



# Occurrence of IgE in foals: Evidence for transfer of maternal IgE by the colostrum and late onset of endogenous IgE production in the horse<sup>☆</sup>

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## Abstract

IgE is the key antibody involved in type I allergies. Allergen mediated crosslinking of IgE bound to high affinity Fcε-receptors on mast cells and basophils stimulates cellular degranulation and release of inflammatory mediators and cytokines. In this report, we demonstrate that IgE antibodies can be transferred from the mother to offspring in horses via the colostrum. We found a clear correlation between the IgE concentration in colostrum and the total IgE concentration in foal sera on day 2 after birth ( $r_{sp} = 0.83$ ). Maternal IgE was detected in foal sera by ELISA and on peripheral blood leukocytes of foals by flow cytometry. Both serum and cell membrane-bound IgE were undetectable in newborn foals before colostrum uptake and peaked on days 2–5 after birth. Cell-bound IgE became undetectable at 2 months after birth. Serum IgE disappeared from the circulation within the first 3–4 months of age. These kinetics suggest that the IgE antibodies which are detectable in foals during the first 4 months after birth are of maternal origin only. The endogenous IgE production was found to begin at 9–11 months of age, when IgE could be detected on peripheral blood leukocytes and in foal sera again. After 18 months of life, the total IgE concentrations in foal sera were comparable to those detected in their dams. The late onset of endogenous IgE production offers an explanation for observations that IgE mediated allergies are generally not observed in horses before puberty. The roles of the passively

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transferred maternal IgE in newborn foals are not yet known, but could be manifold, ranging from passive immunity and induction of immunoregulatory functions to determinative influences of maternal IgE on the antibody repertoire in the offspring. © 2005 Elsevier B.V. All rights reserved.

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## 1. Introduction

IgE and its role in allergy has been known for almost 40 years (Ishizaka et al., 1966). As in other species, equine IgE is believed to be involved in parasitic infections (Suter and Fey, 1981 and Suter and Fey, 1983) and in type I mediated allergic diseases (Matthews et al., 1983), such as chronic obstructive pulmonary disease (recurrent airway obstruction) (McGorum et al., 1993; Schmallenbach et al., 1998), summer eczema (Larsen et al., 1988; Ungar-Waron et al., 1990), and urticaria or head-shaking (Mansfield et al., 1998).

In adult horses, IgE can be detected readily in sera and on peripheral blood leukocytes. Total IgE concentration in equine serum ranges broadly from a few to hundreds of µg/ml, depending on the individual (Wagner et al., 2003). Similar IgE levels to those in horses were found in sheep (Shaw et al., 1997). Distinctly lower serum IgE concentrations were observed in humans from western countries (Janeway et al., 2001). These variations in the total IgE concentration in sera might be a result of different environmental and hygiene conditions in animals and humans. In particular, the parasitic burden in farm animals might influence and increase their total IgE levels.

Total IgE needs to be distinguished from the small subsets of antigen- or allergen-specific IgE antibodies, which all together contribute to the total IgE concentration. Allergic type I responses are induced by allergen-specific IgE antibodies only. These allergen-specific antibodies are crucial in the pathogenesis of the disease.

However, for some allergic diseases in humans a predisposition has been found for people with high total IgE concentrations in serum. Allergies such as asthma (Burrows et al., 1989; Sears et al., 1991; Platts-Mills, 2001) or atopic dermatitis (Kawai et al., 1992; Laske and Niggemann, 2004) are positively correlated with total IgE concentrations in the serum, while

others such as hay fever, could not be correlated to total IgE in serum (Burrows et al., 1989). In the horse, high concentrations of total IgE in serum of animals affected with a skin hypersensitivity called summer eczema could not be linked to allergy predisposition or occurrence of clinical signs of the disease (Wagner et al., 2003). Today, the number of studies addressing the linkage of total IgE concentrations in serum to predisposition to allergic conditions is still limited in equine medicine, and a correlation might exist for other allergic diseases of the horse.

In addition to its occurrence as a free antibody in serum, IgE can be detected on the cell surface of a small percentage of peripheral blood leukocytes ( $1.3 \pm 0.5\%$ ) in adult horses. The IgE<sup>+</sup> cells represented various cell populations which had bound IgE via Fcε-receptors, and were composed of approximately 32% basophils, 53% monocytes and/or large lymphocytes, 13% small lymphocytes and 2% eosinophils (Wagner et al., 2003).

