Neonatal infections in England: the NeonIN surveillance network

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ABSTRACT

Introduction Neonatal infection is an important cause of morbidity and mortality. Neonatal infection surveillance networks are necessary for defining the epidemiology of infections and monitoring changes over time.

Design Prospective multicentre surveillance using a web-based database.

Setting 12 English neonatal units.

Participants Newborns admitted in 2006–2008, with positive blood, cerebrospinal fluid or urine culture and treated with antibiotics for at least 5 days.

Outcome measure Incidence, age at infection, pathogens and antibiotic resistance profiles.

Results With the inclusion of coagulase negative Staphylococcus (CoNS), the incidence of all neonatal infection was 8/1000 live births and 71/1000 neonatal admissions (2007–2008). The majority of infections occurred in premature (<37 weeks) and low birthweight (<2500 g) infants (82% and 81%, respectively). The incidence of early onset sepsis (EOS; ≤48 h of age) was 0.9/1000 live births and 9/1000 neonatal admissions, and group B Streptococcus (58%) and Escherichia coli (18%) were the most common organisms. The incidence of late onset sepsis (LOS; >48 h of age) was 3/1000 live births and 29/1000 neonatal admissions including CoNS and the most common organisms were CoNS (54%), Enterobacteriaceae (21%) and Staphylococcus aureus (18%, 11% of which were methicillin resistant S aureus). Fungi accounted for 9% of LOS (72% Candida albicans). The majority of pathogens causing EOS (95%) and LOS (84%) were susceptible to commonly used empiric first line antibiotic combinations of penicillin/gentamycin and flucloxacinil/gentamicin, respectively (excluding CoNS).

Conclusions The authors have established NeonIN in England and defined the current epidemiology of neonatal infections. These data can be used for benchmarking among units, international comparisons and as a platform for interventional studies.

INTRODUCTION

Neonatal infection is an important cause of morbidity, prolonged hospital stay and mortality among infants, particularly those born preterm and of very low birthweight (VLBW).1 Pathogens causing neonatal infections and their antibiotic susceptibility patterns may change over time2–4 and differ between countries.5 It is therefore essential to monitor the epidemiology of neonatal infections to inform policy and clinical practice.

Neonatal infection surveillance networks have been established in a number of countries, and include the Australasian Study Group for Neonatal Infections (ASGNI),6 the Israeli National VLBW Infant Database,7 the Canadian Neonatal Network (http://www.canadianneonatalnetwork.org), the US National Institute of Child Health and Human Development (NICHD) Neonatal Research Network (http://www.nichd.nih.gov), the Vermont Oxford Network (https://www.vtoxford.org/home.aspx) and the German Krankenhaus-Infektions-Surveillance-System (NeoKISS).8 With the exceptions of the ASGNI and Canadian groups, existing neonatal infection networks focus on VLBW babies cared for in tertiary neonatal units. A variety of data are collected on culture proven sepsis, as well as presumed and possible sepsis.8 Definitions of sepsis vary between networks, but all consist of a combination of clinical and laboratory parameters.3 Neonatal infection networks have been useful for benchmarking practice,3 as a platform for interventional studies10 and for monitoring changes in

What is already known on this topic

► Neonatal infections are an important cause of morbidity and mortality, particularly in very low birthweight infants.

► Neonatal infection surveillance networks have been established in several countries and are useful for documenting changes in clinical practice, benchmarking, monitoring changes in pathogens and their antibiotic resistance over time, informing policy and improving quality of care.

What this study adds

► The neonatal infection surveillance network (NeonIN) monitors the epidemiology and empiric antibiotic treatment of neonatal infections.

► Penicillin and gentamicin in combination cover 95% of early onset pathogens; flucloxacinil and gentamicin cover most late onset pathogens.

► Compared with the USA, group B Streptococcus causes a higher proportion and Escherichia coli a lower proportion of early onset infections.

