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Estimating the National Burden of Early Onset Neonatal Sepsis

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ABSTRACT

Estimating the National Burden of Neonatal Early Onset Sepsis

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Bacterial sepsis of the newborn is a notable etiology of mortality. Infections classified as early-onset sepsis (EOS) are evident within the first 72 hours of life. We performed a retrospective secondary data analysis by applying incidence rates derived from an active laboratory and population-based surveillance system to national natality data to estimate the national pathogen-specific and all-cause incidence rates of neonatal early onset sepsis. We estimated that 3183 cases of EOS occurred in 2005 (0.77 cases per 1,000 live births), 2898 in 2006 (0.79 cases per 1,000 live births), and 3195 in 2007 (0.74 cases per 1,000 live births), for an average of 3092 cases of EOS (0.77 cases per 1,000 live births) annually during each of the years 2005-2007. Black preterm infants had the highest incidence of all cause EOS annually. An overall case fatality rate (CFR) of 9.5% was calculated from ABCs NNS data. The CFR from 2005-2007 was greatest for Black preterm neonates, (21-33%) compared with their non- Black preterm counterparts (15-17%). Black and non-Black preterm infants had the highest incidence of both ampicillin-sensitive (0.54 and 0.56 per 1,000 live births) and ampicillin-resistant *E.coli* (1.03 and 0.45 per 1,000 live births) isolates during each of the years from 2005-2007. This assessment highlights a racial and gestational age predisposition for EOS. Understanding these risk factors could translate into improved maternal and infant health.

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ABBREVIATIONS

ABCs:	Active Bacterial Core Surveillance
CDC:	Centers for Disease Control and Prevention
CFR:	Case Fatality Rate
EIP:	Emerging Infections Program
EOS:	Early Onset Sepsis
NCHS:	National Center for Health Statistics
NNS:	Neonatal Surveillance

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INTRODUCTION

Infections in the neonatal period account for over one million deaths annually worldwide (1). Mortality rates range from 100 deaths per 1,000 live births in Africa to 20 deaths per 1,000 live births in the Americas. In the United States, 6.69 infant deaths were noted per 1,000 live births in 2006 (2). Bacterial sepsis of the newborn was the eighth leading cause of infant mortality, accounting for 807 deaths in 2006, based on ICD-10 codes. This represents 2.8% of total infant deaths and a rate of 0.19 per 1,000 live births in 2006. Although bacterial sepsis of the newborn is a less substantial etiology of infant mortality than other conditions, it is a substantial cause of morbidity (3), (4), (5). Furthermore, a substantial proportion of episodes may be preventable with peripartum screening and intrapartum antimicrobial administration.

Bacterial sepsis classically consists of bloodstream infections and meningitis. Infections during the neonatal period are categorized into early-onset (EOS) and late-onset sepsis (LOS), infections based on the age of the neonate at the time of submission of a specimen for organism isolation. This temporal characterization into EOS and LOS has implications for the suspected route of acquisition. EOS is often associated with vertical transmission via organisms that ascend from the lower genital tract, maternal bacteremia resulting in transplacental translocation, or acquisition via passage through the birth canal. Classically, onset is within the first 72 hours of life, although organism isolation within the first 7 days of life has been considered to be EOS by some investigators. In contrast, LOS is classified as pathogen isolation after 72 hours (or after 7 days by some investigators) of life up to 90 days of age. Although some infectious etiologies associated with LOS have also been hypothesized to be acquired via passage through the maternal canal at birth, but with later presentation, nosocomial and community sources are more

predominant etiologies of LOS. Since most etiologies of EOS are temporally associated with maternal factors, these provide ideal opportunities for the focus of prevention strategies.

The relative contribution of organisms associated with EOS has varied over time and geographic location. However, within the last 20 years, group B streptococci (GBS) have been recognized as the most common etiology of EOS in the United States with rates of infection ranging between 1.4 to 1.7 per 1000 live births (6, 7). Universal screening and intrapartum prophylaxis administered to pregnant women colonized with group B streptococci have been shown to be effective for the prevention of EOS GBS neonatal disease (8). The introduction of risk-based screening of pregnant women for GBS colonization contributed to a decline in case fatality rates among very low birth weight infants (<1500g) from 5.9 per 1,000 live births from 1991-3, prior to risk-based screening and intrapartum prophylaxis to 1.7 per 1000 live births following the institution of risk-based screening and intrapartum prophylaxis from 1998 -2000 (9). This decline in case fatality rates may have also been the result of improvements in postnatal care that occurred during the interim decade before and after evaluations.

