Report for the National Audit Office

Trends in rates of Healthcare Associated Infection in England 2004 to 2008

Background

Healthcare associated infections (HCAI) are often caused by micro-organisms that are not transmissible in the classical sense but which take advantage of breaches in the hosts defences against infection (e.g. bowel flora causing wound infection after surgery, skin organisms causing bloodstream infection via a vascular device). Micro-organisms that cause HCAI may be derived from the patients' own flora or may have been acquired from other patients, the environment or staff through contact with people or equipment, or more rarely via the air. The factors that contribute to the acquisition of HCAI are therefore complex and their prevention depends on a range of infection control procedures. For example, preventing a patient becoming colonised with meticillin-resistant *Staphylococcus aureus* (MRSA) is dependant on hand hygiene being performed between patient contacts, however, preventing the patient subsequently developing a surgical wound infection caused by MRSA depends on appropriate antimicrobial prophylaxis, good surgical technique, a high standard of infection control during surgery and effective protection of the wound from contamination post-surgery.

Recent major changes to the delivery of healthcare mean that patients may spend short period of time in hospital but receive ongoing healthcare in community settings. In addition, as life expectancy increases many elderly people are living in the community with chronic illness which makes them vulnerable to both infection and repeated admission to hospital for treatment. These factors make it difficult to discriminate between infections associated with healthcare delivered in hospital, associated with community healthcare, or acquired in the community. Such distinction is important since it helps to inform and drive prevention strategies.

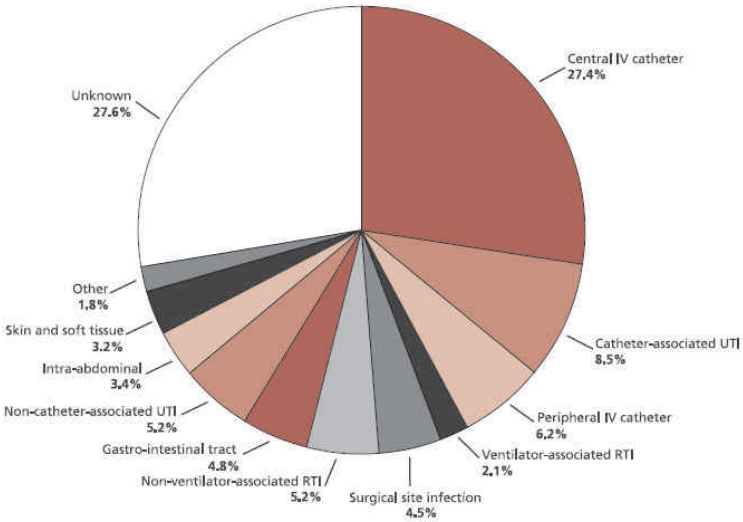
Surveillance is used to monitor trends in the occurrence of infections and to provide signals to trigger investigation and action. Such data has limited value in explaining the cause of infection or the reasons underlying changes in trends because information is generally only collected on the infection and cannot be directly related to the population in which the infections occur. Epidemiological studies are required to explore these relationships.

In recent years most of the focus on healthcare associated infections (HCAI) and associated surveillance has been mostly directed at a few infections that account for a small proportion of HCAI - bloodstream infections caused by MRSA, *Clostridium difficile* infection and surgical site infection (SSI) following orthopaedic surgery. This review therefore aims to describe trends in rates of HCAI since the last National Audit Office (NAO) report was published in 2004 for which surveillance data are available and evaluate them in the broader context of the range of infections that occur in patients receiving healthcare.

Bacteraemia

Whilst bacteraemia is a relatively uncommon cause of HCAI accounting for around 7% of primary HCAI (and 4% secondary to another focus of infection)[1], it is more readily subject to surveillance because diagnosis can be based on a positive laboratory culture rather than a complex set of clinical signs and symptoms required to identify many other HCAI. Data from more than 10 000 bacteraemias acquired in hospital patients from 96 hospitals that participated in the Nosocomial Infection National Surveillance System (NINSS) between 1997 and 2002 suggests that approximately 44% of hospital-acquired bacteraemias are associated with invasive devices, with nearly two thirds of these related to central vascular devices (figure1).[2]

Figure 1: Probable source of hospital-acquired bacteraemia
Source: NINSS, Health Protection Agency



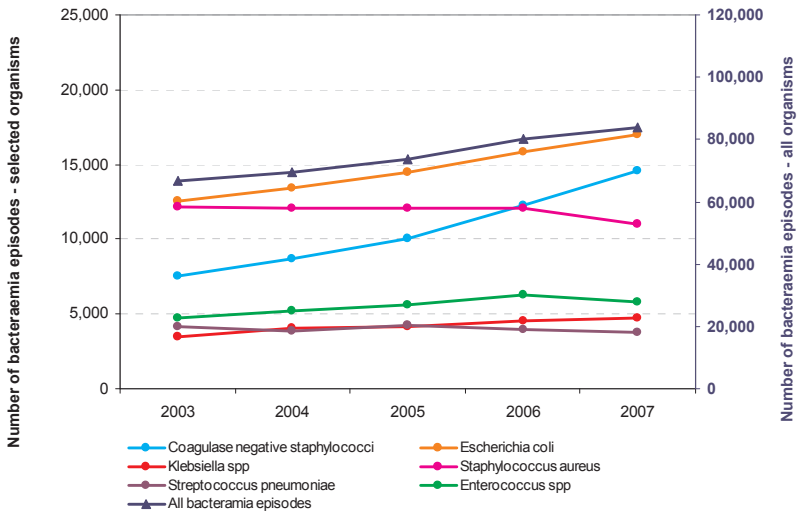
Whilst the mandatory surveillance of bacteraemia is focused on MRSA, the Health protection Agency (HPA) has collected data via a voluntary laboratory-based surveillance system on all pathogens causing bacteraemia since the mid-1970s. These data therefore provide an indication of the wider range of pathogens that cause these serious infections. In 2007, over 100,000 bacteraemia reports were received. However, although the number received has increased steadily, the trend has to be interpreted with caution as it is influenced by the completeness and reliability of reporting which varies between laboratories and over time. In particular, when a laboratory moves from manual to automated data transfer systems marked changes may occur in the volumes of reports and types of pathogen reported. To eliminate some of these effects, a subset of 174 (70% of the total 250 reporting to the system) laboratories that have reported consistently for each year of the last five years have been used to evaluate trends. The 404,144 episodes of infection included in this subset represent 70% of all episodes reported to Labbase during this time period. Whilst the majority (78%) of the specimens where the location of the patient is known are derived from patients in hospital, those that have acquired the infection in the community cannot be reliably distinguished and it is also not possible to confirm whether all the bloodstream infections reported are clinically significant.

Figure 2 illustrates trends in bacteraemia caused by the six most common pathogens in this subset of consistent reporters. It demonstrates that the Gram negative bacterium *Escherichia coli* is the most common cause of bacteraemia reported accounting for 20% of infections and with the number of episodes of infection increasing by 36% between 2003 and 2007. *E.coli* bacteraemia are most likely to represent a secondary infection, frequently linked to urinary tract infection,

gastrointestinal sepsis or surgical infections. Reports of the other main Gram-negative pathogen from blood, *Klebsiella spp*, have also increased by 36% over the same time period. The number of episodes of coagulase-negative Staphylococci (CNS) bacteraemia has doubled since 2003. It is now the second most common reported pathogen accounting for 17% of all reports. CNS are examples of skin commensal organisms that can act as opportunistic pathogens, particularly in association with central vascular devices in the seriously ill. Since blood culture specimens are prone to contamination with CNS this marked increase in reports could be explained by changes in reporting by laboratories rather than reflecting a real increase in clinically significant infections. However, such a marked increase is unlikely to be explained solely by case ascertainment and merits further investigation. In addition, the increasing significance of CNS as a cause of HCAI was previously demonstrated by the NINSS data where CNS accounted for 16% of organisms reported as causing bacteraemia.[2] *S. aureus* is now the third most common bacteraemia pathogen causing 13% of bacteraemias, with MRSA only responsible for about 4%.

These trends may be explained by a number of factors including increasing medical treatment of an aging population, blood cultures being more commonly taken to diagnose infection or results being more commonly reported to the HPA. However, the most likely underlying reasons are difficult to determine as there is little data on the patients who acquire the bacteraemia or the primary sources of infection, and the extent to which these infections can be prevented is therefore not clear.

Figure 2: Pathogens causing bloodstream infection reported to the voluntary laboratory surveillance system (subset of consistent reporting laboratories in England). *Source: HPA, LabBase*



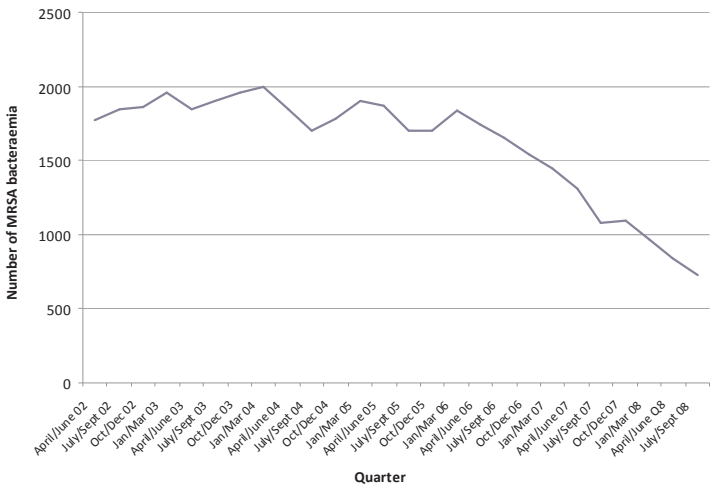
MRSA as a cause of bloodstream infection

The number of MRSA bacteraemia reported to the HPAs voluntary laboratory surveillance system had increased by 25% between 2000 and 2004. The mandatory surveillance of MRSA bacteraemia was initiated in 2001 and for the first 3 years required acute Trusts to report only the number of cases of MRSA bacteraemia. A web-based reporting system, capturing data on individual cases of MRSA bacteraemia, was introduced in October 2005.