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METHODS
An episode of neonatal infection was defined as a positive culture collected from a normally sterile site (blood, cerebrospinal fluid [CSF] and suprapubic aspirate) for which clinicians prescribed at least 5 days of antibiotic treatment. Repeatedly positive samples with the same organism were considered as part of the same episode if the period between samples was less than 7 days for all organisms, other than coagulase negative Staphylococcus (CoNS) and fungal infections, for which the specified period was 10 days. Early onset sepsis (EOS) was defined as infection in the first 48 h of life and late onset sepsis (LOS) as infection occurring thereafter. This age cut-off was chosen as it probably best reflects the transition between infections caused by pathogens acquired by vertical transmission and those acquired by horizontal transmission, and because it has also been used by other surveillance networks. However, the data were collected such that other time points could also be applied (see online supplementary tables 1–3). Small for gestational age (SGA) was defined as birthweight <10th centile for gestational age.

Because bacteraemia with CoNS may indicate a true infection or may reflect contamination, there is debate about its inclusion in infection surveillance data. From 2007 onwards we elected to include CoNS bacteraemia if the episode was treated for a minimum of 5 days with appropriate antibiotics (i.e., generally vancomycin), a definition consistent with that used by other networks (http://www.nichd.nih.gov; https://www.vtoxford.org/home.aspx).

A standardised proforma was completed by the clinician for each positive culture, and included the infant’s demographic details (birth weight, gestational age, gender), clinical information and microbiological data, as well as the antibiotic resistance profile of the isolate. Currently most microbiology units use automated antibiotic sensitivity testing techniques such as VITEK 2. However, as the database is not a suitable mechanism for longitudinal surveillance. Additionally, the Health Protection Agency (HPA) routinely collects data on bacteraemias in England and Wales, but it captures only limited clinical data. In 2004 NeonIN was established with the objective of collecting clinical and microbiological data on episodes of neonatal infection on a longitudinal basis. Currently, 16 units are contributing data from across England. We report the incidence, pathogens and antibiotic resistance profiles of infections captured in NeonIN in the 3 years between 1 January 2006 and 31 December 2008.

RESULTS
All infections for 2006–2008 (excluding CoNS)
Data were available for eight units in 2006, nine in 2007 and 12 in 2008. A total of 541 episodes of neonatal infection (representing 443 infants) were recorded. Of the participating units, two were level 2, and 10 were level 3; 50% of the units managed infants with surgical conditions (general, abdominal and cardiac surgery). Infection was more common in male (n=247, 56%) than in female infants (n=196, 46%; p=0.0001). The majority of episodes of infection (n=443, 82%) occurred in infants born at <37 weeks gestation, of which 71% (n=313) occurred in infants ≤32 weeks gestation. Similarly, 81% of episodes occurred in infants with a birth weight <2500 g (n=457), of which 53% (n=237) occurred in infants <1000 g, 18% (n=81) of infants were SGA, 9% (n=51) had more than one episode of sepsis, and 3% (n=16) had more than one pathogen cultured during the same episode.

While most episodes were diagnosed through positive blood cultures (505, 93%), pathogens were isolated from the CSF in 505, 93%, pathogens were isolated from the CSF in 279, 5% of episodes. Eight of the latter were late onset, with a median age of infection of 17 days (range 6–92 days). The pathogens responsible for late onset meningitis were GBS, Enterobacter cloacae (two), Escherichia coli (two), Citrobacter, Streptococcus viridans and Enterococcus faecalis. The early onset blood culture negative meningitis was caused by Candida albicans.

The remaining isolates were obtained from urine (27, 5%). All urine infections were late onset (median 19 days, range 3–279 days).

The incidence of neonatal infection was 4.1 per 1000 live births (541/130,763) and 38 per 1000 neonatal admissions (541/14,225) and was similar across the 3 years of surveillance (p>0.1). The distribution of infections was also comparable for birth weight and gestational age categories. However, in 2008 there was a smaller proportion of infection episodes in infants over 2500 g and a larger proportion in extremely premature infants (≤32 weeks gestation) compared to previous years (p<0.001 and p=0.002, respectively; table 1).