In contrast to adult horses, foals (3–6 months of age) neither had detectable IgE in their sera nor detectable cell-bound IgE on peripheral blood leukocytes (Wagner et al., 2002, 2003). The finding that foals have no detectable IgE for a distinct period of life might offer an explanation for observations made in equine summer eczema: the development of clinical signs of skin hypersensitivity could never be observed in horses until they reached an age of two to 4 years, even if they were exposed to the allergen from birth (Wilson et al., 2001).

This study was inspired by a previous observation we made in a 4-day-old foal: IgE<sup>+</sup> leukocytes were detectable in the peripheral blood of this foal in numbers comparable to adults. Horses have a noninvasive epitheliochorial placenta (Mosmann, 1987; Allen and Stewart, 2001). This form of placentation does not allow any transfer of immunoglobulins from the mother to the fetus during pregnancy (Jeffcott, 1972), and immunoglobulins detected before colostrum uptake are considered to

be of fetal origin only. In the case of the foal mentioned above, we hypothesized that the IgE detected on peripheral blood leukocytes might have been of maternal origin and was passively transferred to the foal after birth. The aims of the work described here were (1) to investigate whether maternal IgE can be detected in foals after birth, (2) if maternal IgE is present, how it is transferred, and (3) at which age the endogenous production of IgE starts in the horse.

## 2. Material and Methods

### 2.1. Horses and Samples

The horses used for this study were of mixed breeds (Warmblood, Thoroughbred, Draft horse and pony). When the foals were born, the mares were 8–18 years of age. Serum and heparinized peripheral blood were collected from both mares and their foals at the day of birth (day 0) and on days 2, 5, 16, 30, 44, 58, 72, 86 post partum. The samples from the foals on day 0 were taken before colostrum uptake. In addition, a colostrum sample was collected from the mares within 30 min after foaling. Four of the foals were still available after day 86 and samples were continuously collected every 4 weeks up to 18 months of age.

### 2.2. ELISA

The ELISA to detect total serum IgE in the horse was previously described (Wagner et al., 2003). In brief: two monoclonal antibodies recognizing different epitopes of the IgE heavy chain constant region were used for coating (anti-IgE 176) and detection (anti-IgE 134), respectively. Serum samples were titrated in 2-fold dilution starting with a serum dilution of 1:200. The monoclonal anti-IgE detection antibody was biotinylated. After incubation of the biotinylated antibody, peroxidase conjugated streptavidin (Jackson ImmunoResearch Lab., West Grove, PA, USA) was added to the plates, followed by substrate solution. The same ELISA was used to measure IgE in the colostrum samples. The colostrum samples were spun down (2200 × g, 5 min, room temperature) to separate the fat. The lower hydrophilic phase was used as sample in the ELISA using an initial dilution of 1:200 which was titrated in two-fold dilutions. The IgE

concentrations were determined using purified serum IgE with a defined IgE concentration as standardized by recombinant IgE (Wagner et al., 2002). The analytic sensitivity of the ELISA was 20 ng/ml.

Determination of IgGa and IgGb in serum and colostrum by ELISA was performed as previously described, using a polyclonal goat anti-horse IgG(H + L) antibody (Jackson ImmunoResearch Lab., West Grove, PA, USA) for coating of the ELISA plates and the monoclonal antibodies CVS 45 (anti-IgGa) or CVS 39 (anti-IgGb), both generously provided by Dr. D.P. Lunn, for detection (Wagner et al., 1998).

### 2.3. Flow cytometric analysis

Peripheral blood leukocytes were isolated from heparinized equine blood and cell membrane-bound IgE was detected using the monoclonal anti-equine IgE 134 antibody and a secondary FITC conjugated polyclonal goat-anti mouse IgG(H + L) antibody (Jackson ImmunoResearch Lab., West Grove, PA, USA) as previously described (Wagner et al., 2003). Monoclonal mouse IgG1 was used for isotype control. A total of 100,000 peripheral blood leukocytes per sample were measured by flow cytometric analysis using a FACS Calibur<sup>TM</sup> (Becton Dickinson, San Jose, CA).

### 2.4. Statistical analysis

The data were tested for Gaussian distributions by the Shapiro-Wilks test which is a goodness-of-fit test for a Gaussian distribution (large *P* values indicate normality). When non-Gaussian distributions were indicated, the data were analyzed with rank-based tests, and summarized as minimum, median, and maximum. Gaussian data were summarized as mean and S.D., and Gaussian-based tests were used.