In 2003-4, 7647 MRSA bacteraemias were reported to the mandatory surveillance system. The number of MRSA bacteraemia reported had increased up to the

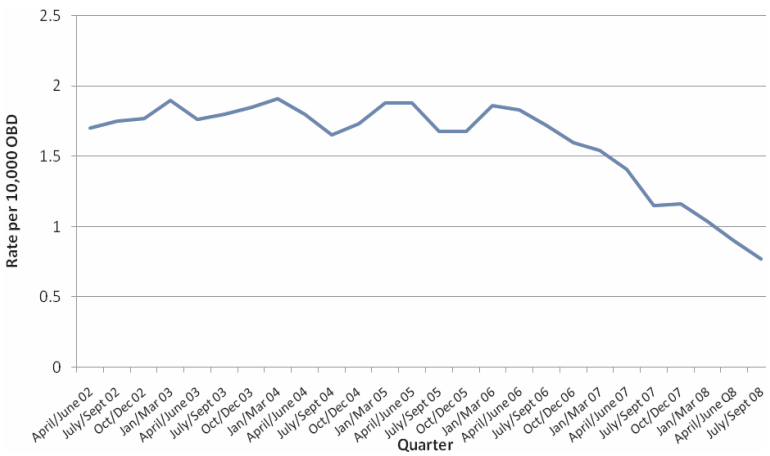
October/December 2003 reporting period and then appeared to stabilise until the April/June 2006 reporting period when the number of reports began to decline rapidly (figure 3). The average quarterly count of MRSA bacteraemia was 1925 in the 2003/4 financial year (used by the Department of Health as the baseline for the reduction target). By the January to March quarter in 2008 this number had reduced by 57% to 836.

Figure 3: Number of MRSA bacteraemia reports to the mandatory surveillance system.
Source: HCAI surveillance system



This picture is more accurately illustrated in Figure 4 which shows the rate of MRSA bacteraemia per 10 000 occupied bed days (OBD). These rates are derived by using the average daily occupied beds in the acute Trusts included in the surveillance as a denominator. Since the average number of daily occupied beds in these Trusts has fallen steadily since 2004 this calculation adjusts the number of MRSA bacteraemia reports by activity, although the adjustment can only be made by using an average over the whole year and therefore may mask temporal changes. It suggests that the rates were fluctuating between 1.6 and 1.8 cases per 10 000 OBD with evidence of winter increases in rates but no clear indication of a sustained reduction until the April/September 2006 reporting period when the rate began to decline steadily. By April 2008 the rate had declined by 34% compared to the baseline year, and by September 2008 had reduced by 59% compared to the last quarter of the baseline year.

Figure 4: Rate of MRSA bacteraemia per 10 000 occupied bed-days
Source: HCAI surveillance system and KH03 Hospital Activity Data



Trust type and regional trends

Prior to April 2004 there were marked variations in the rate of MRSA bacteraemia between regions and type of acute Trust, with rate of MRSA bacteraemia in the London region almost double that of other regions. Similarly, rates were considerably higher in acute teaching Trusts compared to other Trust types (figures 5 and 6). Marked reductions in rates in acute teaching Trusts between 2002 and 2004 were offset by increases in other Trust types. Since 2006, the reduction in rates of MRSA bacteraemia has occurred across all Trust types and although a small number of Trusts have achieved very large reductions, these Trusts account for less than 10% of the overall decrease.

Figure 5: Rate of MRSA bacteraemia by 10 000 occupied bed days by NHS Trusts type.
Source: HCAI surveillance system and KH03 Hospital Activity Data

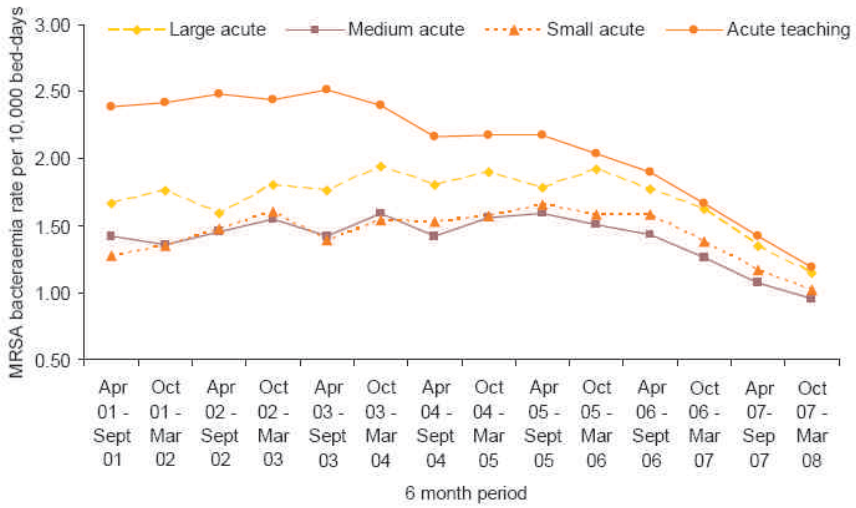
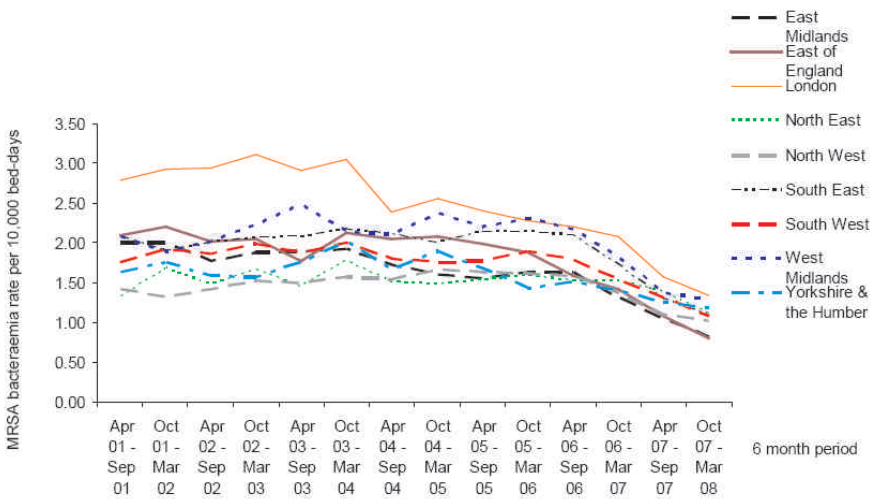
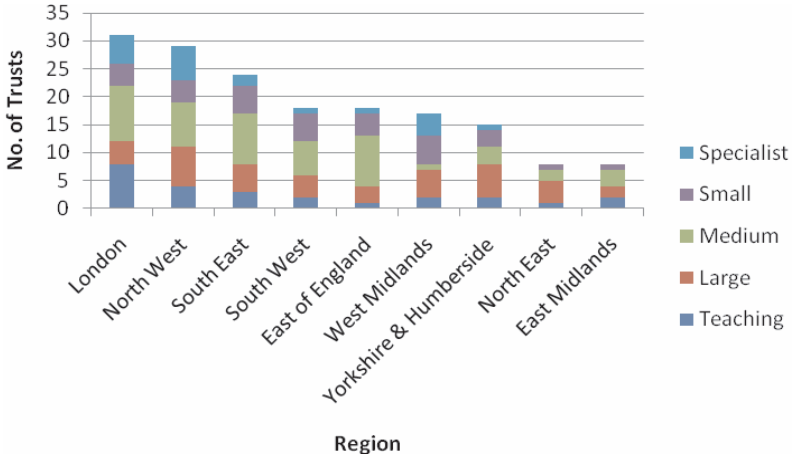


Figure 6: Rate of MRSA bacteraemia by 10 000 occupied bed days by region
Source: HCAI surveillance system and KH03 Hospital Activity Data



Regional variation needs to be interpreted with caution because it reflects significant variation in the number and type of Trusts between regions (figure 7).

