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**Prenatal Contact with Inhalant Allergens**

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ABSTRACT

Pollen contact in early infancy may enhance the risk for subsequent pollen allergy. In this study likelihood of a prenatal antigen contact, as a result of inhalation of pollen allergens by the mother, was investigated. Due to the seasonal occurrence of allergens studied, the date of priming can be estimated, and this can supply data about the maturation of the fetal immune system. Proliferative responses of umbilical cord blood mononuclear cells (UCB MNCs) to the recombinant major allergens of birch (rBet v 1) and timothy grass (rPhl p 1) were analyzed throughout the whole year. A positive proliferative response was regarded as the criterion for a prenatal contact of the immune system with the allergen. Prenatal priming with both allergens was observed. Timothy grass pollen displayed considerably higher antigenicity than did birch pollen. The susceptibility of the fetal immune system to be primed by these allergens varies during the gestation period. The majority of positive responses to rPhl p 1 and rBet v 1 were found in UCB samples in which antigen contact (the respective pollen season) took place in the first 6 mo of pregnancy. Our results offer indirect evidence that, shortly after migration of T cell precursors to the epithelial thymus, T cells are mature enough for priming with antigens. No relationship was found between the susceptibility of the fetal immune system to be primed by these allergens and the clinical history of the family concerning type I allergy.

Abbreviations: **SI**, stimulation index; **Phl p**, major allergen of *Phleum pratense*; **r**, recombinant; **UCB**, umbilical cord blood; **MNC**, mononuclear cell; **Bet v**, major antigen of *Betula verrucosa*

Repeated contact with a potential antigen is possibly one of the main factors involved in the development of allergic diseases (1). Antigen-specific T cells, primed at the first contact, expand at further exposure. This, depending on the genetic predisposition, can lead to B cell stimulation and differentiation to IgE-producing plasma cells, presumably responsible for allergic symptoms. The early postnatal period has been identified as a time of increased risk for primary sensitization to aeroallergens (2-5). However, the occurrence of prenatal priming and its importance regarding a possible later allergic status is not fully clarified (6, 7). In the presence of phytohemagglutinin, T cells isolated from fetal thymus tissue proliferate as early as 10 wk of gestation (8). However, T cell responses to specific antigens during the whole time of gestation have not yet been investigated.

We studied the likelihood of a fetal antigen contact as a result of inhalation of antigens by the mother. As allergens are known to induce a strong immune response in predisposed individuals, we analyzed the proliferative responses of UCB MNCs to inhalant allergens, such as the recombinant major allergen of birch (*Betula verrucosa*) and timothy grass (*Phleum pratense*) throughout the whole year. Positive proliferative responses were regarded as an indication of a prenatal contact with these inhalant allergens. The pollen allergens are present in the environment only at a certain time of the year (in Austria, birch pollen from March until the middle of May, grass pollen from May until the middle of August). Due to this seasonal occurrence, positive proliferative responses of the cord blood lymphocytes to these allergens can supply data about the date of priming, and consequently data about the maturation of the fetal immune system during the whole time of gestation. Furthermore, we investigated

the relationship between the ability of the fetal immune system to be primed by these allergens and the clinical history of the family concerning type I allergy.

METHODS

Subjects. Cord blood samples were obtained by aspiration from the umbilical vein of the placenta of 114 normally delivered term babies at birth, between January 1994 and April 1995. The samples were taken randomly, independently of the clinical history of the family. Clinical history concerning type I allergy was established by interviewing the parents.

Measurement of total and specific IgE. Cord serum was analyzed for total IgE with a commercially available enzyme-linked immunoassay (Kallestad, Sanofi Diagnostics Pasteur, sensitivity, 0.5 IU/mL). In cases when maternal blood was also available ($n = 81$), sera were assessed for specific IgE to birch and timothy grass pollen (RAST system, Pharmacia Biotech Inc., Piscataway, NJ) and for total IgE.

Five groups were defined according to the allergic status of the family (parents and/or siblings). Group A, pollen (birch, grass, hazel, alder, rye) allergy; group B, other IgE-mediated allergies (food, animal dander); group C, elevated specific IgE to birch and/or grass pollen (RAST class 2 or higher) in the maternal serum without clinical manifestation with or without elevated total IgE level; group D, elevated total IgE level (>100 IU/mL) without elevated specific IgE to birch and/or grass pollen and without clinical symptoms; and group E, negative allergic status, no allergic manifestation in the family, no elevated specific IgE to birch and/or grass and low level of total IgE in the maternal serum.