Differences between IgE concentrations in the serum and IgE<sup>+</sup> leukocytes in peripheral blood were created so that foals' values were subtracted from their own mares' values. The percentages of IgE<sup>+</sup> cells were reduced by the values of the corresponding isotype controls before the differences were calculated. Differences between mares and foals in serum concentrations at each day were tested by Wilcoxon's signed-rank test. Experiment-wise alpha was 0.05

(one-sided, assuming mares would have greater concentrations than foals). With Bonferroni correction for the 9 days of comparisons, the  $P$  had to be  $<0.0056$  for any one test to be considered significant. We also related the mare and foal concentrations with nine Spearman's rank correlations and Bonferroni-corrected  $P$ -values.

The associations of mare serum, colostral, and foal serum concentrations of IgE were evaluated by comparing the colostral concentration to the serum ones by Spearman's rank correlation after confirming reasonably monotonic patterns by scatter diagram. With Bonferroni adjustment, the  $P$ -value for significance was 0.025 (two-sided).

The data analyses were done with a commercial software package: Statistix<sup>TM</sup>8 (copyright 1985–2003; Analytical Software; Tallahassee, FL, 32317-2185).

### 3. Results

#### 3.1. Occurrence of IgE in foal sera after birth

The total IgE concentrations in sera of eight mares and their foals were determined by ELISA. Serum samples were obtained over a period of 3 months at several time points (Fig. 1). The first samples (day 0) were taken after birth and the foals were allowed to suckle colostrum immediately after sampling. At day

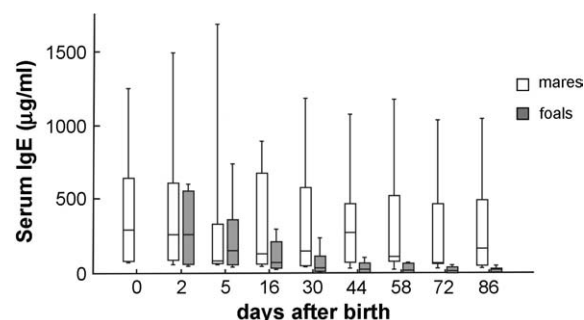


Fig. 1. Total IgE concentrations in sera of eight mares and their foals during the first 3 months after birth. IgE was detected in the sera by ELISA using two monoclonal anti-equine IgE antibodies which recognize different epitopes of the IgE constant heavy chain region. The sensitivity of the IgE detection using this assay was determined as 20 ng/ml. On day 0, samples of the foals were obtained before colostrum uptake and none had detectable IgE.

0, no IgE could be detected in any of the foal sera. On days 2 and 5 p.p., the total IgE concentration in foal and mare sera were not significantly different (both  $P = 0.10$ ). After day 5, the total IgE concentration in foal sera decreased by inspection constantly over time. A cut off of  $P = 0.0056$  was used for significance. Compared to the corresponding mare sera significantly lower total IgE concentrations were found in foal sera on days 0, 16 and 30 (all  $P = 0.004$ ). The difference on day 44 was  $P = 0.016$  and those for days 58–86 were suggestive of mares having higher total IgE concentrations in serum (all  $P = 0.008$ ). IgE in foal serum became undetectable by 3–4 months after birth.

A broad range of total serum IgE concentrations were detected in mare serum samples at all time points, and also in the foal sera on days 2 and 5 (Fig. 1). The individual serum IgE concentrations ranged from 27 µg/ml up to around 1.6 mg/ml in mares and from 41 µg/ml up to 742 µg/ml in foals (days 2 and 5). The mare IgE serum concentrations were reproducible for each animal over the 3-month period, i.e. individual mares had constantly low or high serum IgE concentrations with only small variations between time points by inspection (data not shown).

#### 3.2. Cell-bound IgE in foals during the first 3 months after birth

In foal sera, the highest IgE concentrations were observed on days 2–5 after birth. Afterwards, the IgE concentration decreased constantly. In vivo, soluble IgE antibodies are bound to high-affinity IgE receptors (FcεRI) on mast cells and basophils. Thereby, the soluble IgE detected on days 2 and 5 in foal sera might have disappeared from the serum by binding to free FcεRI on foal cells. In adult horses, peripheral blood leukocytes include approximately 1% of cells containing Fcε-receptor bound IgE on their cell surface. These cells were detected by anti-IgE antibodies in adult horses. The IgE<sup>+</sup> cells represent a mixed cell population, containing approximately 30% basophils (Wagner et al., 2003).

