

# CLINICAL INVESTIGATION

*Medicina (Kaunas) 2012;48(2):71-6*

## ***Escherichia coli* Colonization in Neonates: Prevalence, Perinatal Transmission, Antimicrobial Susceptibility, and Risk Factors**

Rasa Tamelienė<sup>1</sup>, Eglė Barčaitė<sup>2</sup>, Dalia Stonienė<sup>1</sup>, Jūratė Buinauskienė<sup>1</sup>, Eglė Markūnienė<sup>1</sup>, Aušrelė Kudrevičienė<sup>1</sup>, Astra Vitkauskienė<sup>3</sup>, Daiva Jomantienė<sup>3</sup>, Rūta Nadišauskienė<sup>2</sup>

<sup>1</sup>Department of Neonatology, Medical Academy, Lithuanian University of Health Sciences,

<sup>2</sup>Department of Obstetrics and Gynecology, Medical Academy, Lithuanian University of Health Sciences,

<sup>3</sup>Department of Laboratory Medicine, Medical Academy, Lithuanian University of Health Sciences, Lithuania

**Key words:** *Escherichia coli*; neonate; colonization; risk factors; antibiotic resistance.

**Summary.** *Escherichia coli* is one of the leading causes of early-onset neonatal sepsis in many industrialized countries. However, there is a lack of studies on *Escherichia coli* colonization in women and neonates. The study aimed at determining the prevalence *Escherichia coli* among pregnant women and newborns, perinatal transmission, antimicrobial susceptibility, and risk factors for neonatal colonization.

**Material and Methods.** In this prospective, cross-sectional study, 827 infants born to 808 mothers were enrolled. The study was carried out from October 1, 2006, to June 30, 2007. Women were screened for *E. coli* carriage at 35–37 weeks of gestation or on admission for premature rupture of membranes and delivery; neonates, within 15 minutes of their lives. Risk factors for colonization were collected by a questionnaire and were recorded during labor.

**Results.** Maternal *E. coli* colonization rate was 19.9%; neonatal, 14.4%; and transmission rate, 21.4%. Less than one-fourth (22.7%) of neonatal *E. coli* strains were resistant to ampicillin. Logistic regression analysis revealed that anal sexual intercourse (OR, 3.91; 95% CI, 1.87–8.19), one sexual partner (OR, 2.01; 95% CI, 1.30–3.11), maternal vaginal *Escherichia coli* colonization (OR, 1.81; 95% CI, 1.12–2.93), maternal body mass index of  $\leq 27$  (OR, 1.77; 95% CI, 1.15–2.73), and maternal education lower than university level (OR, 1.70; 95% CI, 1.06–2.74) were associated with neonatal *Escherichia coli* colonization.

**Conclusions.** The prevalence of maternal *Escherichia coli* colonization was higher in this study than other studies (19.9%). Neonatal *Escherichia coli* colonization was 14.4%. The resistance of *Escherichia coli* isolates to ampicillin was not high (22.7%). Improvement of maternal education and modification of mothers' sexual habits need to be undertaken to prevent neonatal *Escherichia coli* colonization.

### **Introduction**

Neonatal sepsis is one of the leading causes of morbidity, long-term disability, and mortality among children of this age group in the majority of developed countries (1–3). The main causative agents of early-onset neonatal sepsis (EONS) that sets on within 72 hours after delivery are B-group  $\beta$  hemolytic streptococcus (GBS) and *Escherichia coli* (*E. coli*) (2, 4), which can easily enter the mother's genital tract from the rectum via the perineum (5, 6). It has been reported that 10%–40% of women (in Lithuania, 15.3%) have vaginal or rectal GBS colonization and transmit this agent to the newborns before or during labor (7, 8). Due to the administration of preventive antibiotic therapy to

women with vaginal GBS colonization during labor and a reduction in neonatal morbidity and mortality from early-onset GBS-induced neonatal sepsis, *E. coli* infection is becoming increasingly recognized as a more common cause of sepsis, and the resistance of this causative agent to ampicillin reaches as much as 85% (2, 4). Vaginal *E. coli* colonization is detected in 7%–13% of pregnant women and in the blood of 21% of fetuses that have died during the third trimester of pregnancy (9–12). The prevalence of neonatal *E. coli* colonization has not been studied yet, and the risk factors for this colonization are not known. It is still unclear whether a general screening of pregnant women for this microorganism is required. So far, no preventive measures against *E. coli*-induced neonatal sepsis have been applied anywhere in the world.