Despite notable reductions in overall incidence and case fatality rates, racial disparities associated with EOS GBS infections persist, with higher rates of disease and higher case fatality rates consistently noted among black infants (10). Additionally, preterm infants experience a greater burden of disease from EOS. Understanding these observations could provide opportunities for targeted prevention efforts. To quantify the impact of early onset neonatal sepsis in the United States, we applied current information from a population and laboratory-based surveillance system to national live birth data to develop overall, race and gestational age specific estimates of the burden of early onset neonatal sepsis from 2005 to 2007.

BACKGROUND

Following the identification of GBS as a notable cause of neonatal bacterial sepsis in the 1970s, the Centers for Disease Control and Prevention (CDC) developed guidelines for screening and administration of antibiotic prophylaxis published in 1996. Providers could choose to administer therapy based on risk factors or universally perform a late antenatal culture (11). A population-based evaluation of these two options demonstrated that culture-based screening was >50% more effective than risk-based screening (12). The revision of these guidelines in 2002 to include universal maternal screening at 35-37 weeks of gestation has resulted in a notable reduction among EOS attributable to GBS, especially among term infants whose mothers have had the benefit of late antenatal screening. However, missed opportunities for screening and intrapartum prophylaxis were noted in 13% of neonatal group B streptococcal disease in a retrospective cohort following the institution of universal screening guidelines (13).

The emergence of antimicrobial resistant organisms in community and nosocomial settings has raised concerns regarding the widespread use of intrapartum antibiotic prophylaxis. Suggestion of the promotion of antimicrobial resistance among neonatal pathogens with this practice has been considered as ampicillin-resistant *Escherichia coli* become more prevalent Additionally, the increasing prevalence of community and hospital acquired methicillin-resistant *Staphylococcus aureus* (MRSA) has also raised concerns regarding use of intrapartum antimicrobial therapy in both colonized and symptomatic women.

Data to assess the emergence of antimicrobial resistant organisms responsible for EOS in neonates is lacking due to the difficulty of surveillance and challenges in the definition of clinical versus laboratory confirmed etiologies. Most evaluations count cases where an infectious etiology can be confirmed. However, inclusion only of cases with laboratory identification of an

organism excludes cases where a neonate displays signs suggestive of infection with an invasive pathogen. Although trends in neonatal mortality based on national database coding of clinical sepsis as defined by International Classification of Diseases, Ninth Revision (ICD-9) codes reveal similarities between rates of mortality for culture confirmed and clinical sepsis (14), trends for other indicators of morbidity are unknown. This analysis focuses on determination of the national burden of pathogen-confirmed neonatal EOS, realizing that pathogen confirmed infections might represent only a portion of the true burden of neonatal EOS.

While individual institutions have reported their site specific epidemiology of neonatal sepsis (15) and multicenter collaborations (16) have described the epidemiology of pathogens in low birth weight infants, estimates for the national burden of pathogen-confirmed EOS among all infants are largely unknown. The objective of this evaluation was to estimate the national burden of early onset neonatal sepsis, and derive race and gestational age specific incidence rates and assessment of the burden of disease.

METHODS

Null Hypotheses

The primary null hypothesis is that early onset sepsis (EOS) due to bacterial pathogens does not account for a substantial proportion of neonatal mortality and morbidity annually in the United States. Secondary null hypotheses include 1) the absence of a racial predisposition for bacterial EOS, 2) the absence of predisposition for EOS based on gestational age and 3) the minimal contribution of antimicrobial-resistant organisms to the burden of EOS.

Study Design

This retrospective secondary data analysis involved the application of incidence rates derived from an active laboratory and population-based surveillance system to national live birth data to estimate the national pathogen-specific and all-cause incidence rates of neonatal early onset sepsis.

