aged <5 years, to cause septicemia rather than meningitis, and to result in a fatal outcome [14]. The responsible MenW isolates were highly clonal (ST-11/ET-37 complex, [W]ET-37 clone) and indistinguishable from the Hajj-related outbreak isolates.

In sub-Saharan Africa, the Hajj-associated MenW strain caused epidemics in several countries across the meningitis belt during the early 2000s [14]. In 2012, cases of MenW belonging to cc11 reemerged, 2 years after the mass vaccination program with a meningococcal group A (MenA) conjugate vaccine [19]. In Burkina Faso, for example, a total of 5807 meningitis cases were reported through enhanced surveillance in 2012, of which 2353 (41%) were laboratory confirmed. MenW accounted for 62%, and all 61 isolates that were characterized belonged to cc11. The predominant strain was P1.5,2; F1–1; ST-11 (cc11), which accounted for 90% (55/61) of isolates, and was the same genotype as that of the 2002 Burkina Faso epidemic strain. Other sub-Saharan countries have similarly reported an increase in MenW disease, also in young children, following the successful implementation of the MenA conjugate vaccine [20, 21]. A small but significant increase in cc11 MenW has also been reported in southeastern China during 2011–2012.
where a comparable MenW strain was isolated from patients, their close contacts, and healthy carriers, suggesting possible establishment and local spread of this particular strain.

With such high volumes of international travel, it is not surprising that some countries have observed an increase in travel-associated MenW infections. In France, for example, half of the 16 MenW cases diagnosed during January–April 2012 (compared with <5 annual cases previously) could be linked with travel to, or contact with someone who had traveled to, sub-Saharan Africa during the MenW epidemic [22]. The 8 imported cases were caused by MenW:2a strains of the genotype P1.5,2:F1–1:ST-11 (cc11), the same as that observed among cases from Cote d’Ivoire [23].

Other than the United Kingdom, as far as we are aware, no other European country has yet reported a recent increase in endemic MenW disease. The core genome comparison among MenW:cc11 isolates causing IMD in England and Wales has afforded unprecedented resolution of what normally appears to be a single clone using conventional multilocus sequence typing. The tight clustering (mean distance of 33 loci) of the majority of the recent English and Welsh MenW:cc11 isolates is consistent with this being a relatively rapid clonal expansion. By comparison, the mean distance among the relatively stable and endemic cc22 (n = 32) isolates over this time period was 185 loci (SD, 87 loci) (data not presented). The only isolate belonging to the relatively rare, historical cluster was obtained from a toddler of black African ethnicity, who had never traveled outside England, although this does not preclude recent import and subsequent transmission. It is tempting to speculate that this cluster (also containing historical isolates from South Africa, Burkina Faso, and England) represents that of the so-called Hajj outbreak strain—indeed, the corresponding English/Welsh case from 2001 involved a recently returned pilgrim. Nonetheless, the small number of fully characterized isolates from this period and the apparent complexity of the MenW:cc11 population structure highlight the need for more detailed characterization of geographically and temporally diverse MenW:cc11 isolates to fully reconcile past and current episodes of increased MenW:cc11 cases.

The emergence and rapid clonal expansion with continuing increase in endemic MenW:cc11 disease in the United Kingdom will require close monitoring in the coming years, particularly given that natural immunity against this capsular group is low across all age groups [24]. MenW is potentially preventable through vaccination with the quadrivalent MenACWY conjugate vaccine [25], and if the trend continues to establish itself in children and young adults (as it has done in South Africa, Latin America, and other regions), then it may be prudent to consider supplementing the current MenC conjugate vaccine program with the quadrivalent conjugate vaccine. Vaccination of an age group with high MenW carriage, if such a group could be identified, could also potentially prevent overall cases through indirect (herd) protection.

Supplementary Data

Supplementary materials are available at Clinical Infectious Diseases online (http://cid.oxfordjournals.org). Supplementary materials consist of data provided by the author that are published to benefit the reader. The posted materials are not copyrighted. The contents of all supplementary data are the sole responsibility of the authors. Questions or messages regarding errors should be addressed to the author.

Notes

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Potential conflicts of interest. R. B. and S. N. L. have performed contract research for the PHE and St George’s University of London, respectively, on behalf of Pfizer, GlaxoSmithKline, and Novartis Vaccines, but have not received any personal remuneration. The Immunisation Department at PHE Colindale also provides vaccine manufactures with postmarketing surveillance reports that the Marketing Authorisation Holders are required to submit to the UK Licensing authority in compliance with their Risk Management Strategy. A cost recovery charge is made for these reports. All other authors report no potential conflicts.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

References