The aim of the study was to determine the prevalence of *E. coli* colonization among pregnant women

Correspondence to R. Tamelienė, Department of Neonatology, Medical Academy, Lithuanian University of Health Sciences, Eivenių 2, 50028 Kaunas, Lithuania  
E-mail: rasatameliene@yahoo.com

and newborns, its perinatal transmission, antimicrobial susceptibility, and risk factors for neonatal colonization.

### Material and Methods

This prospective cross-sectional observation study was carried out at the Clinic of Obstetrics and Gynecology and Clinic of Neonatology, Hospital of Lithuanian University of Health Sciences (HLUHS), between October 1, 2006, and June 30, 2007. HLUHS is a tertiary care university hospital, where 3500 women deliver annually, which comprises approximately 10% of all births in Lithuania. The studied contingent included neonates starting from 22 weeks of gestational age who were born during the period of the study at the Clinic of Obstetrics and Gynecology, HLUHS, as well as mothers of these neonates. The study included women who agreed to participate and 1) attended the maternity clinic at HLUHS during the 35th–37th week of pregnancy; 2) were treated at the Department of Obstetrics, HLUHS, and planned to deliver at HLUHS; and 3) delivered at the Maternity Unit, HLUHS; the neonates of these women were included as well. Neonates whose mothers 1) experienced severe vaginal bleeding during delivery; 2) were treated with antibiotics 2 weeks before the delivery; 3) delivered a stillborn neonate; and/or 4) refused to participate in the study were excluded; the neonates who required urgent postnatal assistance (severe birth defects or required resuscitation) were excluded as well.

**Microbiology.** Upon receiving a woman's written consent, microbiological swab samples (without washing the external genitals or using specula) were obtained from the lower third of the vagina (sample 1) and the anus (sample 2); in neonates, samples were taken within 15 minutes since birth from the nasopharynx (sample 1) and ear (sample 2). The samples were subsequently introduced into the Amies nutrient medium (Brescia, Italy). Swabs were immediately placed in the Amies transport medium (Brescia, Italy) and sent within 24 hours to the laboratory. *E. coli* was identified by standard biochemical tests, such as lactose fermentation, catalase production, and lysis of decarboxylase, ability to ferment mannite, indole production in peptone broth, and inability to utilize citrate or cleave urea. The Phoenix ID system (Becton Dickinson, USA) was used to confirm the strains as *E. coli*. Susceptibility to antibiotics was determined by the disk diffusion method, using Mueller-Hinton agar (Oxoid, UK), which was incubated at 35°C for 16–20 hours. The results were interpreted according to the recommendations of the Clinical Laboratory Standard Institute (formerly CLSI) (13).

**Identification of Risk Factors.** On the day of the sampling, the patients filled out the questionnaires designed by the authors of this study, covering the patients' social and demographic status, lifestyle,

personal hygiene and sex life habits, the course of previous pregnancies and deliveries, and previous diseases (response rate, 85%). The analysis included the data from these questionnaires, risk factors for early-onset infection during delivery, data about antibiotics administered during delivery, the neonates' anthropometric data, and the neonates' condition after birth.

**Preventive Antibiotic Therapy During Delivery.** Antibiotic therapy during delivery for the prevention of GBS in neonates was administered following the recommendations of the Center for Disease Control and Prevention (CDC), the American Academy of Pediatrics (AAP), and the American College of Obstetricians and Gynecologists (ACOG) (2002) (14).

**Statistical Analysis.** The Statistical Package for Social Sciences program (SPSS 13.0, Chicago III, USA) was used for data analysis. For the verification of statistical hypotheses, a significance level of 0.05 was chosen. Quantitative data were described using descriptive statistics: mean value, standard deviation (SD), median, 25th–75th quartile values, and mean rank. The Kolmogorov-Smirnov test was used to determine the distribution of quantitative values. The mean values of the quantitative variables of two independent groups were evaluated. The Student *t* criterion was used for variables with normal distribution, and the Mann-Whitney *U* test was employed for nonnormally distributed data. The frequency of qualitative attributes between two groups of subjects was compared by applying the arcsine criterion or the exact comparative criterion of probability. In cases where more than two groups were involved, the testing of whether the frequency hypotheses were equal was conducted by applying the  $\chi^2$  criterion. When comparative analysis was conducted, the Spearman correlation was used to assess correlations between attributes. The prognostication of *E. coli* colonization in neonates was conducted by applying the stepwise procedure of binary logistic regression analysis.