Data Sources and Subjects

Two data sources were utilized to derive the national estimates of early onset neonatal sepsis, 1) ABCs-NNS and 2) NCHS Natality Data

ABCs-NNS

The Active Bacterial Core Surveillance (ABCs) is a component of the Centers for Disease Control and Prevention's (CDC) Emerging Infections Programs Network (EIP); a collaboration between CDC, state health departments and academic centers. ABCs is an active laboratory and population-based surveillance system for invasive bacterial pathogens of public health importance. For each case of invasive disease in the surveillance population, a case report with basic demographic information is completed, and bacterial isolates are sent to CDC and other reference laboratories for additional evaluation. ABCs was initially established in four

states in 1995; surveillance data from this program was instrumental in advancing the revised recommendations for preventing neonatal GBS Disease (17, 18). ABCs currently includes 10 EIP sites across the United States, representing a population of over 38 million persons and 450,000 live births (11% 0f U.S. live births). A subset of the ABCs surveillance system, the Neonatal Surveillance (NNS) includes a 3 county area in California (Bay Area), the state of Connecticut, 18 hospitals in metropolitan Atlanta (8 counties) in 2005. The state of Minnesota was added to ABCs-NNS in 2006.

ABCs-NNS case finding is both active, population and laboratory-based for all cases of invasive bacterial infections, defined as isolation of a pathogen associated with neonatal sepsis from a normally sterile site (blood or cerebrospinal fluid for live births; placenta or amniotic fluid for fetal deaths) from birth up to 72 hours of life. Microbiology laboratories in acute care hospitals and reference laboratories are contacted regularly to assist with case identification. In other laboratories, electronic line listings of all sterile site isolates are submitted routinely for evaluation as part of the ABCs-NNS. Clinical records corresponding to these isolates are reviewed. Antimicrobial susceptibility information of isolates is recorded and submitted to ABCs-NNS. Surveillance sites collect demographic, neonatal, and obstetric information from medical records. Race and ethnicity information is extracted from labor and delivery records, case-report forms, or infants' birth certificates. Imputation procedures are used to reconcile missing data for race and gestation. To ensure that all cases of disease under surveillance are being reported, audits of the reporting laboratories are performed every January and July for the previous six-month period. Surveillance sites assess the completeness of information collected for each case. If information is consistently incomplete, the data collection methods are reassessed to address the deficiencies.

NCHS Live Birth (Natality) Data

Natality data available from the National Center for Health Statistics (NCHS) for 2005 (19) and 2006 (20) were used as the denominators for incidence rate calculations; incidence rates for 2007 were calculated using 2006 data (20) due to unavailability of the 2007 data at the time that the analyses were performed. The natality data consists of descriptive tabulations of the data reported on the birth certificates of the 4.1 million births (19) and 4.3 million births (20) that occurred in 2005 and 2006 respectively. The natality data is based on 100 percent of the birth certificates registered in all states and the District of Columbia, which includes more than 99 percent of births in the United States (19).

Outcome variables

The primary outcome variable was the mean estimated burden of early onset neonatal sepsis derived from 2005-2007 data. Secondary outcome variables included total annual estimates, and estimates by race and gestation of infants with EOS.

Predictor variables

Race and gestational age of infants were the predictor variables in this analysis. Cases were categorized into one of four categories based on race and gestational age. Subjects defined as Black race included those individuals who were identified as Black or Black, non-Hispanic in the ABCs-NNS and the natality data for the corresponding year. All other race designations were identified as non-Black. Cases defined as preterm were those with a gestational age at birth that was recorded as less than 37 weeks. Cases defined as term and post-term were those with a gestational age at birth that was 37 weeks and 0/7 days or greater. Each case of EOS reported in ABCs-NNS was placed into one of four race-gestational age categories, 1) Black preterm, 2) Black term 3) non-Black preterm, and 4) non-Black term. These categories were selected to

account for the over-representation of Black term and under-representation of non-Black preterm infants included in ABCs compared with natality data (Table 1). These categorizations were also selected to account for the predisposition of EOS group B streptococcal infections in Black infants (8).

