

PS-187 **DEVELOPMENT OF GUT MICROBIOTA: EFFECT OF INTRAPARTUM ANTIBIOTIC PROPHYLAXIS IN BREASTFED TERM NEWBORNS**

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Background and aims Intrapartum antibiotic prophylaxis (IAP) is the most effective strategy to prevent early-onset Group B Streptococcal (GBS) sepsis. A possible role of IAP on neonatal microbiota is assumed. We aimed to evaluate the effect of IAP on the bacterial colonisation of neonatal gut at 7 and 30 days of life (DOL).

Methods Term newborns, vaginally delivered, were recruited at 2 DOL and allocated into four groups. Group 1: infants exclusively breastfed, not exposed to IAP. Group 2: infants partially breastfed (receiving at least 50% of own mother's milk), not exposed to IAP. Group 3: infants exclusively breastfed, exposed to IAP. Group 4: infants partially breastfed, exposed to IAP. Faecal samples from the enrolled infants were collected at 7 and 30 DOL. The count of *Bifidobacterium* spp., assessed by real-time PCR, was compared between the four groups.

Results Fifty-five newborns were recruited: 25 in Group 1, 7 in Group 2, 17 in Group 3, 6 in Group 4. On day 7, IAP-exposed newborns showed a significantly lower count of *Bifidobacterium* spp. ($p < 0.05$). Among infants not exposed to IAP, *Bifidobacterium* spp. count was significantly higher in Group 1 compared to Group 2. On day 30, a significant increase in *Bifidobacterium* spp. count ($p < 0.05$) compared to day 7 was observed in all groups.

Abstract PS-187 Table

	<i>Bifidobacterium</i> spp. [Log(CFU/g)] [Median (IQR)]	
	Day 7	Day 30
Group 1	7.93 (7.40 – 8.27)	8.69 (8.02 – 9.44)
Group 2	6.56 (6.13 – 7.07)	8.43 (7.08 – 8.51)
Group 3	6.01 (5.71 – 6.47)	8.51 (7.83 – 9.01)
Group 4	6.01 (5.59 – 6.23)	8.68 (8.34 – 9.02)

Conclusions Early neonatal microbiota is significantly affected by IAP, resulting in a reduced *Bifidobacteria* colonisation. Breast-feeding promotes the development of bifidogenic flora and possibly contributes to increase *Bifidobacterium* spp. count in IAP-exposed newborns at 30 DOL.

PS-188 **INTESTINO-TROPHIC EFFECTS OF MINIMAL ENTERAL NUTRITION IN PRETERM AND TERM PIGLETS**

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Background and aims In preterm infants, slow advancement of minimal enteral nutrition (MEN), combined with parenteral nutrition (PN), may be important to increase food tolerance and minimise the risk of necrotizing enterocolitis (NEC). We hypothesised that MEN for five days would increase gut growth and gut hormone release relative to total parenteral nutrition (TPN).

Methods Preterm and term piglets were delivered by caesarean section and fed TPN or PN+MEN (MEN: 0–64 mL bovine colostrum /kg/d) for five days. From day 5–26 all pigs were fed total enteral nutrition with milk-replacer. Pigs were euthanized at 0, 5 or 26 days of age, and gut weight, mucosal volume, L-cell density and plasma levels of GIP and GLP-2 were measured.

Results Body weight gain was markedly reduced in preterm vs. term ($P < 0.01$). Relative to TPN feeding, MEN for 5 days increased relative gut weight, mucosa volume and plasma GLP-2 and GIP in both preterm and term pigs (all $P < 0.05$). At 26 days of age, mucosa volume tended to be higher in preterm MEN versus preterm TPN ($P = 0.11$), whereas relative gut weight, L-cell density and plasma GLP-2 and GIP levels were similar between term/preterm and MEN/TPN.

Conclusion Despite the compromised growth in preterm pigs, the intestine is highly growth-responsive to MEN just after birth in both preterm and term pigs. The effects of MEN on gut dimensions and gut peptide release are minimal after few weeks of full enteral nutrition. MEN provision may be important for short term gut maturation in preterm infants.