**Ethical Aspects.** The study was initiated with the permission of Kaunas Regional Bioethics Committee (No. BE-2-39).

### Results

**Characteristics of Women and Neonates Enrolled Into the Study.** During the period of the study, 2238 women gave birth at HLUHS. According to the exclusion criteria, 602 women were excluded from the study: 480 refused to participate, 54 used antibiotics during the last 2 weeks, 35 had profuse genital bleeding, and in 33 cases, the fetus was stillborn; 638 women were excluded from the study due to intensive work and human characteristics of the personnel. In total, 998 women were included into the study. As 28 women did not arrive at HLUHS for delivery, the data of 970 women and 827 neonates were analyzed. Data on 970 women were used to

determine the prevalence of maternal colonization with *E. coli*, and data on 808 women (who delivered 827 neonates, including 19 pairs of twins) were employed to evaluate the prevalence of perinatal transmission and identify the risk factors for neonatal *E. coli* colonization. In 162 neonates of mothers who participated in the study, microbiological test samples were not obtained due to excessive workload and the fact that these neonates required urgent assistance. The main characteristics of women and neonates who did not participate in the study for reasons other than meeting the exclusion criteria were analyzed as well. These characteristics did not differ significantly from those of the participants, and thus the studied sample reflected the whole population of neonates and women who gave birth at HLUHS during the studied period.

The mean age of women who participated in the study was 27.3 years (SD, 5.9). The majority of the women lived in urban areas (71.2%), had a constant partner (92.3%), and were employed (75.7%). Nine participants (0.9%) of the study did not attend prenatal care during pregnancy. The remaining subjects attended prenatal consultations, on the average, 8.4 times (SD, 3.2). More than one-tenth (11.1%) of the subjects smoked up to 10 cigarettes per day during pregnancy; and 2.6% of women, more than 10 cigarettes. In total, 10.2% of women reported that they used vodka, wine, or beer during pregnancy (the amount was not measured in alcohol units). More than half (55.5%) of the women were delivering for the first time. Samples for *E. coli* were obtained, on the average, at 37.4 weeks (SD, 2.6) of pregnancy (between 24 and 41 weeks); in 65.8% of cases, samples were obtained due to premature rupture of the amniotic membranes or during delivery.

Of the 827 neonates included in the study, 76.7% were born vaginally. The Apgar score of the neonates at 1 and 5 minutes after birth was 8.7 (SD, 0.8) and 9.3 (SD, 0.7), respectively. There were 50.1% of boys and 49.9% of girls. The mean birth weight was 3339 g (SD, 615.75); in 7.3% of neonates, the birth weight was less than 2500 g, and 2.4% of neonates were small for their gestational age. Birth occurred, on the average, on the 38.5th week (SD, 2.4) of gestation (in 1.6% of cases, on the 22nd–31st week of gestation, and in 12.3% of cases, on the 32nd–36th week of gestation). The mean hospital stay of the neonates was 5.64 days (SD, 5.41); 6.4% of the neonates were admitted to the Neonatal Intensive Care Unit.

**The Prevalence of *E. coli* Among Pregnant Women and Newborns, its Perinatal Transmission, and Antimicrobial Susceptibility.** A total of 193 women had vaginal *E. coli* colonization; the prevalence of colonization was 19.9%. *E. coli* colonization was documented in 119 (14.4%) of the 827 neonates. In 22.7% of the neonates, the isolated *E. coli* colonies were resistant to ampicillin (Table 1). The percentage of am-

Table 1. Antibiotic Susceptibility Profiles for *E. coli* Strains Isolated from Neonates (n=119)

Antibiotic	Susceptible	Resistant
Ampicillin	92 (77.3)*	27 (22.7)
Piperacillin	109 (91.8)	10 (9.2)
Ampicillin/sulbactam	114 (95.8)	5 (4.2)
Piperacillin/tazobactam	118 (99.2)	1 (0.8)
Ciprofloxacin	117 (98.3)	2 (1.7)
Cefuroxime	119 (100)	0
Ceftazidime	119 (100)	0
Cefotaxime	119 (100)	0
Imipenem	119 (100)	0
Meropenem	119 (100)	0
Amikacin	119 (100)	0
Gentamicin	119 (100)	0

Values are number (percentage).