Early onset sepsis (<48 h of age)
EOS incidence was 0.9 per 1000 live births and 9 per 1000 neonatal admissions and was similar for the 3 years of surveillance (p>0.1).
Of the 125 episodes of EOS, 74% (93) were due to gram-positive organisms, 25% (31) to gram-negative organisms and one to \textit{C albicans} (table 2); GBS was the most frequent organism isolated. The incidence of early onset GBS was not significantly different over the 3-year period, averaging 0.5 per 1000 live births (65/130,763). The majority of organisms (95%) were susceptible to the two most commonly used empiric antibiotic combinations. The major exception to this was \textit{S aureus}, all of which were resistant to the benzylpenicillin and gentamicin combination.

**Late onset sepsis (>48 h of age)**

The incidence of LOS in each of the 3 years of surveillance was similar (overall 3/1000 live births and 29/1000 neonatal admissions, n=416; p>0.1 for comparison between years). The median age at infection was 18 days (range 3–386 days) and the majority of episodes (70%) occurred in extremely premature infants (85% were <37 weeks gestation). Overall, gram-positive organisms accounted for 49% (203/416), gram-negative for 42% (177/416) and fungi for the remaining 9% (36/416) of episodes. The predominant fungus isolated was \textit{C albicans} (72%) and the proportion of VLBW infants with one of more episodes of \textit{Candida} sepsis was 1.8% in 2006, 1.2% in 2007 and 1.8% in 2008.

The most common pathogens causing LOS are listed in table 2. With the exclusion of CoNS, the majority of isolates were susceptible to the two most commonly used empiric antibiotic combinations, although susceptibility to flucloxacillin and gentamicin was higher (84%) than to amoxicillin and cefotaxime (79%) due to the relatively high resistance rate of \textit{Enterobacteriaceae} to cephalosporins (table 3).

**CoNS infections (2007–2008)**

There were 358 episodes of CoNS sepsis in 321 infants (166 females and 155 males) in 2007–2008, increasing the total incidence for all infections to 8/1000 live births (728/91,243) and 71/1000 neonatal admissions (728/10,315).

CoNS were associated with 21 episodes of EOS (representing 20% of all EOS) and 337 episodes of LOS (representing 54% of all LOS). The majority of CoNS infections were in infants ≤32 weeks gestation (507, 86%) and in extremely low birthweight infants (234, 65%).

**Age at disease by pathogen (2007–2008)**

Almost a quarter (23%) of all neonatal infections occurred in the first 48 h of life (early onset) and the majority of these occurred on the first day (88%). Using 72 h as the threshold for early onset infections increased the percentage of early onset infections to 26% and made the most difference (proportionally) to the following pathogens: \textit{Enterobacter}, other \textit{Streptococci}, \textit{C albicans} and \textit{S aureus} (see online supplementary tables 1–3). The \textit{E coli} associated with EOS (<48 h) also appeared to be different to those associated with LOS based on antibiotic susceptibilities; 83% of early onset \textit{E coli} were amoxicillin resistant but were otherwise fully susceptible, whereas 18% of late onset \textit{E coli} were amoxicillin resistant, 24% were resistant to augmentin, 28% to cepazidime, 27% to ciprofloxacin, 12% to gentamicin and 25% to tobramycin. This difference is noted in \textit{E coli} beginning from day 3 of life. Finally, using a definition for early onset infections of 0–6 days, the percentage of early onset infection increases to 33% but the number of GBS infections barely changed (an additional three between days 3 and 6).