Figure 7: Number of Trusts by type and Region



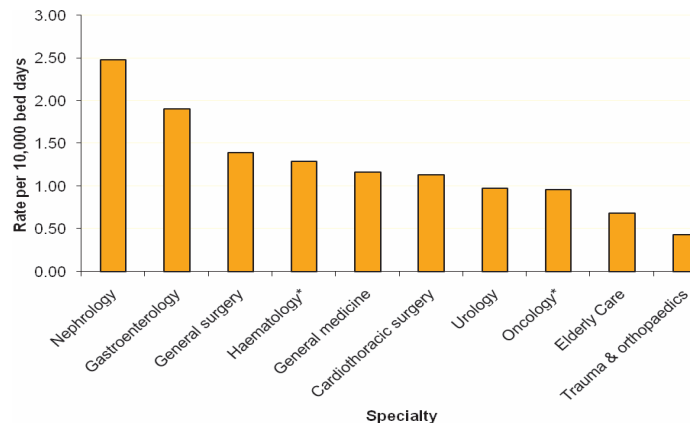
The greater number of acute teaching and large Trusts will, at least in part, account for the higher rates in the London region. The rates in both London region and acute teaching Trusts fell by 22% and 10% respectively between March and September 2004. However, since then both the rates and trends in rates across Trusts types and region have been similar.

Risk factors for MRSA bacteraemia

The risk of developing MRSA bacteraemia varies considerably between specialties, reflecting a number of factors including the age and severity of illness of patients admitted to the specialty, the prevalence of MRSA colonisation in the patient group, and the extent of invasive treatment that increase their susceptibility to bacteraemia in general, and *S. aureus* bacteraemia in particular. Thus the rate of bacteraemia is greatest in nephrology with contributory factors being patients with compromised immune systems and the requirement for repeated access to the vascular system in those in established renal failure (Figure 8).

Approximately one third of MRSA bacteraemia occur in patients admitted from the community, and 64% occur in men. Some potential risk factors for MRSA bacteraemia are available from the mandatory surveillance system and have role in evaluating common factors that might explain local increases or decreases in counts. However, without specific denominator data related to patients at risk of bacteraemia in both hospital and community settings, the value of this surveillance data in evaluating contributory causes of MRSA bacteraemia is limited.

Figure 8: Rate of MRSA bacteraemia by admission specialty (data collected between 2005 and 2007). *Source: HCAI Surveillance System*



MRSA bacteraemia in patient receiving renal dialysis

Nearly 5% of all MRSA bacteraemias occur in patients in established renal failure (ERF). By using data provided by the UK Renal Registry it has been possible to determine an accurate rate of MRSA bacteraemia in these patients. This analysis shows significant variation in rate of MRSA bacteraemia between renal units with a average of around 1 per 100 prevalent dialysis patients/year but a range of between 0 and 3 between renal units.[3] There is an 8 fold increase in risk in patients receiving dialysis via a central vascular device rather than an arteriovenous graft or fistula, illustrating the potential impact of care delivery on the risk of bacteraemia. Although over 60% of the MRSA bacteraemias in patients in ERF occurred in men, the ratio of men to women in ERF is 2:1 and hence the actual risk of bacteraemia was very similar in both genders.

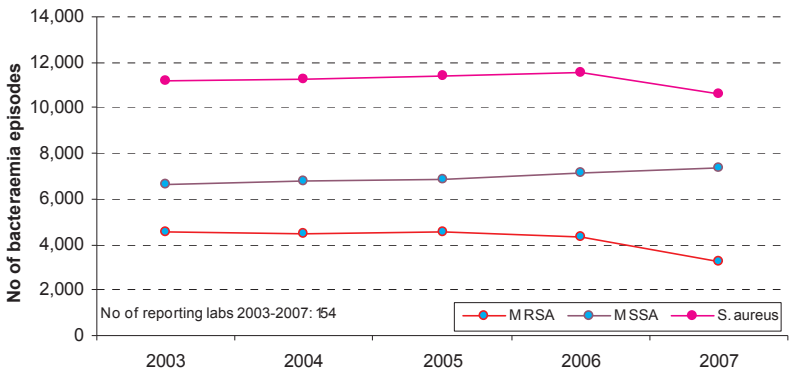
Proportion of S.aureus bacteraemia caused by MRSA

The change in rate of MRSA bacteraemia needs to be seen in the context of bacteraemia caused by *S. aureus* in general, since if the decrease has been brought about by strategies that have prevented or effectively treated colonisation with MRSA, then meticillin-sensitive *S.aureus* (MSSA) bacteraemia may have replaced those infections previously caused by resistant strains.

In data collected by a set of sentinel laboratories in England, Wales and Northern Ireland and all laboratories in Scotland for the European Antimicrobial Resistance Surveillance System (EARSS) in 2007, 36% of *S.aureus* isolates were resistant to meticillin. This proportion had decreased from the peak of 44% reported in 2001.[4]

The proportion of *S. aureus* bacteraemia due to meticillin-resistant strains was reported by the mandatory surveillance system until September 2005. At this time 39% were MRSA. Since complete data on MSSA is not collected by the HCAI web-enabled data collection system the proportion of *S.aureus* that are now MRSA can only be estimated from the routine voluntary laboratory reports. Figure 9 shows the trend in reports of MRSA and MSSA bacteraemia to the voluntary surveillance system. This data indicates that by 2007 the proportion of *S. aureus* bacteraemia caused by MRSA had decreased to 29%. However, episodes of MSSA have increased by 9% between 2004 and 2007 and therefore the overall burden of *S. aureus* bacteraemia seen in these reports has only declined by 5% over this time period.

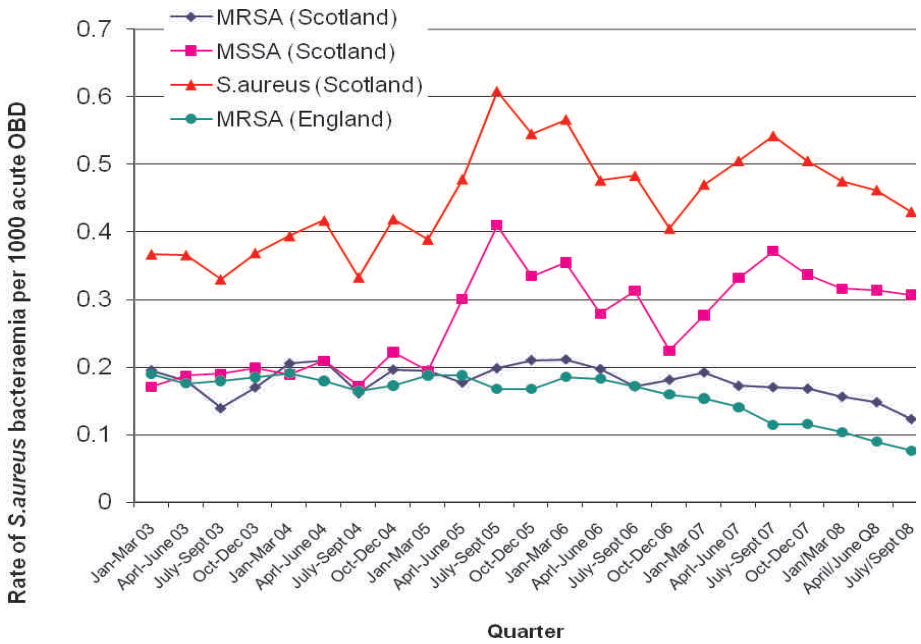
Figure 9: Trends in number of episodes of *S. aureus* bacteraemia, including MRSA and MSSA reported to the voluntary bacteraemia surveillance system (subset of consistent reporting laboratories in England). *Source: HPA, LabBase*



Although the analysis of a subset of consistently reporting laboratories eliminates some of the effects of variation in reporting over time, the data do not equate with the MRSA reported via the mandatory surveillance system and rates adjusted for occupied bed days cannot be readily calculated.

In Scotland, the mandatory bacteraemia surveillance includes both MSSA and MRSA, and therefore enables trends in rates of MRSA bacteraemia to be interpreted in the context of all *S. aureus* bacteraemia using a standard approach to data collection. In figure 10 data from the mandatory *S.aureus* bacteraemia surveillance in Scotland has been combined with data from the mandatory surveillance of MRSA bacteraemia in England.[5] The data should be interpreted with caution because of slight differences in the derivation of the OBD denominator. However it does suggest that the overall rate of MRSA bacteraemia is also decreasing in Scotland, although the decline appears to have commenced later than in England, from about April 2007. In the latest quarter in Scotland MRSA accounted for 30% of *S. aureus*. The fluctuation in rate of MSSA between 2004 and 2007 probably reflects variation in case reporting as the system became established. However, MSSA reporting has been more stable since April 2007 and suggests that in Scotland rates of MSSA have also begun to decline although to a lesser extent than MRSA, with the reduction in MRSA and MSSA between July 2007 and September 2008 of 28% and 18% respectively (figure 10). However, in Scotland whilst strategies directed at reducing rates of MRSA bacteraemia have been similar, differences in approach to prevention and the broader focus of surveillance on all *S. aureus* bacteraemia may have influenced these trends.