Cell culture. MNCs were obtained by gradient centrifugation over Ficoll-Paque (Pharmacia). Cells, 10^5 /well, were cultured in 96-well round-bottom plates at 37°C , 5% CO_2 in Ultra culture medium (BioWhittaker, Walkersville, MD) supplemented with 2 mM L-glutamine, 1% heat-inactivated human serum, 20 μM 2-mercaptoethanol, and 170 mg/L gentamicin in the presence of rBet v 1 and rPhl p 1 in 0.5-2.0 μg /well concentrations. Recombinant allergens were produced according to Ferreira *et al.* (9) and Laffer *et al.* (10). Cultures with 4 U/well human rIL-2 (Boehringer Mannheim, Indianapolis, IN) served as positive control. After 6 d, MNCs were pulsed with 0.5 μCi of [^3H]thymidine (Amersham Corp., Arlington Heights, IL) for 16 h and then harvested. The radioactivity was measured by liquid scintillation counting. The SI is expressed as the ratio between the mean counts/min values measured in stimulated triplicate cultures and the mean counts/min measured in triplicate unstimulated cultures. Proliferative responses

with stimulation indices >2 were regarded as positive, representing a previous antigen contact.

RESULTS

Proliferative response of the UCB lymphocytes to rBet v 1 and rPhl p 1. The proliferative response of 114 UCB MNCs to rBet v 1 and rPhl p 1 was analyzed throughout the whole year. The results are summarized in Table 1. Positive responses to both allergens were obtained, 25.4% (29/114) to rPhl p 1 and 4.4% (5/114) to rBet v 1, respectively. The values of the positive SIs vary from 2 to 8 (data not shown). Furthermore, in several cases (particularly if SI was very high or proliferative responses were positive with both allergens or the mother was allergic to birch or grass pollen), the proliferative response of the maternal MNCs was also determined. Some of the data are shown in Table 2. There are remarkable differences between the SI values of the maternal and UCB MNCs. Especially interesting are the data for the P1 UCB-maternal blood MNCs, where SI of the maternal lymphocytes is remarkably higher than that of the UCB lymphocytes to rBet v 1, whereas to rPhl p 1 an opposite result was obtained.

Month of birth	No. of UCB samples	No. of positive proliferative responses	
		rBet v 1	rPhl p 1
January	10	0	4
February	13	0	10
March	12	0	0
April	10	0	0
May	15	1	2
June	10	0	0
July	5	2	2
August	8	1	0
September	15	1	4
October	5	0	0
November	3	0	1
December	8	0	4
Total = 114		4.4% of total	25.4% of total

Response with SIs >2 were regarded as positive.

Table 1. Proliferative response of UCB MNCs to rBet v 1 and rPhl p 1

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Patient code	SI of MNCs	
	Cord blood	Maternal
P1*		
rBet v 1	1	9
rPhl p 1	8	2
O1		
rBet v 1	5	2
rPhl p 1	7	2
W1		
rBet v 1	2	2
rPhl p 1	4	1
P2*		
rBet v 1	2	1
rPhl p 1	1	6
K1*		
rBet v 1	1	3
rPhl p 1	1	4

* Mother is allergic to birch or grass pollen.

Table 2. Comparison between the stimulation indices of cord and maternal blood MNCs

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The time course of the proliferative response. Table 3 shows the frequency of positive responses (expressed as percent of the number of samples investigated in the related month) during the whole gestation period. The months of pregnancy in which the mother was exposed to birch or timothy grass pollen are listed in the second and third column (birch pollen season, March-May inclusive; timothy grass season, May-August inclusive). Surprisingly, the UCB MNCs, which could only have had contact with the allergens in the third trimester, proliferated in none of the cases to rBet v 1 or in relative few cases to rPhl p 1. For rBet v 1 the majority of the positive responses were recorded when exposure to the birch pollen occurred during the middle of pregnancy (months of birth: July, August, September). The most sensitive period for prenatal priming to grass was found even earlier, around the end of the first trimester (months of birth: December, January, February).

Month of birth	Months of pregnancy during the pollen season		Responses of positive proliferative responses (%)	
	Birch pollen	Timothy grass	rBet v 1	rPhl p 1
March	1	1, 2, 3, 4	0	0
April	0	1, 2, 3, 4	0	0
May	1	1, 2, 3, 4	0	0
June	1	1, 2, 3, 4	0	0
July	1	1, 2, 3, 4	0	0
August	1	1, 2, 3, 4	0	0
September	1	1, 2, 3, 4	0	0
October	1	1, 2, 3, 4	0	0
November	1	1, 2, 3, 4	0	0
December	1	1, 2, 3, 4	0	0
January	1	1, 2, 3, 4	0	0
February	1	1, 2, 3, 4	0	0

Table 3. Frequency of positive proliferative responses of UCB MNCs in relation of allergen exposure of the mother during pregnancy

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Total IgE level of UCB and maternal sera. The total IgE level of the UCB serum samples ranged from 0.5 to 3.0 IU/mL. Of the maternal sera, 34.6% (28/81) had an elevated total IgE level (>100 IU/mL).