**Table 1** Infection episodes per year as a percentage of all live births in each birth weight category

<table>
<thead>
<tr>
<th>Birth weight (g)</th>
<th>2006</th>
<th>2007</th>
<th>2008</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1000</td>
<td>CoNS 25.3 (94/372)</td>
<td>19.3 (95/492)</td>
<td>21.0 (127/602)</td>
</tr>
<tr>
<td></td>
<td>+CoNS 40.8 (201/492)</td>
<td>43.2 (260/602)</td>
<td></td>
</tr>
<tr>
<td>1000–1499</td>
<td>CoNS 4.5 (21/458)</td>
<td>5.6 (26/464)</td>
<td>3.6 (23/646)</td>
</tr>
<tr>
<td></td>
<td>+CoNS –</td>
<td>12.9 (60/464)</td>
<td>8.2 (53/646)</td>
</tr>
<tr>
<td>1500–2500</td>
<td>CoNS 1.3 (16/1187)</td>
<td>1.5 (21/1404)</td>
<td>1.0 (16/1541)</td>
</tr>
<tr>
<td></td>
<td>+CoNS –</td>
<td>2.1 (30/1404)</td>
<td>2.3 (36/1541)</td>
</tr>
<tr>
<td>&gt;2500</td>
<td>CoNS 3.2 (38/1886)</td>
<td>1.3 (33/2526)</td>
<td>1.0 (29/2314)</td>
</tr>
<tr>
<td></td>
<td>+CoNS –</td>
<td>1.9 (40/2526)</td>
<td>1.4 (40/2014)</td>
</tr>
</tbody>
</table>

*CoNS, excluding CoNS; +CoNS including CoNS.
CoNS, coagulase negative \textit{Staphylococcus aureus}.

**Table 2** Most common pathogens

<table>
<thead>
<tr>
<th>Pathogens</th>
<th>CoNS (N=124)</th>
<th>LOS (N=416)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>GBS</td>
<td>65</td>
<td>50</td>
</tr>
<tr>
<td>Escherichia coli</td>
<td>23</td>
<td>18</td>
</tr>
<tr>
<td>Listeria monocytogenes</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>Streptococcus sp.</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>Staphylococcus aureus (MSSA)</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Staphylococcus aureus (MRSA)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Haemophilus influenzae</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Bacillus sp.</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Enterococcus</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Enterobacteriaceae†</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Candida sp.</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Acinetobacter</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>Pseudomonas</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Other†</td>
<td>3</td>
<td>2</td>
</tr>
</tbody>
</table>

Other organisms causing EOS: \textit{Micrococcus} (1), \textit{diphtheroids} (1), \textit{Neisseria} sp. (1); other organisms causing LOS: \textit{diphtheroids} (4), \textit{Proteus} (2), \textit{Micrococcus} (3), \textit{Stenotrophomonas} (2), \textit{Staphylococcus aureus} (8), \textit{Burkholderia cepacia} (1).

**Table 3** LOS resistance

<table>
<thead>
<tr>
<th>Flucloroxacin/gentamicin</th>
<th>Amoxicillin/cefotaxime</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of isolates tested</td>
<td>Sensitivity (%)</td>
</tr>
<tr>
<td>CoNS</td>
<td>122</td>
</tr>
<tr>
<td>Enterobacteriaceae†</td>
<td>72</td>
</tr>
<tr>
<td>Staphylococcus aureus (MSSA)</td>
<td>24</td>
</tr>
<tr>
<td>Enterococcus</td>
<td>4</td>
</tr>
<tr>
<td>Escherichia coli</td>
<td>36</td>
</tr>
<tr>
<td>GBS</td>
<td>7</td>
</tr>
<tr>
<td>Pseudomonas</td>
<td>14</td>
</tr>
<tr>
<td>Streptococcus</td>
<td>5</td>
</tr>
<tr>
<td>Acinetobacter</td>
<td>7</td>
</tr>
<tr>
<td>Bacillus</td>
<td>3</td>
</tr>
<tr>
<td>Overall</td>
<td>84</td>
</tr>
</tbody>
</table>