Analysis:

SAS (Cary, North Carolina) version 9.1 statistical software was used to obtain frequency data and distributions to build the tables of variables of interest from the ABCs-NNS dataset. The results of these analyses were used to calculate the incidence rates of neonatal sepsis cases in total, by year, and by pathogen for each of four race and gestational age categories. Incidence rates derived from the ABCs-NNS data were applied to national natality data to estimate the overall burden of national cases of neonatal EOS. The incidence rates from ABCs-NNS were also applied to national natality data by corresponding race and gestational age categories to derive national estimates of pathogen-specific infections overall, by race and term categories, by race and gestational age categories for the most common pathogens, and by race and gestational age categories to derive national estimates of antimicrobial resistant infections.

RESULTS

National Representativeness of ABCs-NNS

The ABCs-NNS captured 4.1% of live births in 2005; the state of MN was not included in the surveillance during this year. ABCs-NNS surveillance covered the equivalent of 6.5% percent of live national births in 2006 and 2007; 2006 denominator data was used in both 2006 and 2007. From 2005 to 2007, ABCs-NNS captured a mean of 2.9% of Black preterm live births compared with a mean of 3.0% of live births nationally in 2005; a mean of 15.6% of Black term births compared with a mean of 13.3% of live Black term births nationally, a mean of 8.2% of non-Black preterm births compared with a mean of 10.5% of non-Black preterm births nationally, and a mean of 73.2% of non-Black term live births compared with a mean of 73.7% of non-Black term live births nationally (Table 1).

All Cause EOS

Using ABCs NNS data and national natality data for the corresponding year, with 2007 represented by 2006 live birth data, we estimated that 3183 cases of EOS occurred in 2005 (0.77 cases per 1,000 live births), 2898 in 2006 (0.79 cases per 1,000 live births), and 3195 in 2007 (0.74 cases per 1,000 live births), for an average of 3092 cases of EOS (0.77 cases per 1,000 live births) annually during each of the years 2005-2007. Based on incidence rates by race and gestational age categories from the ABCs-NNS, Black preterm infants had the highest incidence of all cause EOS annually resulting in a projected yearly average of 514 cases of EOS (Table 2).

Outcomes

Among cases for which outcome data was available, 46 deaths were noted from 2005-2007, yielding an overall case fatality rate (CFR) of 9.5% from ABCs NNS data. Eight cases of EOS were without outcome data. The CFR from 2005-2007 was greatest for Black preterm neonates, ranging from 21-33% compared with their non-Black preterm counterparts in whom CFRs ranged from 15-17%. There were no deaths among Black term infants in 2007 or non-Black term infants in 2005 (Table 3).

Pathogen-Specific Rates of EOS

Both GBS and *E. coli* had the highest incidence among Black Preterm infants (1.57 per 1,000 live births for each organism). Black preterm and term infants had the highest incidence of EOS GBS (1.57 and 0.59 per 1,000 live births); both Black and non-Black preterm infants had the highest incidence of *E.coli* EOS (1.57 and 1.01 per 1,000 live births respectively). Viridans group streptococci comprised the third most common group of EOS bacterial pathogens with the highest incidence among Black preterm infants (0.43 per 1,000 live births), followed by both typable and non-typable *Haemophilus influenza*e (0.33 per 1,000 live Black preterm infant births) and *Staphylococcus aureus* (0.05 per 1,000 live Black preterm infant births). Black preterm infants had the highest incidences of EOS with GBS, *E.coli*, viridans group streptococci, *H. influenzae*, and *S. aureus* (Tables 4).

Antimicrobial-Resistant EOS Organisms

Black and non-Black preterm infants had the highest incidence of both ampicillin sensitive (0.54 and 0.56 per 1,000 live births) and ampicillin resistant *E.coli* (1.03 and 0.45 per 1,000 live births) isolates during each of the years from 2005-2007. Both Black and non-Black term infants had lower incidences of ampicillin sensitive *E. coli* isolates (0.08 and 0.05 isolates

per 1,000 live births) and lower incidences of ampicillin resistant isolates (0.03 and 0.02 per 1,000 live births). EOS *E. coli* infections comprised (116/482) 24% of the pathogen-specific isolates and ampicillin-resistant *E. coli* accounted for 55/116 (47%) of the total EOS *E. coli* infections (Table 5). During the 2005-2007 surveillance periods, one methicillin-resistant *Staphylococcus aureus* was isolated from a Black term infant. The remainder of the 21 *Staphylococcus aureus* isolates during this period were methicillin-sensitive.