We isolated peripheral blood leukocytes from 12 foals and corresponding mares. The cells were labeled for membrane bound IgE and analyzed by flow cytometry. Isotype controls were included for each

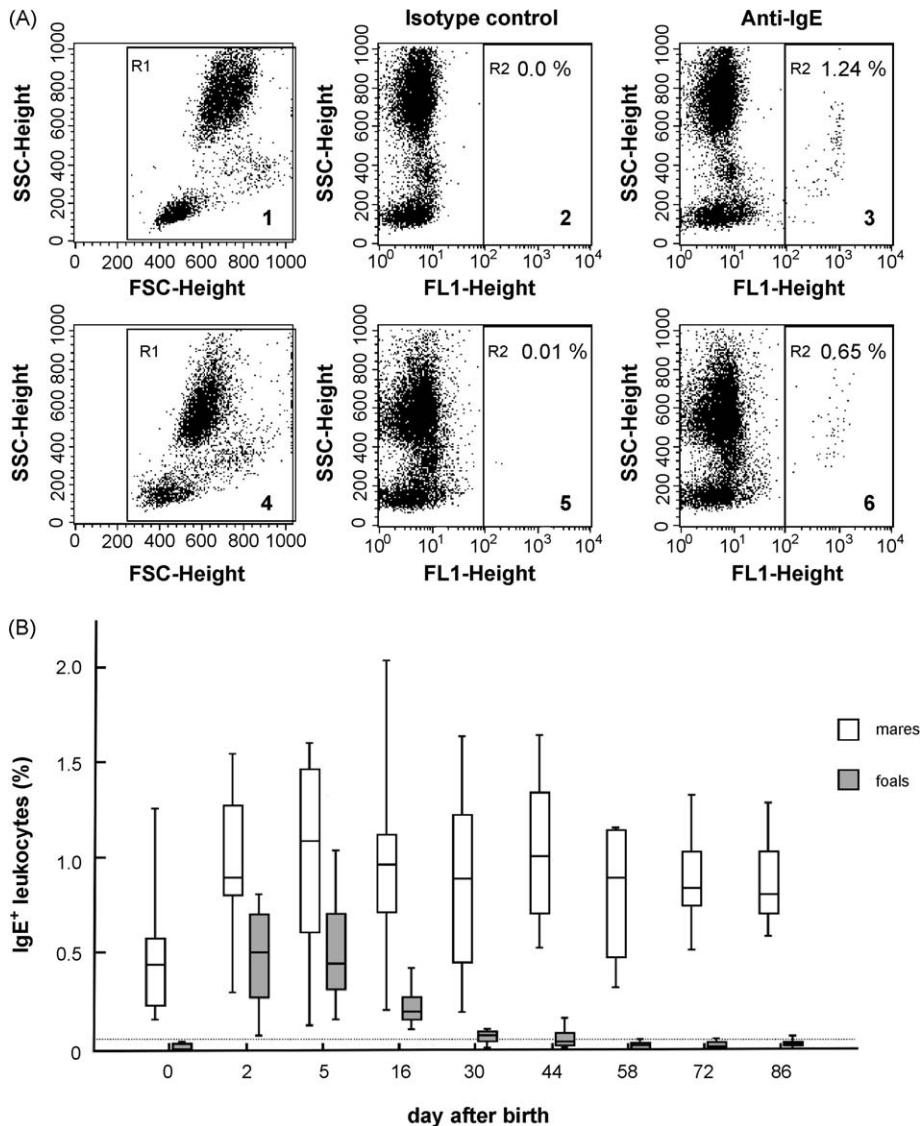


Fig. 2. IgE<sup>+</sup> peripheral blood leukocytes in mares and their foals were analyzed by flow cytometry for a period of 3 months after birth. (A) Equine peripheral blood leukocytes were stained for cell membrane bound IgE using monoclonal anti-equine IgE antibody and a secondary FITC conjugated goat anti-mouse IgG antibody. An irrelevant mouse monoclonal antibody was used for isotype control. Plots 1–3 show a representative sample of a mare: (1) Cell morphology, cells located in the R1 gate were analyzed for fluorescent labeling using (2) the isotype control and (3) the anti-IgE antibody for staining. Plots (4)–(6) represent the corresponding samples of a foal on day 5 after birth: (4) cell morphology, (5) isotype control and (6) anti-IgE. The percentages of IgE<sup>+</sup> cells in the gate R2 are given. (B) IgE<sup>+</sup> peripheral blood leukocytes of 12 foals and their mares during the first 3 months after birth. On day 0, blood samples of the foals were collected before colostrum uptake. The dotted horizontal line represents the median of the isotype controls. The percentage of IgE<sup>+</sup> cells in mares and foals were similar on day 5 ( $P = 0.02$ ). On all other days they were significantly different ( $P \leq 0.0049$ ).