Relationship between the proliferative response of the UCB MNCs and the allergic

predisposition. The relationship between the SI values of the UCB MNCs and the clinical history of the family concerning type I allergy, as well as maternal total IgE level and RAST classes for birch and grass pollen, was investigated. The results are summarized in Table 4. For rBet v 1, the evaluation is difficult because of the low number of positive responses. However, the proliferation rates to rPhl p 1 show unambiguously that the genetic predisposition has no effect on the priming of fetal T cells.

Allergic status group	No. of UCB samples	No. and percentage of positive proliferative responses	
		rBet v 1	rPhl p 1
A	9	0 (0%)	2 (22%)
B	8	1 (13%)	0 (0%)
C	7	0 (0%)	2 (29%)
D	19	2 (11%)	4 (21%)
Positive clinical history A+B+C+D	Total = 43	3 (7%)	8 (19%)
E	38	1 (3%)	11 (29%)

Five groups were defined according to the allergic status of the family (parents and/or siblings). Group A, pollen (birch, grass, hazel, alder, rye) allergy; group B, other IgE-mediated allergies (food, animal dander); group C, elevated specific IgE to birch and/or grass pollen (RAST class 2 or higher) in the maternal serum without clinical manifestation with or without elevated total IgE level; group D, elevated total IgE level (>100 IU/ml) without elevated specific IgE to birch and/or grass pollen and without clinical symptoms; group E, negative allergic status, no allergic manifestation in the family, no elevated specific IgE to birch and/or grass and low level of total IgE in the maternal serum (n = 81).

Table 4. Relationship between the proliferative responses of UCB MNCs to rBet v 1 and to rPhl p 1 and the allergic status of the family

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DISCUSSION

We observed lymphocyte proliferation in cord blood MNCs to two inhalant allergens. Prenatal priming with timothy grass was much more pronounced than with birch pollen. It is remarkable that, according to statistical surveys in Austria, approximately twice as many individuals are allergic to grass pollens as to birch pollen (C. Ebner, unpublished data). Because in humans the migration of T cell precursors to the epithelial thymus takes place at 7-8.5 wk of gestation (11), positive proliferative responses should not be obtained with cord blood MNCs that had antigen contact exclusively in the first 2 mo of pregnancy (lymphocyte proliferation would indicate contamination with the maternal blood). As expected, we did not record proliferation in UCB samples collected in March and April for rPhl p 1, neither in samples from December and January for rBet v 1, where antigen contact could have occurred in the first 2 mo of pregnancy (Table 3). Furthermore, we observed considerable differences between SIs of UCB and the related maternal blood MNCs (Table 2). In many cases higher proliferation was recorded with UCB MNCs than with maternal MNCs. Taken together, these results indicate that proliferative responses are exclusively due to the proliferation of the cord blood MNCs. Consequently prenatal priming with both pollen allergens might occur. Piccinni *et al.*. (6) observed similar seasonal dependent reactivity of T cells to *Lolium perenne*.

In our study, no correlation was found between the atopic/allergic status of the family and the frequency of positive proliferative responses to rPhl p 1 (Table 4). The related results of other research groups are rather contradictory (6, 17, 18). We do believe that lymphocyte proliferation in the presence of a potential antigen has only secondary importance concerning the manifestation of allergic symptoms. We frequently record high proliferation rates to inhalant or food antigens in MNCs of adults who did not show any clinical symptoms and did not have elevated IgE to the related antigen (especially when the

individual investigated had a strong antigen exposure shortly before the test)(unpublished data). IgE production and the consequent clinical symptoms are much more related to the production of IL-4 by TH₂ helper cells after allergen challenge (19).

According to our results, the susceptibility of the fetal immune system to be primed with birch or grass pollen antigens varies during gestation. The majority of positive proliferative responses to our model allergens rPhl p 1 and rBet v 1 was obtained when maternal pollen exposure occurred during the first 6 mo of pregnancy. Our results offer indirect evidence that shortly after migration of T cell precursors to the epithelial thymus at 7-8.5 wk of gestation (11), T cells are mature enough for priming with antigens. The elevated lymphocyte proliferation rates measured with rPhl p 1 (in February, Table 3) indicate that the fetal immune system can already be primed in the 10-12th wk of gestation. Other studies also refer to related findings. According to Lobach *et al.* (12, 13), the lymphoid components of the thymus are established during the first trimester of gestation. CD3⁺ and TCR-[beta]⁺ cells were detected at 8.5 and 9.5 wk of gestation, respectively (14). The critical window of time for thymic development is between 7 and 14 wk of fetal gestation (12). Byrne *et al.* (15) found RO⁺RA⁻ T cells (believed to represent the memory cells) in spleen at 14-wk gestational age (in the youngest sample they analyzed).