\textit{Enterobacteriaceae} includes \textit{Klebsiella}, \textit{Serratia}, \textit{Enterobacter} and \textit{Citrobacter}. CoNS, coagulase negative \textit{Staphylococcus}; GBS, group B \textit{Streptococcus}; LOS, late onset sepsis; MRSA, methicillin resistant \textit{Staphylococcus aureus}; MSSA, methicillin sensitive \textit{Staphylococcus aureus}.
Approximately one half of infections on day 1 occurred in infants over 2500 g (49%) and 63% occurred in infants of 32 weeks gestation or above (figure 1). Of all late onset infections, 18% occurred between days 3 and 7 and 66% in the first month of life. The age at disease was not influenced by gender (p=0.62) but varied significantly by pathogen. GBS occurred most often on the first day of life, followed by E coli and CoNS. Among late onset pathogens, E coli infections peaked on day 6 and CoNS on days 6–11 (remaining prevalent for the first month), while Enterobacteriaceae, S aureus and Enterococci occurred later, without a discernable pattern (figure 2).

**DISCUSSION**

We have established a neonatal surveillance network and described the burden of infections in a number of English neonatal units. Preterm and low birthweight babies carry the highest burden of infection, with 26% of all babies <1000 g experiencing one or more episodes of infection during their neonatal stay. Neonatal infections in this population are associated with prolonged hospitalisation, adverse neurodevelopmental outcome1 and increased mortalities.25

The two main pathogens causing EOS were consistently GBS and E coli, accounting for approximately two-thirds of all episodes. The incidence of early onset GBS disease averaged 0.5/1000 live births, equivalent to the 0.48/1000 reported from the national BPSU surveillance conducted in 2000–2001.15 It therefore seems unlikely that a significant reduction in early onset GBS disease occurred despite the introduction of national GBS prevention guidelines in 2003.26 This is supported by passive surveillance data reported to the HPA.27 An explanation for this is offered by a recent audit conducted through the NeonIN units which indicated that a significant proportion...
of early onset GBS cases did not receive intrapartum antibi-
otic prophylaxis (IAP) despite the presence of risk factors.
In contrast, in the USA and Australasia the incidence of early
onset GBS has decreased with the introduction of robust
IAP policies. Attention to new or improved GBS prevention
policies in the UK is urgently required.

A wider range of pathogens accounts for LOS, although over
80% were due to CoNS, *S aureus*, *Enterobacteriaceae*, *E coli* and
*Enterococci*. Preterm infants have the highest burden of LOS,
with 19% of all babies <1000 g experiencing one or more epi-
sodes of late onset infection, excluding CoNS, increasing to
42% with the inclusion of CoNS.

Comparisons can be made between NeonIN data and that of
several other UK studies. The incidence of MRSA infections
over the 3-year period (eight episodes, 6/100 000 live births) is
comparable with that of a recent national MRSA surveillance
study (6/100 000 live births). We can report that NeonIN infants
with MRSA infections were predominantly female, of
low birthweight (median 820 g) and experienced their infec-
tion at a median of 36 days of age (all were late onset). A single
centre study by Haque et al reported a similar incidence of
neonatal sepsis (1% of live births and 5% of neonatal admis-
sions) and distribution of causative pathogens, as did a per-
natal mortality study conducted in the North of England.

The incidence of VLBW infants experiencing invasive fungal
infection (14/1000 over 5 years) is comparable with the annual
incidence of 10/1000 reported from the national BPSU study
in 2003–2004. The slightly higher incidence may indicate an
increase over the last 5 years or may reflect the predominance
of tertiary units in our network, and is worthy of further study.
Taken together with our equivalent incidence of early onset
GBS disease to that of national surveillance, these compari-
sons provide some support for the generalisability of NeonIN
surveillance data to the UK neonatal population.

Comparison of NeonIN data with data from other interna-
tional neonatal infection surveillance networks provides some
interesting insights. There is a striking predominance of *E coli*
sepsis in the USA, partly accounted for by a lower proportion of
GBS and CoNS episodes (see online supplementary table 4).
Similarly, when compared to the Australasian data, GBS is a
more common pathogen in UK units (NeonIN: 0.49/1000 live
births vs ASGNI: 0.25/1000 live births), while *E coli* is less
commonly found (NeonIN: 0.17/1000 live births and 2.9/1000
VLBW vs ASGNI: 0.3/1000 live births and 6.2/1000 VLBW). A
plausible but partial explanation is the differences in use of
IAP as discussed earlier. Time-trend analysis of NICHD data
showed that the decline in GBS coincided with the rise in
*E coli*, and specifically of ampicillin resistant strains. This,
however, had not been observed in the Australasian series.