Figure 10: Trend in rate of *S.aureus* bacteraemia in Scotland and rate of MRSA bacteraemia in England. *Source: Health Protection Scotland, HPA HCAI Surveillance System*



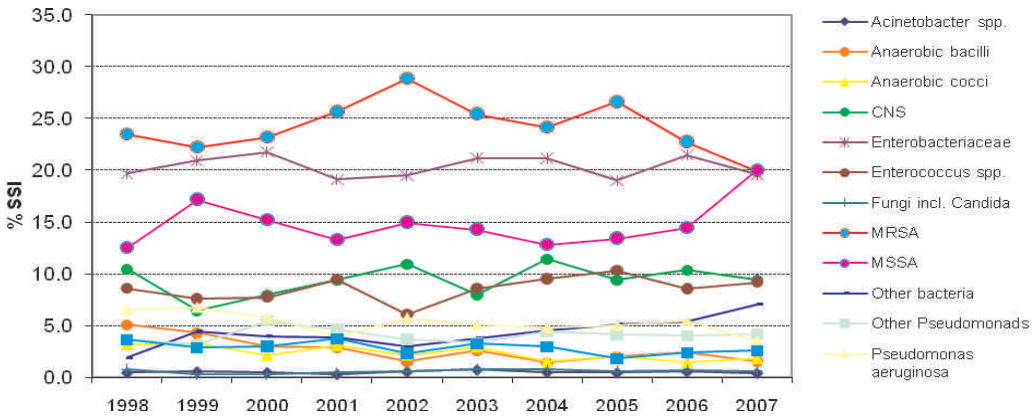
Although these comparisons are crude they suggest that in England the marked reductions that have occurred in MRSA bacteraemia since 2006 have not been accompanied by the same overall decline in *S.aureus* bacteraemia. More data on the sources of bacteraemia in these patients and underlying risk factors for bacteraemia are required to improve understanding of the factors that may have influenced these trends.

MRSA as a cause of surgical site infection (SSI)

Since the Surgical Site Infection Surveillance Service (SSISS) (previously NINSS) was established in 1997 it has collected data on the organisms considered to be responsible for SSI. This data is available on about 80% of the infections. Across a range of categories of surgical procedure, *S.aureus* has been reported as the cause of around 40% of surgical site infection (SSI) and between 2000 and 2005 MRSA consistently accounted for over two-thirds of these *S.aureus* SSI (nearly double the proportion of MRSA vs MSSA seen in bacteraemia) or approximately 25% of all SSI. The reasons for this higher proportion of MRSA in S.aureus causing SSI are not clear but probably reflect the extent to which MRSA colonises the skin of the largely elderly patient population admitted to hospital and are therefore available to cause SSI. The mostly likely source of SSI is endogenous infection in patients colonised with the organism at the time of surgery or who acquire it immediately after surgery before the incision has healed. As demonstrated by the prevalence survey, SSI accounts for 15% of HCAI and therefore the high proportion caused by MRSA represents a significant burden of infection caused by this antimicrobial-resistant pathogen.

Since 2006 there is evidence that, as with MRSA bacteraemia, the overall proportion of *S. aureus* SSI due to MRSA has decreased from over 25% of SSI in 2005 to less than 20% in 2007, although there is variation in trend between categories that needs further investigation. However, over the same time period the proportion of SSI caused by MSSA has increased by a similar amount suggesting that MRSA has been replaced as a cause of SSIs by MSSA (figure 11).

Figure 11: Trends in percentage of pathogens associated with SSI, including *S.aureus* (MRSA and MSSA). All SSISS categories. *Source: HPA, Surgical Site Infection Surveillance Service*



These trends in proportion of *S.aureus* causing SSI that are MRSA may be the result of increased detection and decolonisation of patients with MRSA prior to surgery, although such an approach would be expected to also eliminate MSSA. It may therefore reflect more widespread screening/decolonisation reducing the overall prevalence of MRSA colonisation among patients admitted for surgery.

Factors that may have contributed to the reduction in rates of MRSA bacteraemia

There had been a large number of national initiatives that predated the sharp fall in rates of MRSA bacteraemia in 2006, the decline began about 6 months after the launch of Saving Lives and pre-dated the inspections teams. However, it is difficult to draw conclusions about the factors contributing to this decrease given that a wide-ranging set of interventions have been initiated at a national level since 2004 but

implemented at different times and with varying intensity at a local level and with effects that are unlikely to have been immediate. For example, an increase in the screening and decolonisation of MRSA carriers is likely to have taken many months to have an impact on transmission of carriage and subsequent reduction in the number of patients vulnerable to MRSA bacteraemia. In addition, many Trusts implemented their own local strategies. The CHART project undertaken in 75 wards in 24 hospitals found sustained and significant decreases in rates of ward-acquired MRSA that started in early 2004 which was attributed to the impact of the concurrent national CleanYourHands Campaign.[6] The reduction in acquisition would translate over time into a lower prevalence of MRSA colonisation and decreased risk of acquiring endogenous MRSA infection.

The declining trend may reflect change in epidemiology of MRSA. One explanation for the marked increase in rates of bacteraemia caused by MRSA in the late 1990s was the emergence of new epidemic strains of MRSA (EMRSA 15 and 16), which by 2001 were the dominant strains in the UK.[7] The EMRSA 3 and 1 strains that were previously the most prevalent strains were associated with less invasive disease. Since MRSA typing is not undertaken in the majority of cases of infection, and typing undertaken to investigate unusual or severe outbreaks may not be representative of endemic strains, evidence for a changing epidemiology is not currently available.

Since many MRSA bacteraemia may be associated with intravenous devices a possible reason for the decline in rates could be marked improvements in the management of invasive devices. If this was a factor then it would be likely that similar reductions would be seen in both resistant and sensitive strains of *S.aureus*, however, this is not borne out by the trends in routine bacteraemia reports or the mandatory surveillance data on all *S. aureus* available in Scotland which suggest that MSSA bacteraemia are not declining or declining to a lesser extent. The more extensive implementation of screening and decolonisation of patients with MRSA may be an important factor since the decrease in proportion of SSI caused by MRSA since 2006 suggests a general reduction in the number of patients who become colonised with MRSA (and therefore susceptible to MRSA infection).

Other highly resistant bacteria

The importance of microbial resistance to different antimicrobial agents varies according the pathogen and antibiotics of choice for treatment. Data on levels and trends in resistance is available from the voluntary laboratory reporting systems but this data is incomplete, lacks clinical details about the patient and the infection, and does not distinguish between infections acquired in hospital and community settings.

Other more complete data on resistance patterns has been published by the British Society of Antimicrobial Chemotherapy (BSAC) bacteraemia resistance surveillance programme, although this is based on sentinel surveillance in 30 laboratories that are mostly in large or teaching hospitals where a more seriously ill patient population is at increased risk of infection by antimicrobial resistant pathogens.[7] Apart from MRSA bacteraemia the only other pathogens for which data collection is mandatory are bacteraemia caused by enterococci resistant to glycopeptides.

Glycopeptide-resistant enterococcus bacteraemia

Enterococci are part of the normal gut flora but are opportunistic pathogens causing infection in patients hospitalised for prolonged periods that are immunocompromised and have serious underlying illness. There are two main species that cause infections *E. faecalis* or *E. faecium*, with the latter more commonly associated with

hospital outbreaks of resistant strains. Both species are resistant to many groups of antimicrobial agents but in recent years strains of *E. faecium* resistant to vancomycin have emerged. There are limited therapeutic options for these highly resistant strains and controlling their spread in healthcare settings has been seen as a priority. Enterococcal bacteraemia accounts for approximately 7% of all bacteraemia reported via the voluntary laboratory reporting system.

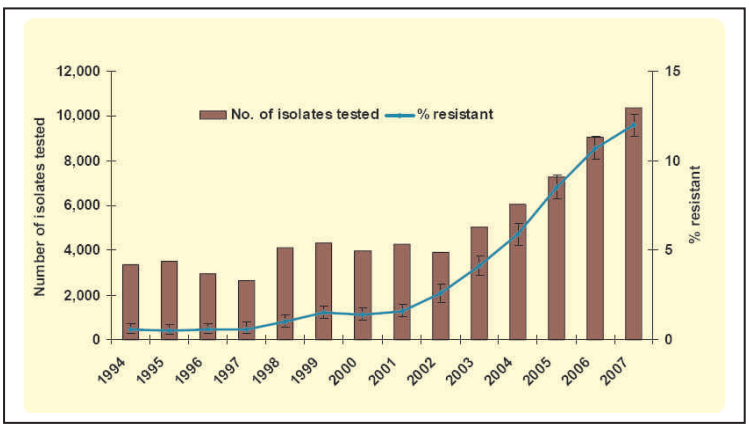
Mandatory surveillance of glycopeptides-resistant enterococci (GRE) bacteraemia was started in October 2003. This requires all Trusts to report the number of blood cultures from which GRE is reported (excluding repeat specimens taken within 14 days) although does not distinguish the two main species of enterococci. The number of reports increased by 30% in the first two years of this surveillance (from 628 in 03/04 to 903 in 05/06). However, the increase stabilised in the last year for which data is available with a similar number of reports in 2006 and 2007. Reports of GRE tend to be localised in a few highly specialist hospitals and the levels of resistance in *E. faecium* of 30% reported in England are much lower than in North America and some European countries.[9]