The susceptibility of fetal lymphocytes for priming with birch and timothy grass pollen antigens shows a decreasing tendency toward the end of pregnancy (Table 3). Jones *et al.* (16) studied the proliferative responses of fetal lymphocytes to nutritive antigens in the course of pregnancy. They recorded higher proliferation ratios in the third trimester compared with the second for [beta]-lactoglobulin, whereas there was no difference for ovalbumin. The decreasing tendency we observed might correspond either to the decreasing permeability of the placental barrier for these two antigens or to an enhanced sensitivity of the fetal lymphocytes for priming during the early phase of pregnancy.

The use of seasonal allergens offers a helpful model for further investigation of the reactions of the fetal immune system upon antigen contact in several phases of gestation. Moreover, follow-up studies investigating the course of the clinical status of the individuals as well as the cellular responses to rBet v 1 and rPhl p 1 may provide new insights in the development of atopic diseases.

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Table 1

Month of birth	No. of UCB samples	No. of positive proliferative responses	
		rBet v 1	rPhl p 1
January	10	0	6
February	13	0	10
March	12	0	0
April	10	0	0
May	15	1	2
June	10	0	0
July	5	2	2
August	8	1	0
September	15	1	4
October	5	0	0
November	3	0	1
December	8	0	4
Total = 114		4.4% of total	25.4% of total

Responses with SIs >2 were regarded as positive.

Table 1. Proliferative response of UCB MNCs to rBet v 1 and rPhl p 1
 From: VAN DUREN SCHMIDT: *Pediatr Res*, Volume 41(1).January 1997.128-131

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Table 2

Patient code	SI of MNCs	
	Cord blood	Maternal
P1*		
rBet v 1	1	9
rPhl p 1	8	2
O1		
rBet v 1	5	2
rPhl p 1	7	2
W1		
rBet v 1	2	2
rPhl p 1	4	1
P2*		
rBet v 1	2	1
rPhl p 1	1	6
K1*		
rBet v 1	1	3
rPhl p 1	1	4

* Mother is allergic to birch or grass pollen.

Table 2. Comparison between the stimulation indices of cord and maternal blood MNCs
 From: VAN DUREN SCHMIDT: *Pediatr Res*, Volume 41(1).January 1997.128-131

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Table 3

Month of birth	Month(s) of pregnancy during the pollen season		Frequency of positive proliferative responses (%)	
	Birch	Timothy grass	rBet v 1	rPhl p 1
January	1	1, 2, 3, 4	0	60
February		1, 2, 3	0	76
March	9	1, 2	0	0
April	8, 9	1	0	0
May	7, 8, 9	9	7	13
June	6, 7, 8	8, 9	0	0
July	5, 6, 7	7, 8, 9	40	40
August	4, 5, 6	6, 7, 8, 9	12	0
September	3, 4, 5	5, 6, 7, 8	7	27
October	2, 3, 4	4, 5, 6, 7	0	0
November	1, 2, 3	3, 4, 5, 6	0	33
December	1, 2	2, 3, 4, 5	0	50

Frequency is expressed as percent of the number of samples investigated in the related month. The second and third column show the month(s) of pregnancy during which the mother was exposed to birch or timothy grass pollen.

Table 3. Frequency of positive proliferative responses of UCB MNCs in relation of allergen exposure of the mother during pregnancy

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Table 4

Allergic status groups	No. of UCB samples	No. and percentage of positive proliferative responses	
		rBet v 1	rPhl p 1
A	9	0 (0%)	2 (22%)
B	8	1 (13%)	0 (0%)
C	7	0 (0%)	2 (29%)
D	19	2 (11%)	4 (21%)
Positive clinical history A+B+C+D	Total = 43	3 (7%)	8 (19%)
E	38	1 (3%)	11 (29%)

Five groups were defined according to the allergic status of the family (parents and/or siblings). Group A, pollen (birch, grass, hazel, alder, rye) allergy; group B, other IgE-mediated allergies (food, animal dander); group C, elevated specific IgE to birch and/or grass pollen (RAST class 2 or higher) in the maternal serum without clinical manifestation with or without elevated total IgE level; group D, elevated total IgE level (>100 IU/ml) without elevated specific IgE to birch and/or grass pollen and without clinical symptoms; group E, negative allergic status, no allergic manifestation in the family, no elevated specific IgE to birch and/or grass and low level of total IgE in the maternal serum ($n = 81$).

Table 4. Relationship between the proliferative responses of UCB MNCs to rBet v 1 and to rPhl p 1 and the allergic status of the family

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