\* $P \leq 0.002$ , compared with other antibiotics.

picillin-resistant *E. coli* serotypes grown in cultures from the vagina was 25.9%. A total of 159 women with *E. coli* colonization transmitted the pathogen to 34 neonates; the prevalence of perinatal transmission was 21.3%. Eighty-five neonates infected with *E. coli* were born to women in whom vaginal *E. coli* colonization was not detected. There were no cases of sepsis caused by *E. coli* in the studied group.

**Risk Factors for Neonatal Colonization With *E. coli*.** A survey of the returned questionnaires showed that the proportion of missing responses ranged between 13% and 16%. Univariate analysis was applied to evaluate the following factors that could affect neonatal colonization with *E. coli*: demographic, social, and medical history, the course of pregnancy, sexual life, and personal hygiene factors. The study showed that the neonate-related factors – gestational age, birth weight, weight with respect to the gestational age, maternal diseases, known obstetric risk factors for early-onset neonatal infection, or antibiotic prophylaxis for the mother during childbirth – did not have any significant influence on neonatal colonization with *E. coli*. Meanwhile, according to the univariate analysis, the mothers' social and demographic factors, the birth completion tactics applied, the mothers' sexual habits, and their anthropometric data had a significant effect on neonatal *E. coli* colonization (Table 2).

The factors that were significantly different in the comparative analysis with respect to neonatal groups with and without *E. coli* colonization were analyzed by applying the multiple logistic regression model. Some variables were excluded with respect to their interrelationships. According to our study, the most important factor for neonatal colonization with *E. coli* was anal sexual intercourse (OR, 3.91; 95% CI, 1.87–8.19;  $P < 0.001$ ). Other factors important for neonatal colonization with *E. coli* are presented in Table 3.

**Discussion**

To our knowledge, this work is the first to identify the prevalence of *E. coli* colonization among women and neonates, frequency of perinatal trans-

Table 2. Risk Factors of Neonatal *E. coli* Colonization

Factor	<i>E. coli</i> + n=119	<i>E. coli</i> - n=708	Total n=827	P
Education				
Basic	25 (21.0)	108 (15.3)	133 (16.1)	NS
Secondary	49 (41.2)	251 (35.5)	30 (36.3)	NS
Professional college	13 (10.9)	76 (10.7)	89 (10.8)	NS
University	32 (26.9)	273 (38.6)	305 (36.9)	<0.05
$\chi^2=6.78; df=3; P=0.08$				
Employment				
Employed	79 (66.4)	551 (77.9)	630 (76.3)	<0.01
Not employed	28 (23.5)	109 (15.4)	137 (16.6)	<0.05
Studied	12 (10.1)	47 (6.6)	59 (7.1)	NS
$\chi^2=7.51; df=2; P=0.02$				
Times of delivery				
1st	58 (48.7)	400 (56.5)	458 (55.4)	NS
2nd	47 (39.5)	213 (30.1)	260 (31.4)	<0.05
3rd	6 (5.0)	63 (8.9)	69 (8.3)	NS
4th	2 (1.7)	18 (2.5)	20 (2.4)	NS
≥5th	6 (5.0)	8 (2.0)	14 (2.4)	NS
$\chi^2=10.06; df=4; P=0.04$				
BMI, mean (SD)	27.6 (4.6)	28.3 (4.6)	28.2 (4.6)	0.1
[median, 25th–75th quartile]	[26.4; 24.5–29.4]	[27.4; 25.1–30.5]	[27.2; 25.0–30.5]	–
mean rank	373.35	420.83	–	<0.05
Maternal <i>E. coli</i> colonization	34 (28.5)	125 (17.6)	159 (19.2)	<0.01
Mode of delivery				
Natural way	116 (97.5)	610 (72.0)	626 (75.7)	<0.001
Vacuum extraction	2 (1.7)	6 (0.8)	8 (1.0)	
Caesarean section	1 (0.8)	192 (27.1)	193 (23.3)	<0.001
$\chi^2=39.59; df=2; P<0.001$				
One sexual partner	53 (50.5)	232 (37.8)	285 (39.6)	0.02
Sexual intercourse during menstruations	28 (26.2)	105 (16.9)	133 (18.2)	<0.05
Anal sexual intercourse	13 (12.1)	25 (4.0)	38 (5.2)	<0.001
Using preservatives not during pregnancy				
Do not use at all	90 (84.1)	485 (77.5)	573 (78.5)	NS
With accidental partners only	1 (0.9)	34 (5.5)	35 (4.8)	0.03
Always	16 (15.0)	106 (17.0)	122 (16.7)	NS
$\chi^2=4.63; df=4; P=0.1$				