sample (Fig. 2A). In agreement with the findings in foal sera, no IgE<sup>+</sup> leukocytes were detected in the peripheral blood of foals before colostrum uptake (day 0), confirming that no endogenous IgE is produced in

foals during intrauterine development. On days 2 and 5, IgE<sup>+</sup> leukocytes were detected in the peripheral blood of all foals (Fig. 2B). Similar to the total IgE concentration in foal serum, the number of IgE<sup>+</sup> cells

decreased afterwards. There was no significant difference between mares and foals on day 5 ( $P = 0.02 > \text{Bonferroni adjusted } \alpha = 0.0056$ ). On all other days, the IgE<sup>+</sup> leukocytes in foals were significantly lower than in mares ( $P \leq 0.0049$ ). After 2 months of age, IgE<sup>+</sup> cells were no longer detectable in the foal blood. When IgE was detected on foal leukocytes, the morphology of the IgE<sup>+</sup> cell population was comparable between mares and foals (Fig. 2A), suggesting that the foals expressed free Fcε-receptors shortly after birth or up-regulated them in response to free IgE antibodies in sera.

### 3.3. Maternal IgE is transferred with equine colostrum

After birth and before colostrum uptake, no IgE was detectable in foals. Both total serum IgE concentrations and cell-bound IgE on peripheral blood leukocytes showed highest levels on days 2–5 p.p. and decreased constantly afterwards. Cell-bound IgE became undetectable again at the age of 2 months after birth. Serum IgE disappeared completely from the circulation at 3–4 months of age. These kinetics suggested that the IgE antibodies which were detectable in foals after birth and within the first 3–4 months of life were of maternal origin only. To confirm this hypothesis, colostrum samples of 8 mares were tested for IgE antibodies by ELISA. IgE was detected in all colostrum samples with a median concentration of 773 µg/ml (Table 1). Compared to total IgE concentrations in the sera of the same mares on day 0, IgE was 2.7-fold enriched in the colostrum samples. IgGa and IgGb antibody isotypes in the same colostrum samples were 4.3-fold and 7.1-fold enriched, respectively (data not shown). The comparison of individual colostrum IgE concentrations to

Table 1

Descriptive statistics (and Shapiro-Wilks  $P$  for normality) of colostrum and total serum IgE concentrations (µg/ml) of eight mares on the day of birth and total IgE concentrations in the serum of their foals on day 2 after birth

| Sample         | N <sup>a</sup> | Minimum | Median | Maximum | $P$ -value |
|----------------|----------------|---------|--------|---------|------------|
| Colostrum IgE  | 8              | 185     | 773    | 2573    | 0.17       |
| Mare serum IgE | 8              | 66      | 254    | 1950    | 0.0048     |
| Foal serum IgE | 8              | 54      | 261    | 720     | 0.038      |

<sup>a</sup> N: number of samples.

Table 2

Spearman rank correlations ( $r_{sp}$ ) between colostrum and serum total IgE concentration of eight mares and their foals

| Variables                                                | $r_{sp}$ | $P$ -value |
|----------------------------------------------------------|----------|------------|
| Mare serum IgE <sup>a</sup> ; colostrum IgE <sup>a</sup> | 0.86     | 0.01       |
| Foal serum IgE <sup>b</sup> ; colostrum IgE <sup>a</sup> | 0.83     | 0.01       |

<sup>a</sup> At birth.

<sup>b</sup> On day 2 after birth.

total IgE in the sera of corresponding mares (day 0) and foals (day 2 after birth) showed a clear correlation (Table 2). Mares with higher serum IgE concentrations had also higher IgE levels in their colostrum ( $r_{sp} = 0.86$ ), and the IgE concentrations detected in foal sera on day 2 also were dependent on maternal colostrum IgE concentrations ( $r_{sp} = 0.83$ ). We concluded that the IgE detected in foals after birth was of maternal origin only, and that the foals did not produce endogenous IgE during the first months of life.