Trends in antimicrobial resistance in Gram negative bacteria

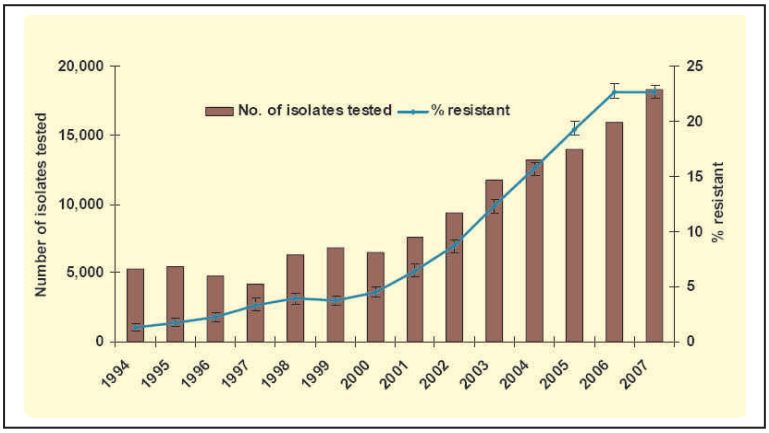
The Enterobacteriaceae are a common cause of bacteraemia, with *Escherichia coli* alone accounting for 20% of reported cases, and other species of *Klebsiella, Proteus* and *Enterobacter* responsible for a further 12%. A recent combined analysis of HPA routine laboratory surveillance and data from the BSAC resistance surveillance programme has shown dramatic increases in resistance in both *E.coli* and *Klebsiella spp.* to a number of key antibiotics: cephalosporins (largely related to extended-spectrum ß-lacatmases), ciprofloxacin and gentamicin. In the case of *E.coli* a combination of the spread of CTX ESBL plasmids among strains and the dissemination of resistant strains has resulted in a major shift in resistance since 2000. The proportion of E. coli resistant to some cephalosporins has increased 6-fold from around 2% in 2000 to over 12% in 2007; during this time the proportion resistant to ciprofloxacin increased 4-fold from around 6% to over 25% (figure 12) and to gentamicin from 1% to 8%. Resistance to ciprofloxacin has also markedly increased in *Serratia* and *Proteus mirabilis*.[10]

Figure 12: Emergence of antimicrobial resistance in *E. coli* from *b*acteraemia in England, Wales and Northern Ireland; 1994-2007 a) cefotaxime b) ciprofloxacin
Source: HPA, voluntary laboratory surveillance (LabBase)[11]

a) Resistance to cefotaxime



b) Resistance to ciprofloxacin



Approximately 40% of *E.coli* and Proteus bacteraemia were reported as associated with urinary tract infections (UTI), commonly in patients in community settings and illustrate the complex relationship between the hospital and community in the emergence of new challenges in HCAI. However, intravenous line-associated infections are also reported as the source of a significant proportion of Enterobacteriaecea bacteraemia. Although carbapenem resistance is currently rare, escalating resistance to cephalosporins and ciprofloxacin in the Enterobacteriaceae is driving an increase in the use of carbapenems and an emergence of carpapenem resistance presents a real threat for the future.[10]

The other main group of Gram-negative pathogens where resistance to antibiotics is a major problem are the non-fermentative bacteria – *Pseudomonas aeruginosa* and *Acinetobacter*. These opportunistic pathogens and are mostly associated with hospitalised patients in intensive care or with serious illness that compromises the immune system. *Ps. aeruginosa* accounts for around 4% of bacteraemia and although it can exhibit resistance to a wide range of antibiotics the BSAC/HPA surveillance programme indicates that resistance remains relatively low and with no evidence of an increasing trend. However, in *Acinetobacter* multi-resistance is widespread and there is evidence of increasing resistance to imipenem, especially in London and the South-East associated with the spread of carbapenemase-carrying strains. *Acinetobacter* accounts for less than 1% of bacteraemia.[12]

These alarming shifts in antimicrobial resistance among Gram negative pathogens since 2000 are of major concern not least because they account for a significant proportion of bacteraemia and are associated with considerable morbidity and mortality.

Gastrointestinal infection

Clostridium difficile

The voluntary laboratory reporting had been showing an increasing trend in reports of *Clostridium difficile* since the mid-1990s and had been increasing by more than 20% annually between 2001 and 2003, although the increase had slowed to 14% in 2004. Whilst these increases have been dramatic, there are a number of factors that could have affected case-ascertainment and suggest that the trends observed should be interpreted with caution. In particular:

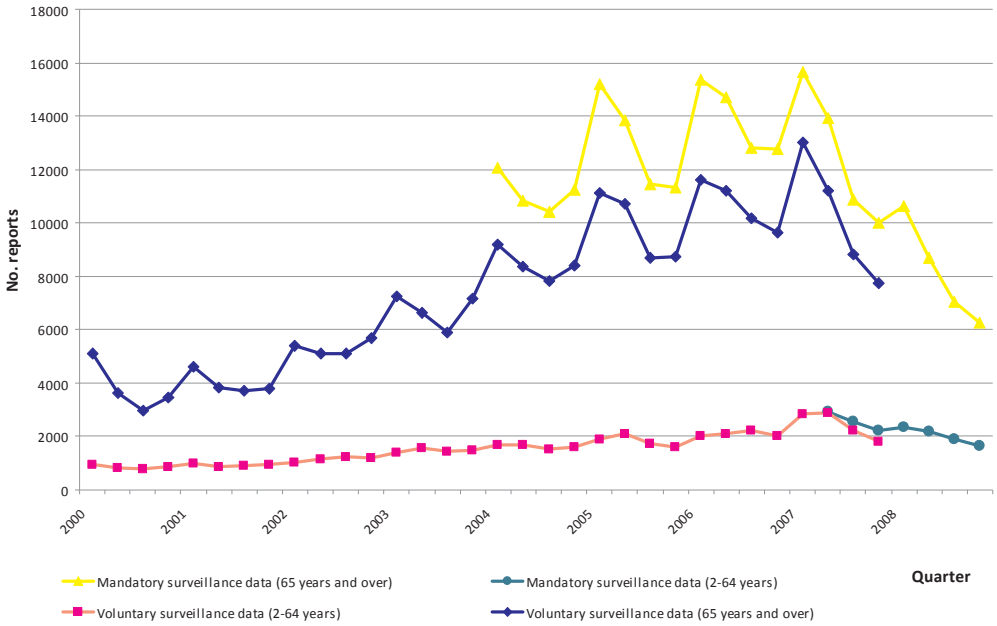
- Prior to April 2004 the counts were based on laboratory reports without defined clinical criteria. When the patient-level mandatory reporting of *C. difficile* disease began in 2004 a case was more clearly defined as a positive test in a liquid stool specimen with guidance that all such specimens should be tested.
- The Health Care Commission survey in 2006 pointed to marked variation in testing practices, with 21% of laboratories not testing all diarrhoeal specimens 20% testing non-diarrhoeal specimens and a quarter not testing community specimens for *C.difficile*. Subsequent re-enforcement of the criteria for testing is likely to have had an effect on the number of reports, particular as it is now recognised that a significant proportion of cases derive from community specimens.[13]
- There have been significant changes in the diagnostic tests for *C.difficile* with a move away from a gold standard cytotoxin assay towards kit-based tests which may be associated with lower sensitivity and specificity. In one recent study, the positive predictive values of 2 commercial assays suggested that less that 60% of specimens testing positive for the disease actually contained *C. difficile* toxin.[14] A recent report by the Purchasing and Supplies Agency confirms that many tests have poor positive predictive values and trends in reports are therefore likely to be influenced by changes in the reliability of tests over time.[15]

In 2004, when mandatory surveillance commenced, there were 44,563 reports of *C. difficile* in patients over 65 years. Subsequent trends in rates of *C.difficile* show a marked seasonal variation, with a peak in the winter months (January to March) in cases reported in patients over 65 years (figure 13). Reasons for this peak are not clear but may be related to increased antimicrobial treatment and admissions to hospital of the elderly with lower respiratory tract infections or increased incidence of norovirus resulting in increased detection of *C.difficile* toxin and/or increased susceptibility to, and transmission from, patients with gastroenteritis.[16] The peak in counts was much less pronounced in January to March 2008 and since April 2008 a higher proportion of cases have been attributed to patients in acute Trusts (54.5%) although this may reflect a change in approach to how this classification is made at this time.

Initially surveillance was focused on toxin-positive reports in patients aged 65 years and over who had not been diagnosed with *C.difficile* infection in the preceding 4 weeks. In April 2007 the instructions were amended to include toxin-positive reports in patients aged 2 or more, excluding repeat results within the same admission episode. Then in January 2008, this was adjusted again to define positive results on the same patient taken within 28 days of the first specimen as a single episode. Between April 2006 and March 2008 the evidence from the PCR ribotyping random sampling scheme indicated that the proportion of *C.difficile* caused by Type 027 strains has increased from 26% to 41%.[17] This strain has been associated with more

severe disease and its emergence may have contributed to the increase in number of reports and cases in younger age groups.[18] However, *C.difficile* remains largely a disease afflicting the elderly. In data collected between April 07 and March 08 82% of reports were from people aged over 65 and 64% from people aged 75 years or more. Nearly 60% of reports were from females.