Values are number (percentage) unless otherwise indicated. NS, not significant.

Table 3. Logistic Regression Model Predicting the Risk of Neonatal *E. coli* Colonization

Variable	Possibility for Neonate to be Colonized With <i>E. coli</i> OR (95% CI)	P
Maternal colonization with <i>E. coli</i>		
No	1	
Yes	1.81 (1.12–2.93)	<i>P</i> <0.05
Maternal BMI		
>27	1	
≤27	1.77 (1.15–2.73)	<i>P</i> <0.01
No. of sexual partners		
>1	1	
1	2.01 (1.30–3.11)	<i>P</i> <0.01
Anal sexual intercourse		
No	1	
Yes	3.91 (1.87–8.19)	<i>P</i> <0.001
Maternal education		
University	1	
Other	1.70 (1.06–2.74)	<i>P</i> <0.05

Overall percentage, 85.3;  $\chi^2=2.33, df=8; P=0.97$  (by Hosmer-Lemeshow).

BMI, body mass index; OR, odds ratio; CI, confidence interval.

mission, and risk factors for colonization; therefore, the comparison of our findings with literature data is limited. According to several studies, vaginal *E. coli* colonization is detected in 7%–13% of pregnant women (9–11). Thus, the prevalence of vaginal *E. coli* colonization during pregnancy, reported in our study (19.9%), was higher than that detected in other studies. This could have been influenced by the fact that our hospital is a tertiary-level institution where women with the most severe pregnancy pathologies are referred for delivery. Of the 827 neonates included into the study, 14.4% were colonized with *E. coli*. As much as 71.4% of the colonized neonates were born to women who were not detected to have vaginal *E. coli* colonization. These neonates were likely to have been colonized with *E. coli* present in their mothers' feces, or the results of testing the samples obtained from the mothers were false-negative.

It has been established that premature rupture of the amniotic membranes is associated with 17% of the neonates being colonized with *E. coli*, and if the mother is colonized, the rate of neonatal colonization

with *E. coli* during childbirth reaches 50% (10, 15). In our study, the prevalence of perinatal transmission was significantly lower, i.e., 21.3%. This could have been influenced by the human factor – a number of people took sample swabs for culture studies.

*E. coli* has been noticed to become increasingly resistant to antibiotics. Nearly one-third of *E. coli* colonies isolated in our study were resistant to ampicillin (the resistance to this antibiotic was significantly higher than that to other antibiotics), while no colonies were found to be resistant to gentamicin. Compared to literature data, the resistance of *E. coli* to ampicillin was not high. The prevalence of neonatal sepsis caused by ampicillin-resistant *E. coli* was 30%–70% (reaching as much as 77%–85% in the group of neonates with very low birth weight), 3% of the causative agents being resistant to gentamicin (2, 16–18). We think that the relatively low resistance of the causative agents to ampicillin may be due to the fact that penicillin rather than ampicillin is used for antibiotic prevention in our country. However, there are data suggesting that either ampicillin or penicillin administered during childbirth may induce the resistance of *E. coli* to ampicillin (19). Rentz et al. stated that whenever possible, penicillin should be the medication of choice in the prevention of GBS-induced sepsis (20).