### 3.4. Onset of endogenous IgE production in the horse

Previous investigations in Icelandic horses indicated the lack of detectable IgE in foals at the age of 5 and 6 months (Wagner et al., 2003). Four foals of this study were available until they reached 18 months of age. Blood and serum samples were taken continuously from these animals to determine the onset of the endogenous IgE production in the horse. IgE could not be detected in these sera within the first 8 months of life by ELISA (Fig. 3). Depending on the individual foal, serum IgE was detectable by 9–11 months of age. After 18 months, the median total IgE concentration in the sera of these foals was 56 (range 50–81) µg/ml, and reached levels comparable to those of their mares

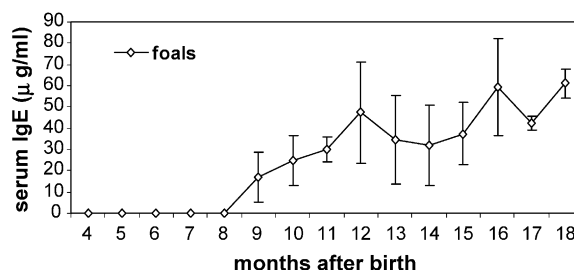


Fig. 3. Total IgE concentrations (means ± S.E.) in sera of four foals between 4 and 18 months of age were determined by ELISA.

(88  $\mu\text{g/ml}$ ; range 69–110  $\mu\text{g/ml}$ ). The occurrence of  $\text{IgE}^+$  peripheral blood leukocytes was observed at the same time in the foals, i.e. between 9 and 11 months of age, and the number of  $\text{IgE}^+$  cells reached adult levels at around 1 year of age (data not shown).

#### 4. Discussion

Maternal antibodies have been found to be important mediators of passive immunity providing the offspring with protection against infections until its own immune systems produces sufficient amounts of antibodies (reviewed by Silverstein, 2000; Zinker-nagel, 2001). Maternal IgG antibodies also have various active immunostimulatory functions which were found to have inductive effects on the neonatal immune system. Although the direct effects of maternal antibodies are restricted to a short period early in the neonatal life, their determinative influences can last life-long (for review: Lemke et al., 2004). Many of the effects of maternal IgG antibodies were found to be mediated by the formation of anti-idiotypic antibodies in the neonate. These anti-idiotypic antibodies play important roles in regulating the neonatal T- and B-cell repertoire, the magnitude of immune responses, and antimicrobial protection in the offspring (Martinez et al., 1986; Elliott and Kearney, 1992; Borghesi and Nicoletti, 1996; Montesano et al., 1999; Lemke and Lange, 2002). In addition, maternal IgG antibodies can induce allergen-specific suppression of IgE responsiveness in offspring (Jarrett and Hall, 1979) which is independent of antigen (Seeger et al., 1998; Lange et al., 2002). These effects of maternal IgG antibodies contribute to the current opinion that the susceptibility to allergic disease is highly influenced by events occurring early in life (Holt and Jones, 2000; Halken, 2003; Holt et al., 2005).

Depending on the form of placentation in different species, maternal antibody transfer is performed by active prenatal transportation across the placenta and/or with the colostrum and milk after birth. In large animals with diffuse, non-invasive epitheliochorial placentae, such as horses, cattle and pigs, the transfer of maternal antibodies only occurs with the colostrum and milk after birth (Jeffcott, 1972; McGuire et al., 1977). The major antibody classes, which are

transferred from the mother to the offspring are IgG and IgA. In addition to the data reported here for the horse, a few reports also described the presence of low levels of IgE in human, bovine and ovine colostrum (Thatcher and Gershwin, 1989; Duchén and Björkstén, 1996; Bendixsen et al., 2004). The roles of passively transferred maternal IgE antibodies in the neonate are unknown. In cattle, it was suggested that maternal IgE provides early protection from disease, especially against intestinal parasites (Thatcher and Gershwin, 1989). In contrast, the levels of IgE in human colostrum and milk were suggested to be too low to have a significant effect (Duchén and Björkstén, 1996).