DISCUSSION

This is the first study to estimate the burden of EOS neonatal sepsis in the United States in the era of intrapartum antimicrobial prophylaxis. We applied data from an active laboratory and population-based surveillance system to linked live birth- infant death data to derive national estimates of the burden of EOS sepsis. This assessment focused on laboratory-confirmed EOS attributed to a bacterial pathogen. Therefore, our estimate that 3,092 cases of EOS (0.77 cases per 1000 live births) may be expected annually is conservative as it does not consider non-bacterial etiologies of EOS nor does it include clinical sepsis in which a pathogen is not recovered. Understanding the burden of EOS is important as organisms traditionally associated with presentation during the first 72 hours of life are likely acquired via peripartum transition and are suitable targets for prevention strategies to reduce the risk for infection. Therefore, the impact on the burden of infection may be greatest for EOS infections.

Overall trends in all cause EOS remained relatively constant among the four race and term categories each year from 2005. An exception was noted among Black term infants in whom a decline in the average incidence of infections from 0.1 in 2005 to 0.07 per 1,000 live Black term births in 2007. Preterm births accounted for an EOS incidence rate of between 0.22 and 0.45 per 1,000 pretem births while the rate among term infants was between 0.04 to 0.1 per 1,000. One of the reasons for this disparity may be attributed to the increasing proportion of near-term births, defined as births occurring between 34 and 37 weeks. Late preterm infants have been noted to have a greater incidence of pulmonary and infectious complications, (21) and may account for the increased incidence of EOS noted in this assessment among births classified as preterm in the ABCs-NNS. Additional analyses with stratification into preterm (<34 weeks, near term between 34 and 37 weeks, and term $\geq$ 37 weeks) may explain the increased incidence of

EOS in infants classified as preterm, many of whom may be more accurately categorized as near-term infants with risk factors for sepsis that are intermediate to term and preterm neonates.

The case fatality rates (CFR) for all-cause EOS mortality were greatest among preterm Black infants (26%), preterm non-Black infants (17%), followed by term Black infants (2%) and term non-Black infants (1%). The distribution of CFRs mirrors those seen with rates of overall EOS.

In this assessment, GBS remained the most common bacterial etiology of EOS, followed by *E.coli*, viridans streptococci, *Haemophilus influenzae*, and *Staphylococcus aureus*,.(Table 4).. Enterococci , other group D streptococci, *Streptococcus pneumoniae, Listeria monocytogenes, Klebsiella pneumoniae,* group A streptococci and other pathogens, primarily gram-negative organisms comprised the remainder of isolates (data not shown). The predisposition of Black preterm infants to sepsis of all etiologies is notable. Interestingly, an incidence of 1.57 cases per 1,000 Black preterm births was noted for both GBS and *E. coli* from 2005 to 2007 while 0.60 GBS and 0.10 *E.coli* cases were estimated per 1,000 live Black term infants from 2005 to 2007, a difference that appears to be due to term status.

Among the neonates with EOS attributed to *E. coli*, non-Black preterm infants had the second highest incidence after Black preterm infants. Although the overall incidence of GBS remained higher than *E. coli* EOS infections, the incidence among both Black and non-Black preterm infants for *E. coli* sepsis emphasizes the predisposition of preterm infants to have an *E. co*li infection in the first 72 hours of life. This predisposition to gram-negative EOS among low birth weight and preterm infants has been described by others, (9)

Viridans streptococci, a large group of either α-hemolytic or non-hemolytic bacteria without Lancefield antigens, were the third most commonly isolated organisms from neonates in

the first 72 hours of life. Similar to the pattern noted with *E. coli*, Black preterm and non-Black preterm infants had a greater incidence of viridians streptococci than their term counterparts. Although the significance of the isolation of one of these organisms, traditionally felt to be part of the commensal oral flora, may be raised, their isolation from a sterile site, in a neonate with signs and symptoms consistent with sepsis fulfills the inclusion criteria of ABCs-NNS. Preterm infants, both Black and non-Black, had lower rates of viridians streptococci sepsis than their term counterparts, (Table 4).