Figure 13: Trends in number of reports of *C.difficile* toxin positive cultures
Source: HCAI Surveillance System

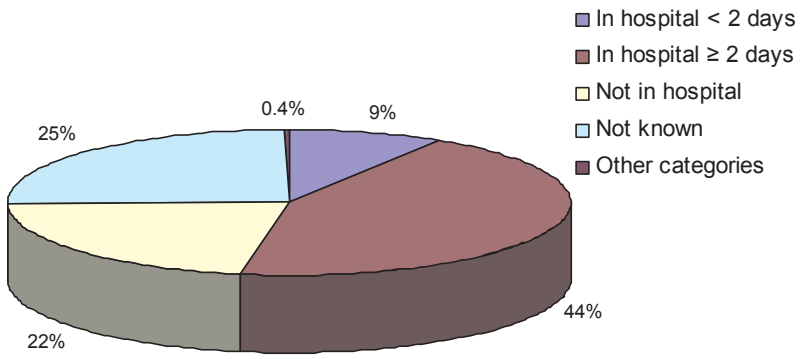


Whilst changes to the definition of a single episode of *C. difficile* infection made between April 2004 and January 2008 are likely to have affected the number of cases reported such changes are unlikely to explain the 41% decrease in the number *C. difficile* infections reported between the years 2006 and 2008.

Community versus hospital presentation

Between April 2007 and March 2008 44% of all reported *C. difficile* infections were detected in specimens taken two or more days after admission to the reporting Trust. This suggests that the infections were likely to have been acquired during that hospital admission, although delays in recognising symptoms or taking specimens may mean that some of these patients were admitted with the infection. In 31% of reported cases the specimen was taken from a patient who had not been admitted to an acute hospital or had been in one less than 2 days. Whilst these patients may have acquired the infection in a community setting, some may have acquired the organism during an earlier admission and developed symptoms after discharge.

Figure 14: Distribution of *C.difficile* reports by location of patient when specimen taken (data collected between April 2007 and March 2008).
Source: HCAI Surveillance System



Changes to the definition of hospital and community-associated cases were made in April 2008 and it is now assumed that patients who have been in hospital less than 3 days (including the day of admission) have not acquired the infection in the hospital. Using this definition approximately 55% of *C.difficile* cases reported between April and September 2008 appear to be hospital-acquired. T his again highlights the complexity of factors that influence the acquisition of HCAI and indicates the importance of strategies focused on community settings in the prevention and control of *C.difficile* infection.

The difficulty of distinguishing between community and hospital acquired infection means that calculating rates of *C.difficile* disease by acute OBD would be misleading since this implies that all cases are associated with the hospital in which the infection is detected. One study provides evidence that 2% of patients in the community who had faecal specimens taken by their GP are positive for *C.difficile toxin;* of these cases a third had not received antibiotics or had contact with hospital.[19] Other studies using different definitions and case-finding methods have reported much higher proportions of community-associated cases with up to 20% of elderly hospitalised patients carrying *C. difficile* asymptomatically.[20]

Variation between regions

Rates of *C.difficile* disease by 1000 occupied bed days have been reported and suggest marked variation between region. However, these differences are highly misleading as many of these *C.difficile* infections are not associated with acute healthcare and the proportion that are community associated varies markedly between regions. For example, in London region only 15% of specimens were from non-acute locations (GP, residential/nursing homes, primary care Trust hospitals) compared to over 20% in most other regions. These differences probably reflect the population age structure and the mix of Trust types with teaching hospitals also reporting a lower proportion of non-acute specimens (see figure 7).[21] Variation between Trust type, although less marked is difficult to interpret for the above reasons. Rates of *C. difficile* may also be influenced by prevalent strain types as data from the C.difficile Ribotyping Network for England (CDRNE) indicates marked difference in strain types between regions.[17]

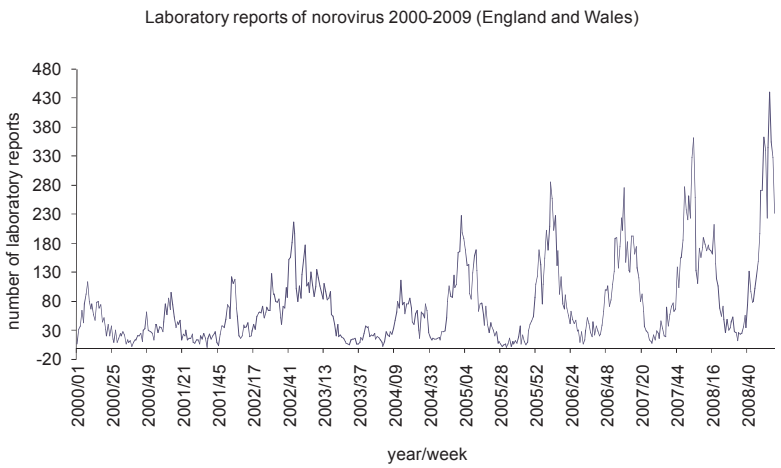
Surveillance data, whilst useful in defining the incidence of disease, does not enable the risk factors contributing to the acquisition of infection to be easily determined. In a case control study of community-associated *C. difficile*[19] use of antibiotics,

especially multiple antibiotics, hospitalisation in the preceding 6 months and exposure to infants of 2 or under were significantly associated with *C. difficile* infection. This suggests a complex picture in which it is difficult to determine where and when the *C. difficile* was acquired in the gut, when and why colonisation became infection, and indeed whether the detection of toxin equates with *C. difficile* disease or reflects gastrointestinal disease caused by norovirus in a patients colonised with *C. difficile.*[22] Whereabouts in the healthcare economy infection prevention strategies should be targeted is therefore difficult to determine, however it seems likely that the implementation of measures directed at antimicrobial prescribing practice, together with improvements in the detection and management of cases of *C. difficile* disease across acute and primary care settings has had an impact on the number of cases reported since 2007.

Norovirus

Norovirus causes an acute, but relatively mild and self-limiting gastroenteritis in both community and healthcare associated settings; transmission is foodborne or person-to-person; and many cases are not confirmed by a laboratory diagnosis. Outbreaks occur more frequently in the winter months and are particularly common in hospitals or nursing homes. New variants of the stain which emerge every few years are associated with an increase in cases. Since 2002 a new variant has been reported as causing large increases in reported outbreaks of norovirus in many European countries, particularly in the hospitalised elderly.[23] It has been suggested that symptoms caused by norovirus may explain the increased detection of *C.difficile* toxin and certainly the winter peak of reports of norovirus coincides with the peak in reports of *C.difficile* toxin. In addition, the marked increase in reports of *C.difficile* coincided with the increase in outbreaks of norvirus associated with the emergence of the new strain. However, the sharp decline in reports of *C.difficile* since July 2007 and minimal peak in cases over the winter of 2008 do not appear to have been accompanied by a similar decline in reports of norovirus over the same period, although these reports do not distinguish hospitals and community acquired cases (figure 15). The hospital outbreak reporting system launched in January 2008 will help to delineate hospital-acquired norovirus.

Figure 15: Laboratory report of norovirus

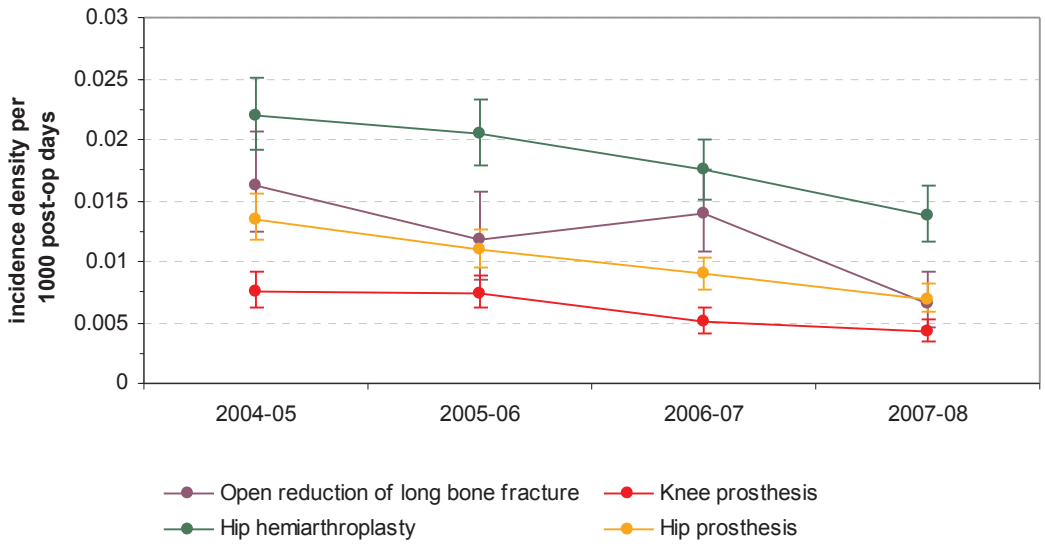


Surgical site infection

The surveillance of surgical site infection (SSI) following orthopaedic surgery became mandatory in April 2004, although Trusts were not required to undertake continuous surveillance, only a minimum of one quarter in at least one of fours categories of procedure: hip replacement, knee replacement, hip hemiarthroplasty or open reduction of long bone fracture. Since 2004 the overall cumulative incidence rate of SSI in these categories has declined significantly from 1.44% to 0.6%. However, since the surveillance has been focused on the inpatient stay and the median length of stay has reduced from 7 days in 2004 to 5 days in 2007/8, this decline in rate could be explained by an increase in the proportion of SSI missed by the surveillance. In addition, SSI are complicated to identify as they are defined by clinical signs rather that positive laboratory reports and therefore it is possible that the observed trend in rates may have been influenced by systematic changes in case-ascertainment.