In this report, we demonstrate that readily detectable amounts of maternal IgE antibodies are routinely transferred via equine colostrum to foals. The maternal IgE is detectable in the foal serum and also binds to a small percentage of peripheral blood leukocytes including basophils. The IgE antibodies which are detectable in foals after colostrum uptake are of maternal origin only. We found that young horses do not ordinarily produce endogenous IgE in utero and the first 8–10 months of life. During this time and after the maternal IgE antibodies have disappeared, neither serum IgE antibodies nor IgE bound to cellular Fc $\epsilon$  receptors could be detected in foals. This gap of complete lack of IgE lasts between 5 and 7 months in foals. Our finding in the horse is in contrast to the report in calves, which can produce endogenous IgE by 3 months of age (Thatcher and Gershwin, 1989). The kinetics of maternal and endogenous IgE in cattle seem to follow those known for IgG antibodies and maternal bovine IgE might provide protection against pathogens during the first 3 months of life, until the calf is able to produce sufficient amounts of its own IgE antibodies.

It is not known if there are consequences of the complete lack of detectable IgE in the foal's early life. Instead of IgE, alternative immune mechanisms or molecules (e.g., IgG antibodies) might provide immunity in the foal against parasitic and other antigens which induce IgE mediated protective immune responses in adults. The lack of endogenous IgE production in young horses underlines the more general observation that immune responses can differ with age. The major strategies and mechanisms of the immune response known today are mainly immune

mechanisms of young adults. Immune responses might be regulated in different ways over the lifetime of an individual, as determined by environmental pressures from pathogens and the internal control of cell growth. In the horse, neonatal infections are important and young foals are highly susceptible to infectious agents that only act as opportunistic pathogens in adults (Galan et al., 1986; Lopez et al., 2003). The transfer of sufficient concentrations of colostral antibodies, in particular of IgG, is crucial to protect the neonate against life-threatening infections (Jeffcott, 1972; McGuire et al., 1977). During intrauterine development, the foal's immune system can produce IgM (McGuire and Crawford, 1973), low concentrations of IgGa (Sheoran et al., 2000) and occasionally small amounts of other IgG isotypes (Wagner and Flaminio, unpublished observations). Compared to mare sera, IgGa, IgGb, IgG(T) and IgA were previously found to be enriched in colostrum and to appear in the circulation of newborn foals shortly after colostrum uptake. After loss of maternal colostrum-derived antibodies in foal serum, synthesis of endogenous IgGa, IgG(T), and IgA was detected within the first 2 months of life, whereas the endogenous production of IgGb was found to be delayed (Sheoran et al., 2000; Holznagel et al., 2003). The IgGb concentration remained still lower than the IgGa and IgG(T) levels until 8 months of age (Holznagel et al., 2003). This was a surprising finding because IgGb represents the major immunoglobulin in serum of adult horses and was suggested to play an essential role for protection against various bacterial and viral pathogens (Nelson et al., 1998; Lopez et al., 2002). Thus, not only the endogenous IgE production, but also the IgGb secretion is delayed in foals within the first 8–10 months after birth. Complementary but not yet identified immune mechanisms might compensate for the lack of IgE and IgGb antibodies early in life of the foal. Although there is yet no direct evidence for a link between the regulatory pathways that result in synthesis of IgGb and IgE, the expression of these two isotypes seems to be directly or indirectly suppressed or not activated for a certain period in foals.

In summary, the roles of the passively transferred maternal equine IgE antibodies are still unknown. In addition to the binding of maternal IgE to peripheral blood basophils reported in this study, we suggest that

colostral IgE might also be bound by high affinity Fcε receptors on local mast cells in the mucosa associated lymphoid tissues (MALT). This cell bound IgE could mediate passive protective immunity against pathogens, in particular intestinal parasites that invade the foal after birth. In addition, the maternal IgE antibodies might have a regulatory role to initiate adaptive immune responses in the foal. Similar to the regulatory effects found for maternal IgG (Lemke et al., 2004), maternal IgE might have determinative effects on the development of the T- and B-cell repertoire in foals. However, this hypothesis requires experimental confirmation, as it could offer an advanced explanation for family predisposition to allergic diseases.

## 5. Conclusions

The fact that horses develop IgE mediated allergies, have relatively high serum IgE levels, a large number of available cells to investigate rare FcεRI expressing cells, and a form of placentation that does not allow the transfer of any immunoglobulins in utero make the horse a valuable model to investigate the influences of maternal IgE on the neonatal immune response. In particular, the direct effects versus regulatory functions of colostrum IgE could be investigated in horses. This would improve our understanding of whether the predisposition to allergy, which is believed to have a strong genetic component, is also influenced by passively transferred maternal immunity.

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