Our study also identified the risk factors for neonatal *E. coli* colonization. Gestational age or birth weight did not have any effect on neonatal *E. coli* colonization in our study, yet *E. coli*-induced sepsis occurred more frequently among the neonates of low birth weight and low gestational age – and it is this group where *E. coli* colonization is expected to be more common (2, 16, 17). Since our study included only 4 neonates with a birth weight of <1500 g and 13 neonates born at <32 weeks of gestation, this tendency was difficult to identify. A significant part of preterm neonates' mothers refused to participate in the study. We think that this may have been due to rather intimate questions of our questionnaire and the psychological state of mothers who delivered preterm children. On the other hand, the participation of all very low birth weight and low gestational age neonates could hardly be expected, as they require postnatal resuscitation most frequently. We think that it would be unethical to first obtain microbiological test swabs, which could lead to the worsening of the condition due to failure to provide timely medical assistance. Moreover, after certain manipulations, we would not be able to ensure that the microorganism was obtained from the mother rather than from the environment.

The risk of neonatal colonization was nearly double among mothers with vaginal *E. coli* colonization. We noticed that *E. coli* colonization was significantly more common among neonates who were born vaginally, compared with those born via

cesarean section, and thus we presumed that the principal mode of transmission of *E. coli* was during movement of the fetus through the genital tract. Women who had a higher education level and were employed significantly less frequently transmitted *E. coli* to their neonates. Multiple logistic regression analysis showed that a higher maternal education level resulted in a 41% reduction in the risk of neonatal *E. coli* colonization. These findings may be explained by both better hygiene habits of better-educated employed women and by them taking better care of their own health, resulting in better observation during pregnancy. Women who gave birth for the second time significantly more frequently transmitted *E. coli* to their children, although such a trend was not observed with a further increase in the number of childbirths. It is obvious that a woman's sexual life is important for both GBS and *E. coli* colonization (21, 22). Mothers of neonates colonized with *E. coli* significantly more frequently had sexual intercourses anally and during menstruations. This confirms the transmission of this microorganism via the fecal-sexual-neonatal route, and blood is a suitable medium for a more prolonged colonization of the vaginal mucosa with these microorganisms. Paradoxically, the risk of neonatal *E. coli* colonization was reduced by half if the mother had more than one sexual partner. This may be due to the fact that different people could carry *E. coli* of different phylogenetic groups that may possibly inhibit each other, but there is no any evidence for this statement. With contrast to GBS, neonatal colonization with *E. coli* was significantly less common among women with greater body mass index. An opposite trend was observed with the risk factors for BGS-induced EONS: in obese parturients with BMI >30, there was an 80% increased risk for verified neonatal EOGBS disease (23).

### Conclusions

Maternal colonization with *E. coli* was higher in this study than other studies (19.9%). Neonatal colonization rate was 14.4%. The resistance of *E. coli* to ampicillin was not high (22.7%). Improvement of the mothers' education and modification of their sexual habits might be significant factors in reducing neonatal colonization with *E. coli*. Further studies could determine the necessity of pregnant women's screening and universal recommendations.

### Acknowledgments

This study was funded by the Lithuanian State Science and Study Foundation (contract No. T-77/07). The authors thank Irena Nedzelskienė for statistical support.

### Statement of Conflict of Interest

The authors state no conflict of interest.