Overall, preterm infants had the highest incidence of ampicillin-resistant *E. coli* isolates; with Black preterm neonates accounting for the most isolates among all preterms. The 76% ampicillin resistance rate among *E. coli* isolates from preterm infants ($\leq$ 37 weeks) is comparable to the 85% rate of resistance reported among a cohort of very low birth weight infants in the National Institutes of Health National Institute of Child Health and Development Neonatal Research Network from 1998 -2000 (16). Our overall rate of 47% ampicillin-resistant *E. coli* isolates among all *E. coli* isolates seems consistent with individual institutional experiences(22, 23) . Our finding of only one methicillin resistant *S. aureus* isolate in the three-year surveillance period from a Black term infant out of 21 total *S. aureus* isolates (4.8%) was greater than the 2.1% (2/96) methicillin-resistant *S. aureus* maternal colonization rate in a single center study from 2005-2006, (24). However, due to the small sample size of isolates both in this assessment and in the cited maternal colonization study, our single isolate from 2005-2007 and the 2 maternal isolates from 2005-2006 may be comparable.

Our estimates of the burden of EOS neonatal sepsis are subject to several limitations. First, the racial and gestational characteristics of the ABCs NNS areas of surveillance may not be representative of the race and gestational age of the national live birth cohort. ABCs NNS

captured a higher proportion of Black term infants from 2005-2007 and lower rates of non-Black preterm infants. The proportion of Black preterm infants and non-Black term infants were comparable between ABCs NNS and national data. Second, the analyses in this study represented a three-year period, with 2006 denominator data used for both 2006 and 2007 denominator data in ABCs NNS and national data. While year to year variations in live births, both in ABCs and the national data appear to be inconsequential, it is possible that an increase in the 2007 denominator data, yet to be finalized, could account for an under or over representation of burden of disease estimates. Of note, isolates and live births in the state of Minnesota (73,486 in 2006, 84% of which were non-Black term births) were not included in the 2005 ABCs NNS analyses. Finally, the racial and gestational age data was entered at the discretion of the individual surveillance officer at each one of the surveillance sites. The potential for misclassification bias exists. The four race-gestational age categories were selected as these characteristics both offered the most complete data and reflect current knowledge regarding risk factors for EOS. However, these categorizations may obscure or be a proxy for an underlying risk factor that has not been recognized to date.

The categorization of infants into term ($\geq$ 37 weeks) and preterm (< 37 weeks) was based on traditionally accepted definitions. A more complete understanding of the predisposition of preterm infants for all cause and pathogen-specific EOS could be obtained by further categorizing preterm infants into early preterm (< 34 weeks of gestation) and late preterm (34 to 37 weeks of gestation). Although current data collection procedures do not readily facilitate this categorization, consideration of these additional analyses could be made for future evaluations.

To further understand the burden of EOS and optimize prevention strategies, additional data sources are needed to validate the point estimates derived in these analyses. Ideally, more

complete information pertaining to maternal and neonatal clinical courses would assist with a broader understanding of risk factors not captured by race and gestational age. Although the race categorizations for ABCs-NNS were extracted from labor and delivery records, case-report forms, or infants' birth certificates and from birth certificates for the national natality data, the reliability of these records and variability by source may not accurately capture a maternal-infant pairs' racial origin. Furthermore, categorization by imposed categories may obscure an underlying factor that is unrelated to an imposed racial category. However, for longitudinal health assessments and comparison of trends, race and ethnicity have been used consistently and, although not ideal, may provide useful demographic information. In an evaluation of of screening and intrapartum antibiotic administration among infants with EOS GBS during 2003 and 2004, missed screening opportunities of mothers who delivered at term accounted for 13.4% of GBS infections. Black race, Hispanic ethnicity, previous delivery of a live infant, history of illicit substance use, and inadequate prenatal care were significantly associated with not undergoing screening (13).