Comparison of LOS data from NeonIN with NICHD and
NeoKISS data reveals a comparable incidence of infection
episodes. NeoIN and NeoKISS surveillance data show less
than half the incidence of fungal sepsis compared with the
NICHD network (see online supplementary table 5).

NeonIN data indicate that approximately 95% of early onset
infections are susceptible to a penicillin/gentamicin combi-
nation, which, therefore, appears to be a satisfactory empiric
choice. The only early onset pathogen that is not covered by
this regimen is *S aureus* and further work may be able to iden-
tify babies at high risk for this pathogen for whom empiric
therapy should be modified. It is worth noting that there were
no deaths among early onset *S aureus* episodes. With the exclu-
sion of CoNS, the majority of LOS infections (84%) are also
susceptible to the relatively narrow spectrum combination of
fluoroxyacin and gentamicin. The British National Formulary
for Children (BNFC) also offers cefotaxime and ampicillin as
an alternative regimen for EOS and LOS; however, our data
do not support this. Indeed this combination provides less
coverage for the relevant disease causing organisms, yet as the
broader spectrum combination, has a greater potential to pro-
mote antibiotic resistance and the emergence of other patho-
gen such as fungi. It is important however, that neonatal
units are aware of their local resistance patterns and that this
is reflected in their empiric antibiotic policies.

There is a wider question regarding the use of aminogly-
cosides that needs to be tackled by the neonatal community
regarding the potential for ototoxicity, especially in those
with the m.1555A>G genetic mutation. This requires further
research and discussion.

An important role for the empiric use of cefotaxime is in
suspected meningitis, where the favourable CSF penetration
of cefotaxime makes it the preferred agent (in combination
with ampicillin and possibly an aminoglycoside). Meningitis
should therefore be excluded as soon as possible.

Neither of the BNFC recommended empiric combina-
tions adequately accounts for CoNS, although it may be
argued that empiric cover for this pathogen is not essen-
tial. CoNS are a rare cause of life-threatening infection in
neonates. Moreover, excessive use of vancomycin may be
associated with antibiotic resistance and emergence of other
pathogens. A reasonable approach is therefore to institute
antibiotic therapy for CoNS only when a positive culture is
obtained and deemed to be clinically relevant. Further study
is required on whether a specific high risk group can be iden-
tified that would merit empiric use of vancomycin. The case
definition used for sepsis may also be criticised as being too
simple. It is clear, however, that this is essential to ensure
the cooperation of busy clinicians and the completeness of
data collection. It is also consistent with that used in other
well established surveillance systems (http://www.nichd.
nih.gov; https://www.vtoxford.org/home.aspx). The poten-
tial weakness of such a definition relates to the interpretation
of positive cultures due to pathogens that are also frequent
skin contaminants (especially CoNS). This is based on the
clinician’s decision to treat with antibiotics for at least 5 days
and is therefore likely to be subjective. This is particularly
so for early onset infections caused by CoNS, which in our
data set are much more numerous than expected. As with late
onset CoNS episodes, we suspect that this reflects an overes-
timation resulting from this pragmatic definition of sepsis.
However, it ensures some consistency in assessing the burden
of CoNS infections and will enable comparison over time and
between units. An alternative is to incorporate more clinical
and laboratory markers into the case definition. However,
this requires more effort from clinicians and may therefore
compromise data completeness. We are investigating ways
that information on markers may be incorporated into the
database and still ensure ease of use.

In summary, NeonIN presents the first national data on
the epidemiology of neonatal sepsis and provides an insight
into the relevant pathogens and the appropriate antibiotics
for empiric therapy. Ongoing expansion of the network across
the UK will strengthen the richness and utility of the database.

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