The effect of changes in length of postoperative stay can, at least in part, be adjusted for by calculating the rate of SSI as an incidence density of SSI. Figure 15 indicates statistically significant reductions in incidence density rates for hip and knee prosthesis and hip hemiarthroplasty since the mandatory surveillance commenced in 2004. More sophisticated small area estimation methods that take account of inter-hospital variation in length of post operative stay over time also suggest that statistically significant reductions in rates of SSI have occurred since 2004.[24]

Figure 14: Incidence density of SSI following orthopaedic surgery by 1000 post-operative inpatient days *Source: HPA, Surgical Site Infection Surveillance Service*



Although the trends in incidence density of SSI have also decreased in some other, non-mandatory categories of SSI these are more difficult to interpret because the numbers of participating hospitals are relatively small and vary over time. The marked reductions in length of post-operative stay highlight the need to establish robust systems of detecting SSI that become apparent after the patient has been discharged from hospital. From July 2008 hospitals have been asked to establish systems to identify patients readmitted with SSI and rates will be adjusted in future to include these infections. This will help to provide a more accurate picture of rates of SSI following surgery. Changes to the data capture system to enable hospitals to more easily report and generate rates that include SSI detected by others methods of

post-discharge were also made in July 2008 and will provide more complete data on the incidence of SSI.

Although surveillance of SSI has been mandatory for orthopaedic surgery it has not been the focus of attention either in respect of national infection control initiatives driven by the Department of Health or setting of targets for reductions in rates. The significant reductions in rates of SSI that have been observed since 2004 have therefore occurred solely in the context of mandation of surveillance; the standard approach of feedback of surveillance results to individual hospitals enabling them to compare their rates with a benchmark rate; and the notification by SSISS of hospitals identified to have a rate above the 90th percentile compared to other participating hospitals. The decrease in proportion of SSI caused by MRSA may have occurred as a result of the initiatives targeted at MRSA bacteraemia, although the data suggests they may have been replaced by MSSA (figure 11).

Mortality data

The Office for National Statistics (ONS) has reported trends in deaths that involved MRSA and *C. difficile.*[25,26] The data they report is based on death certificates where MRSA or *C. difficile* are mentioned as the underlying cause of death (the disease which initiated the train of events leading directly to death) and certificates where the infection is designated as contributory factor to the death. Trends in mortality must be interpreted with caution as the increased public profile of HCAI may have affected the likelihood of their entry onto death certificates. In addition, in both 2005 and 2007 the ONS issued guidance reinforcing the importance of including HCAI on death certificates, subsequently disseminated by the Chief Medical Officer to all registered doctors, and this is likely to have had an impact on the number reported.

In the case of MRSA, there is not a specific code that can be used to classify the infection and potential MRSA-related deaths must therefore be identified using *S. aureus* and infection codes combined with manual searching. The most recent ONS report indicates that in England the number of reports where MRSA was mentioned as a contributory factor or underlying cause increased by 73% between 2003 and 2006 (from 903 to 1556), with most of that rise occurring after the reissue of the guidance in 2005. However the number of reports declined by 5% in 2007. Overall MRSA related deaths account for approximately 0.3% of all deaths (based on deaths reported between 2003 and 2007). MRSA accounts for 74% of all *S.aureus* mentions as underlying cause of death. It is possible that these data overestimate the impact of MRSA as a cause of death since MRSA account for 78% of all *S. aureus* mentions in death certificate, more than two and half times the proportion of *S. aureus* bacteraemia reported as methicillin-resistant. If the proportion of *S.aureus* that are MRSA is assumed to be 35% then the number death certificates mentioning MRSA would suggest the risk of death associated with MRSA was 7 times greater than MSSA. This conflicts with evidence from the literature which indicates the odds ratio is closer to 2[27] and suggests that either deaths associated with MRSA are overreported or those associated with MSSA are underreported. Furthermore, a recent qualitative review of MRSA deaths identified substantial over-reporting (and under-reporting) of MRSA on death certificates.[28]

As expected the mortality rates increase markedly with age as older patients are both more likely to be colonised with MRSA, have severe underlying illness and require treatments that increase their susceptibility to infection. Evidence from the recent qualitative study of factors contributing to MRSA related deaths suggested that three-quarters of patients who die with MRSA bacteraemia had an anticipated life-

expectance of less than 12 months.[28] Although the number of reports of deaths related to MRSA in males is twice that of females, this difference also exists in all *S.aureus* and probably reflects risk factors for infection by *S.aureus* related to differences in underlying illness that affect males and female differently e.g. vascular disease.

Registrations of deaths related to *C.difficile* occur in far greater numbers than MRSA and are reported in association with 0.9% of all deaths in England (based on deaths reported between 2003 and 2007), although in only half of these is it cited as the underlying cause.[26] As with MRSA, the number of deaths related to *C.difficile* increases markedly with age similarly reflecting increased susceptibility to colonisation and infection in the elderly. Reports have increased by 360% (from 1720 to 7916) between 2003 and 2007 and are probably less prone to variation in reporting than MRSA as they can be described using a specific ICD10 code. However, whilst the annual increase of reports of *C.difficile* as underlying cause of death was 70% between 2004 and 2006, reports as an underlying cause increased by only 14% in 2007. These trends need to be seen in the context of marked increases in detection of cases as a result of the mandatory surveillance introduced in 2004, other factors that have influenced case-ascertainment described above, and the guidance reinforcing the importance of documenting HCAI on death certificates.

Prevalence of HCAI

The surveillance data described above is focused on specific pathogens associated with HCAI or specific types of infection. Prevalence surveys provide a useful approach for determining the overall burden of disease and relative importance of different types of infection. A United Kingdom prevalence survey undertaken between May 1993 and July 1994 in 157 hospitals and found a HCAI prevalence of 9%.[29] In 2006, 270 hospitals in England, Wales, Northern Ireland and Republic of Ireland participated in a similar survey between February and May and identified an overall prevalence of HCAI of 7.6%.[1] Although this survey reported differences in rates between the four countries, the analysis does not take into account the multi-centre nature of the study and therefore only the overall prevalence is shown in table 1.[30]

Table 1: Comparison between 1993/4 and 2006 UK prevalence surveys

	1993/4		2006	
	Prevalence	**% of HCAI**	**Prevalence**	**% of HCAI**
Urinary tract	2.4	23	1.7	20
Lower respiratory tract	2.4	22	1.2	14
○ not pneumonia			0.5	6
Gastrointestinal	0.5	5	1.7	21*
Surgical site	1.1	11	1.2	15
○ *in patients who had surgery*			*4.7*	
Bloodstream	0.7	6	0.6	7[+]
Skin & soft tissue	1.0	10	0.9	10
Other		23		7
Overall	9.0	100	7.6	100

Note: Prevalence = proportion of total patients in survey with HCAI. For surgical site infection the prevalence in only those patients who had undergone a surgical procedure is also shown.

*70% of gastrointestinal infections = *Clostridium difficile;* [+]21% of bloodstream infections = MRSA

What is the evidence for changes in prevalence of HCAI (in England) between 2004 and 2008?

Differences in the definitions of HCAI, survey methods and sampling strategy mean that the results of the 2006 survey are not readily comparable with previous national prevalence surveys in the UK. In addition, the gradual decrease in the length of hospital stay over the last decade is likely to have affected the number of HCAI detected in these two prevalence surveys. The 2006 survey does suggest a marked increase in patients with gastrointestinal infections compared to the previous survey that may be attributable to an increase in cases of C*. difficile.* However, the increase in detection of *C.difficile* brought about by mandatory surveillance and changes in testing methods will have had a marked affect on case-ascertainment and the specific requirement to collect additional data on *C. difficile* in the 2006 survey may also have had an effect on detection and reporting. Differences in prevalence of pneumonia and UTI between the two surveys probably reflect variation in the case definitions, which in 1993/4 were based on a simpler set of criteria. The prevalence of SSI, skin and soft tissue infections and bacteraemia remain similar in both surveys.

Relative importance of different HCAI

These prevalence surveys illustrate the relative importance of different HCAI. They indicate that bloodstream infections are relatively uncommon, but lower respiratory tract, urinary tract and SSI account for more than 50% of all HCAI. Since SSIs affect only those patients who undergo surgery, their prevalence among those patients at risk is nearly 5%. In addition, this survey indicates the extent to which national reporting of MRSA infections based only on bacteraemia does not reflect the true extent of MRSA infection. The prevalence of SSI due to MRSA was found to be 1% (in patients who had undergone surgery) and 0.4% in skin and soft tissue infections compared to 0.1% in primary bacteraemia.

A key factor in determining the risk of HCAI is the underlying health of the patient receiving treatment. Thus, in the 2006 survey the prevalence of HCAI in critical care medicine (23%) is three times that of patients in general medicine (8%) and variation between healthcare facilities in rates of HCAI, both overall and those caused by specific pathogens such as MRSA, is influenced by the size and complexity of the services provided by the hospital and hence the underlying illnesses and their associated treatments in the patients admitted to them.

Currently, there are no national surveillance systems that capture data on some of the most common HCAIs identified in the prevalence surveys – UTI, pneumonia and skin and soft tissue infection, only limited surveillance focused on SSI, and surveillance of bacteraemia is mostly targeted at single pathogens. There is also no national data available on HCAI in critical care settings.