This first national estimate of the burden of neonatal early onset sepsis demonstrates that the number of infections is notable: 3,000 annually in the United States. Furthermore, it highlights a racial and gestational age predisposition for EOS as well as the persistence of ampicillin-resistant *E. coli* isolates. As the pathophysiology of EOS is proposed to involve the vertical transmission of organisms from maternal flora to infant, opportunities for prevention of peripartum transmission continue to be the targets for prevention of neonatal infection. Understanding the racial and gestational age predilection for EOS could be applied to evaluating revised recommendations for screening pregnant women earlier in gestation as well as focusing limited resources on prenatal care. Reduction of the national burden of 3,000 annual EOS

infections could translate into improved maternal and infant health while reducing the economic burden of both neonatal EOS and its sequelae.

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Table 1: Percentages of Births by Race/Gestational Age from the Active Bacterial Core Surveillance (ABCs) and from the National Vital Statistics System (2005-2007)

Race/ Gestational Age	ABCs NNS Births	United States Births
Black Preterm		
2005	3.3%	2.8%
2006	2.8%	3.1%
2007*	2.8%	3.1%
Black Term		
2005	17.5%	12.4%
2006	14.6%	13.7%
2007*	14.6%	13.7%
Non-Black Preterm		
2005	8.1%	10.0%
2006	8.3%	10.8%
2007*	8.3%	10.8%
Non-Black Term		
2005	71.1%	74.8%
2006	74.3%	72.4%
2007*	74.3%	72.4%

** 2007 represented by 2006 natality data*

Table 2: National Estimates of Early Onset Sepsis (EOS) by Race and Gestational Age Categories

Race/ Gestational Age	ABCs NNS Incidence (per 1,000 live births)	National Estimate (Number of EOS cases)
Black Preterm		
2005	0.42	445
2006	0.46	519
2007*	0.50	577
Black Term		
2005	0.10	494
2006	0.11	571
2007*	0.07	359
Non-Black Preterm		
2005	0.20	779
2006	0.25	975
2007*	0.24	1020
Non-Black Term		
2005	0.04	1141
2006	0.04	1047
2007*	0.04	1323

** 2007 represented by 2006 live birth data*

Table 3: Case Fatality Rates (CFR) Due to Early Onset Sepsis (EOS) by Race and Gestational Age Categories

Race/ Gestational Age	ABCs NNS CFR (%)	National Estimate (Number of EOS Deaths)
Black Preterm		
2005	23	101
2006	33	173
2007*	21	122
Black Term		
2005	34	17
2006	26	15
2007*	0	0
Non-Black Preterm		
2005	15	120
2006	17	163
2007*	17	177
Non-Black Term		
2005	0	0
2006	14	15
2007*	14	19

** 2007 represented by 2006 live birth data*

Table 4: Pathogen Specific Early Onset Sepsis (EOS) by Race and Gestational Age Categories for 2005-2007

Pathogen	ABCs Incidence*	National Estimate of Cases
Group B Streptococci (GBS)		
Black Preterm	1.57	528
Black Term	0.59	880
Non-Black Preterm	0.52	632
Non-Black Term	0.15	1309
Escherichia coli (E. coli)		
Black Preterm	1.57	528
Black Term	0.11	170
Non-Black Preterm	1.01	1217
Non-Black Term	0.05	455
Viridans streptococci		
Black Preterm	0.43	146
Black Term	0.13	201
Non-Black Preterm	0.27	328
Non-Black Term	0.09	797
Haemophilus Influenzae (H. flu)		
Black Preterm	0.33	109
Black Term	0	0
Non-Black Preterm	0.23	281
Non-Black Term	0.01	76
Staphylococcus aureus (S. aureus)		
Black Preterm	0.05	18
Black Term	0.04	62
Non-Black Preterm	0.02	23
Non-Black Term	0.03	285

* Incidence per 1,000 live births

Table 5: Antimicrobial Sensitive and Resistant EOS *E. coli* by Race and Gestational Age Categories for 2005-2007

Race/ Term	ABCs Incidence*	United States Case Estimate
Ampicillin-Sensitive *E. coli*		
Black Preterm	0.54	181
Black Term	0.08	119
Non-Black Preterm	0.56	677
Non-Black Term	0.05	437
Ampicillin-Resistant *E. coli*		
Black Preterm	1.03	346
Black Term	0.03	46
Non-Black Preterm	0.45	538
Non-Black Term	0.02	190

* Incidence per 1,000 live births