PS-189 **THE EFFECT OF ENTERAL FEEDING ON REGIONAL CEREBRAL OXYGEN SATURATION IN PRETERM BORN INFANTS**

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Background Preterm infants do not always demonstrate adequate cerebral vascular autoregulation (CAR). Higher intestinal demands after enteral feeding may compromise cerebral perfusion in these infants. It remains unclear whether enteral feeding influences cerebral perfusion in preterm infants.

Aim To assess the effect of enteral feeding on regional cerebral oxygen saturation (r_cSO_2) in preterm infants.

Methods This study was part of a larger prospective cohort study. We used near-infrared spectroscopy to measure r_cSO_2 , which is indicative for cerebral perfusion. We measured during two hours on postnatal days 2–5, 8, 15, 22, 29, and 36. Feeding times were manually recorded. We used multi-level analyses to compare preprandial r_cSO_2 values to postprandial r_cSO_2 values, both 10–30 min and 30–60 min after feeding.

Results We included 29 preterm infants with a median GA of 28+1/7 (range 25+1/7–30+4/7) weeks, and a median birth weight of 1025 (range 580–1495) grams. Compared to preprandial values, we found increased postprandial r_cSO_2 values 10–30 min after feeding (mean[SD]: from 67%[15] to 71%[13], $p = 0.014$), and 30–60 min after feeding (mean[SD]: from 67%[15] to 73%[14], $p = 0.000$) on day 8. We observed a trend of decreased r_cSO_2 values 30–60 min postprandial compared to

preprandial values on day 15 (mean[SD]: from 66%[11] to 64% [11], $p = 0.091$).

Conclusion Our results suggest that in preterm infants during their first 36 days of life, cerebral perfusion does not decrease the first hour after feeding. One might reason that most preterm infants may yet be able to regulate cerebral perfusion, or postprandial intestinal perfusion may not increase at all.

PS-190 THE EFFECT OF ENTERAL FEEDING ON REGIONAL INTESTINAL OXYGEN SATURATION IN PRETERM BORN INFANTS

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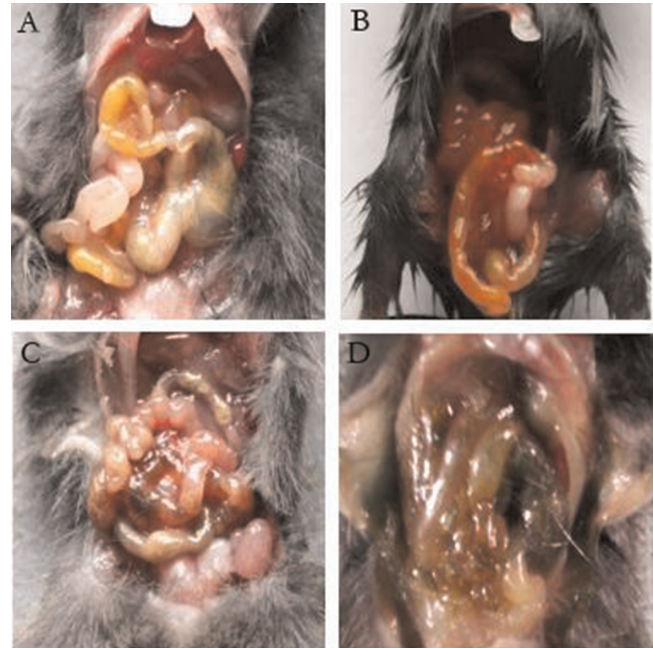
Background Term born infants demonstrate an increase in intestinal perfusion after receiving enteral feeding (postprandial intestinal hyperemia). It remains unclear whether enteral feeding influences intestinal perfusion in preterm infants.

Aim To assess the effect of enteral feeding on intestinal perfusion in preterm infants.