Conclusions

HCAIs are a complex problem with a multiplicity of causes related to healthcare delivered in both hospital and community settings. Whilst some HCAI, such as SSI and ventilator associated pneumonia, are clearly linked to the delivery of care in a hospital setting, in others such as *C. difficile* and some antimicrobial resistant pathogens a direct link to hospital-based care cannot be assumed. Over the past few years there has been mounting evidence of significant reductions in the incidence of particular HCAI's, specifically MRSA bacteraemia, rates of SSI in hip and knee prosthesis surgery, and *C. difficile*. These have all been the focus of active,

mandatory surveillance. Unfortunately, there is no national surveillance data to demonstrate whether similar declines are occurring in other types of HCAI, and some evidence from voluntary surveillance of bacteraemia that rates of these infections are increasing.

There has been a marked shift from MRSA to MSSA as the causal pathogen for SSI in hip and knee prosthesis surgery. There has been an impressive reduction in MRSA bacteraemia by 58% in April 2008 against the 2003/04 baseline, however there is evidence that of rates of MSSA bacteraemia have not declined over this time period. Indeed, the rate of healthcare-associated bacteraemia caused by all pathogens has probably increased over this period with other pathogens, in particular Gram negative bacteria, emerging as more frequent causes of infection. The reasons for the decline in MRSA as a cause of bacteraemia and SSI maybe linked to the general effect of the CleanYourHands campaign and subsequent programme of national initiatives targeted at MRSA, but the effectiveness of specific interventions is difficult to determine.

Whilst extensive data is available on bacteraemias, these only account for 7% of all HCAI and other more prevalent infections are associated with significant morbidity and mortality, and increasing antimicrobial resistance, for example extended spectrum β-lactamases associated with UTI in catheterised patients. In addition, MRSA currently accounts for only 4% of all bacteraemias whereas bacteraemia caused by Gram negative pathogens such as *E.coli* and *Klebsiella* have increased steadily over the last 5 years and now account for nearly a third of all bacteraemias reported. Over this same period there has been a dramatic increase in resistance to key antimicrobial agents in these, and other Gram-negative pathogens, that present considerable challenges to their prevention and control. In addition, there is evidence of significant increases in bacteraemia caused by coagulase-negative staphylococci, the reasons for which require investigation. These trends may reflect increasing severity of illness in patients receiving healthcare, however more data is required to better understand the factors that contribute to them and the extent to which they can be prevented.

Reports of *C. difficile* increased markedly between 2002 and 2007, although many factors influenced case ascertainment during this time. Since 2008 there has been a dramatic decline in reports. The extent to which *C. difficile* is acquired in hospital is difficult to determine as approximately half of *C. difficile* reports occur in people who are not in acute healthcare settings, and some of these have no recent history of hospital contact. More data is required to explore the factors that have contributed to this decline and relative effect of control measures in hospital and community settings. Recent trends in *C. difficile* reports have not been matched by those of norovirus where the numbers of reports have continued to increase year on year with more outbreaks in hospital settings associated with the emergence of a new strain in 2004, although some of this increase may be due to increased reporting.

Registrations of deaths related to HCAI are misleading as they have focused primarily on MRSA and *C. difficile* and do not therefore take into account the wider context of all HCAI. The trends in death registrations of MRSA and *C. difficile* have mirrored the trends reported by the mandatory surveillance system and should be interpreted with caution as the recent prominent media profile and additional specific guidance regarding their certification is likely to have increased the likelihood that they will be mentioned on death certificates.

Whilst it might be expected that initiatives targeted at preventing MRSA bacteraemia will have a wider impact on other HCAI the marked increase in reports of some

pathogens causing bacteraemia suggest that this may not be the case. If anything, the intensive focus on MRSA bacteraemia and *C. difficile* as targets for performance indicators is likely to reduce the priority afforded to other endemic or emerging infections. Highly resistant pathogens such as ESBLs may not be seen as taking precedence over MRSA and *C.difficile* when allocating patients to single rooms or implementing other infection prevention strategies. In addition, since there is a widespread perception among both the public and healthcare workers that HCAIs equate to MRSA and *C. difficile,* there is a danger that the importance of infection control strategies aimed at preventing other HCAI that are associated with considerable morbidity and mortality such as urinary tract infections, ventilator-associated pneumonia and surgical site infections are not recognised.[31,32]

The essential role that surveillance plays in detecting and monitoring key changes in the occurrence and epidemiology of infections that arise in healthcare is without question. However, this review highlights the need for such surveillance to encompass the broad range and complex nature of HCAI and the modern healthcare economy. By focusing on single pathogens at a hospital level there is a significant danger that important trends in other pathogens or types of infection will be overlooked or neglected and that infection prevention activity may be misdirected. In addition, it should be recognised that surveillance by its nature can only provide information for action and that well-designed epidemiological studies are required in order to enhance our understanding of the factors underlying trends in HCAIs and the most effective strategies for their prevention.

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Appendix 1: Summary of available data sources on HCAI

Routine laboratory data

- Data on micro-organisms causing significant clinical infections have been reported to the HPA by many laboratories since the 1970s, with data on C.diff collected since 1990.
- Reports of laboratory-confirmed norovirus gastroenteritis are available from this database, although these data do not distinguish between community and hospital associated cases.
- Data is extracted as 'single patient episodes' and repeat specimens with the same pathogen from the same patient not excluded.
- The limitations of this data are that the trends are vulnerable to variation in case-ascertainment over time. In particular, there has been a steady rise in the number of laboratories reporting since 1990 and the gradual move towards automatic download of data from pathology computer systems has increased the number of reports. However, there is still variation in the consistency and completeness of reporting from some labs.

MRSA bacteraemia enhanced surveillance system (MESS)

- Established in October 2005 to extend the dataset captured by the HPA as part of the mandatory surveillance initiated by the Department of Health in 2001. This dataset includes demographic data about the patient, their treatment speciality and their location at the time the specimen was taken.
- This numerator data is combined with a Trust level denominator of average daily bed occupancy (derived from HES KHO3 data) to calculate a rate per 10 000 occupied bed-days that is intended to reflect Trust activity.
- This surveillance captures data on: *'all MRSA positive blood cultures detected in the laboratories, whether clinically significant or not, whether treated or not, whether acquired in the Trust or elsewhere.'*
- Repeat reports in the same patient within 14 days are not included but those after 14 days should be reported as a new episode.
- A further dataset on patients in established renal failure who develop MRSA bacteraemia is also collected on a voluntary basis and provide some data on risk factors for bacteraemia in these patients such as type of renal access.

Glycopeptide resistant bacteraemia

- Number of reports submitted quarterly to HPA and published by acute Trust
- Repeat reports in the same patient within 14 days are not included but those after 14 days should be reported as a new episode.

C.difficile

- The mandatory surveillance of *C.difficile* was introduced in January 2004 for patients aged 65 years or more and initially collected the aggregate number of reports of *C.difficile* toxin-positive liquid stool specimens by acute NHS Trust.
- Repeat reports in the same patient are now excluded if taken within 28 days of the first positive report (although between April 2007 and Jan 2008 repeat specimens only related to those take n in the admission period).
- In April 2007 the reporting was changed to an enhanced system capturing data on individual patients aged 2 years or more with a report of *C.difficile* toxin-positive liquid stool specimen. The dataset included demographic data and data on treatment specialty and location of the patient when specimen taken.
- In January 2008 the date of admission and NHS number became a mandatory part of the dataset to enable cases of community and hospital origin to be distinguished and allocated to a primary care organisation.
- Data on epidemiological types and antimicrobial sensitivities of *C.difficile* are can be collected but are not mandatory.

Surgical site infections

- A standard dataset of demographic and operation data is collected on individual patients at risks of SSI within defined categories of surgical procedure and these patients then followed up during their in-patient stay to identify those that meet a standard case-definition of SSI.
- Rates are generally calculated as cumulative incidence (% of operation with SSI), however, for comparison between hospitals and for evaluation of trends over time a rate per 1000 post-operative days of follow-up (incidence density) is used in order to adjust for variation in length of post-operative stay.
- The mandatory requirement to undertake a minimum of 3 months surveillance in at least one category of four orthopaedic procedures (hip replacement, knee replacement, hip hemiarthroplasty and open reduction of long bone fracture) in each financial year.
- A web-based data capture system was introduced in April 2004 and upgraded in July 2008
- A standard approach to post-discharge surveillance was added to the SSI surveillance protocol in July 2008, but apart from reporting SSI in patients' readmitted to hospitals, PDS remains voluntary.

British Society of Antimicrobial Chemotherapy Surveillance Project

- Sentinel laboratories across the UK and Ireland contributed up to a fixed quota of isolates of defined bacterial groups. A central laboratory for each programme confirmed the identification of isolates, measured MICs by the BSAC agar dilution method and undertook further testing by standard methods.
- Isolates were classified as susceptible, intermediate or resistant was by BSAC and European Committee on Antimicrobial Susceptibility Testing breakpoints.
- 30 laboratories contributed 15 812 bacteraemia isolates from 2001 to 2006. Although large and teaching hospitals were over-represented, the pattern of bacteraemia organisms seen in the collecting laboratories in England and Wales was similar to that in national data reported to the Health Protection Agency.
- The distribution results from the sentinel laboratories was compared with the overall national distribution of clinically significant bacteraemias reported to the HPA LabBase/CoSurvsystem2 throughout the BSAC surveillance period.