## References

1. Neto T. Group B streptococcal disease in Portuguese infants younger than 90 days. *Arch Dis Child* 2008;93:90-3.
2. Stoll BJ, Hansen N, Fanaroff AA, Wright LL, Carlo WA, Ehrenkranz RA, et al. Changes in pathogens causing early-onset sepsis in very-low-birth-weight infants. *N Engl J Med* 2002;347(4):240-7.
3. Royal College of Obstetricians and Gynaecologists. Prevention of early onset neonatal group B streptococcal disease. Guideline No. 36. 2003. Available from: URL: [http://www.rcog.org.uk/resources/Public/pdf/GroupB\\_strep\\_no36.pdf](http://www.rcog.org.uk/resources/Public/pdf/GroupB_strep_no36.pdf)
4. Verani JR, McGee L, Schrag SJ; Division of Bacterial Diseases, National Center for Immunization and Respiratory Diseases, Centers for Disease Control and Prevention (CDC). Prevention of perinatal group B streptococcal disease: revised guidelines from CDC. *MMWR Recomm Rep* 2010;59(RR-10):1-36.
5. Gibbs RS, Schrag S, Schuchat A. Perinatal infections due to group B streptococci. *Obstet Gynecol* 2004;104:1062-76.
6. Friedman S, Shah V, Ohlsson A, Matlow AG. Neonatal *Escherichia coli* infections: concerns regarding resistance to current therapy. *Acta Paediatr* 2000;89:686-9.
7. Barcaite E, Bartusevicius A, Tameliene R, Kliucinskas M, Maleckiene L, Nadisauskiene R. Prevalence of maternal group B streptococcal colonisation in European countries. *Acta Obstet Gynecol Scand* 2008;87(3):260-71.
8. Barčaitė E, Nadišauskienė R, Maleckienė L, Bartusevičius A, Tamelienė R, Markūnienė E, et al. Nėščiujų B grupės b hemolizinio streptokoko nešiojimas ir perdavimas naujagimiams. (Maternal carriage of group B streptococcus and transmission to neonates.) *Lietuvos akušerija ir ginekologija* 2007;10(4):346-53.
9. Krohn MA, Thwin SS, Rabe LK, Brown Z, Hillier SL. Vaginal colonization by *Escherichia coli* as a risk factor for very low birth weight delivery and other perinatal complications. *J Infect Dis* 1997;175:606-10.
10. Amstey MS, Lewin E, Colaice J. Vaginal colonization with invasive *Escherichia coli* during pregnancy. *Am J Obstet Gynecol* 1980;137(5):534-5.
11. Balaka B, Agbere AD, Baeta S, Kessie K, Assimadi K. Bacterial flora in the genital tract the last trimester of pregnancy. *J Gynecol Obstet Biol Reprod* 2003;32(6):555-61.
12. Maleckiene L, Nadisauskiene R, Stankeviciene I, Cizauskas A, Bergstrom S. A case-referent study on fetal bacteremia and late fetal death of unknown etiology in Lithuania. *Acta Obstet Gynecol Scand* 2000;79(12):1069-74.
13. National Committee for Clinical Laboratory Standards (NCCLS). Performance standards for antimicrobial susceptibility testing. 6th informational suppl. M100-S9. Wayne, PA; 1999.
14. Schrag S, Gorwitz R, Fultz-Butts K, Schuchat A. Centers for Disease Control and Prevention (CDC). Prevention of perinatal group B streptococcal disease. Revised guidelines from CDC. *MMWR Recomm Rep* 2002;51:1-22.
15. Asindi A, Archibong E, Mannan NB. Mother-infant colonization and neonatal sepsis in prelabor rupture of membranes. *Saudi Med J* 2002;23(10):1270-4.
16. Bizzarro MJ, Dembry LM, Baltimore RS, Gallagher PG. Changing patterns in neonatal *Escherichia coli* sepsis and ampicillin resistance in the era of intrapartum antibiotic prophylaxis. *Pediatrics* 2008;121:689-96.
17. Andreu A, Ortega E, Planes AM, Salcedo S. Evolution of perinatal *Escherichia coli* disease in the era of group B Streptococcus prophylaxis. *Med Clin (Barc)* 2001;117(14):521-4.
18. Huang YS, Wang SM, Liu CC, Yang YJ. Invasive *Escherichia coli* infection in infancy: clinical manifestation, outcome, and antimicrobial susceptibility. *J Microbiol Immunol Infect* 2002;35:103-8.
19. Edwards RK, Clark P, Siström CL, Duff P. Intrapartum antibiotic prophylaxis 1: relative effects of recommended antibiotics on gram-negative pathogens. *Obstet Gynecol* 2002;100:534-9.
20. Rentz AC, Samore MH, Stoddard GJ, Faix RG, Byington CL. Risk factors associated with ampicillin-resistant infection in newborns in the era of group B streptococcal prophylaxis. *Arch Pediatr Adolesc Med* 2004;158(6):556-60.
21. Meyn LA, Moore DM, Hillier SL, Krohn MA. Association of sexual activity with colonization and vaginal acquisition of group B *Streptococcus* in nonpregnant women. *Am J Epidemiol* 2002;155(10):949-57.
22. Manning SD, Tallman P, Baker CJ, Gillespie B, Marrs CF, Foxman B. Determinants of co-colonization with group B *Streptococcus* among heterosexual college couples. *Epidemiology* 2002;13:533-9.
23. Hakansson S, Kallen K. High maternal body mass index increase the risk of neonatal early onset group B streptococcal disease. *Acta Paediatr* 2008;97(10):1386-9.

Received 8 December 2011, accepted 29 February 2012