Methods This study was part of a larger prospective cohort study. We used near-infrared spectroscopy to measure regional intestinal oxygen saturation ($r_{int}SO_2$), which is indicative for intestinal perfusion. We measured during two hours on postnatal days 2–5, 8, 15, 22, 29, and 36. Feeding times were manually recorded. We used Multi-level analyses to compare preprandial (baseline) $r_{int}SO_2$ values to postprandial $r_{int}SO_2$ values, both 10–30 min and 30–60 min after feeding.

Results We included 29 preterm infants with a median GA of 28+1/7 (range 25+1/7–30+4/7) weeks, and a median birth weight of 1025 (range 580–1495) grams. Compared to preprandial values, we only found decreased postprandial $r_{int}SO_2$ values 30–60 min after feeding (mean[SD]: from 47%[22] to 41%[19], $p = 0.007$) on day 5. We observed increased $r_{int}SO_2$ values 10–30 min after feeding compared to preprandial values on day 29 (mean[SD]: from 51%[14] to 42%[14], $p = 0.005$).

Conclusion Our results suggest that in preterm infants during their first three weeks of life, intestinal oxygen saturation does not increase, and sometimes even decreases, the first hour after feeding. One might speculate that preterm infants are not yet able to increase intestinal perfusion rates after feeding, an increase which might be necessary to meet metabolic demands.



Abstract PS-191 Figure 1 TLR4-deficient mice were protected from the development of NEC. Abdominal anatomy of mice in the different groups. Wild type C57BL/10j mice (A) or TLR4-deficient C57BL/10ScNJ mice (C) were served as breast-fed controls. NEC was induced in newborn Wild type C57BL/10j mice (B) or TLR4-deficient C57BL/10ScNJ mice (D).

PS-191 TOLL LIKE RECEPTOR-4 SIGNALLING MEDIATED APOPTOSIS IN NECROTIZING ENTEROCOLITIS VIA CASPASES ACTIVATION

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Background and aim The importance of Toll-like receptor-4 (TLR4) signalling in necrotizing enterocolitis (NEC) is well-documented, but the potential mechanisms that regulate enterocyte apoptosis remain unclear. We investigated the role of apoptosis factors-Caspases in NEC and its pathway (endogenous and/or exogenous).

Methods TLR4-deficient C57BL/10ScNJ mice and Lentivirus-mediated stable TLR4-silent cell line (IEC-6) were used. NEC was induced by formula gavage, cold, hypoxia, combined with LPS *in vivo* or LPS stimulation *in vitro*. NEC severity was evaluated by histology. Enterocyte apoptosis was evaluated by TUNEL or Annexin analysis. The expression of TLR4, Caspases8, Caspase9, and Caspase3 were detected by qRT-PCR and western blot. Inflammatory factors including TNF- α , IL-6, IL-10 and IL-2 were examined by Luminex.

Results In TLR4-deficient mice, the severity of NEC was reduced, the expression of caspase8 was decreased, caspase9 was increased, and Caspase3 did not change significantly. In TLR4-silent IEC-6 cell line, expression of caspase3 was decreased and apoptosis rate were significantly reduced. Cytokine level of TNF- α and IL-2 were decreased.

Conclusion TLR4 induced apoptosis plays a critical role in the pathogenesis in NEC. Defect of TLR4 inhibits enterocytes from apoptosis in NEC, predominantly through Caspase8-mediated apoptosis pathway.

Abstract PS-191 Table 1 Sequences of PCR primers

Gene	Primer
β -actin	Sense Primer: 5'- CGTGAAAAGATGACCCAGAT -3'
	Antisense Primer: 5'- ACCCTCATAGATGGGCACA -3'
Caspase3	Sense Primer: 5'- TACAGCACCTGGTTACTATTTC -3'
	Antisense Primer: 5'-TACAGTTCITTCGTGAGCAT -3'
Caspase8	Sense Primer: 5'- GACATAACCCAACCTCCGAAA -3'
	Antisense Primer: 5'- TGCAGTGCAGTCGTCTGATA -3'
Caspase9	Sense Primer: 5'- CCGTGACATTGGTCTCTG -3'
	Antisense Primer: 5'- TCTTGGCAGTCAGGTCGTT -3'