Nasal epithelial cells : effector cells in allergy
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Primary nasal epithelium exposed to house dust mite extract shows activated expression in allergics.

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Abstract

Nasal epithelial cells form the outer layer that protects against environmental factors. However this defense is not just physical, it has been shown that epithelial cells respond by producing of inflammatory mediators that may affect local immune responses. In this research we set out to characterize potential differences between the responses of nasal epithelium from healthy and allergic individuals to house dust mite allergen (HDM). These differences will help us to define local mechanisms that could contribute to allergic disease expression. Epithelial cells were cultured from nasal biopsies taken from five healthy and five allergic individuals. These cultures were exposed for 24 hours to culture medium containing house dust mite allergen, or to culture medium alone. Isolated RNA was used for microarray analysis. Gene ontology of the response in healthy epithelium revealed mainly up-regulation of chemokines, growth factors, and structural proteins. Moreover we saw increased expression of two transcription factors (NF-κB and AP-1) and their regulatory members. The expression pattern of epithelium from allergic individuals in the absence of the HDM stimulus suggests that it already is in an activated state. Most striking is that, while the already activated NF-κB regulatory pathway remained unchanged in allergic epithelium, the AP-1 pathway is down-regulated upon exposure to HDM allergen; this is contrary to what we see in healthy epithelium. Clear differences in the expression pattern exist between epithelial cells isolated from healthy and allergic individuals at baseline and between their responses to allergen exposure; these differences may contribute to the inflammatory response.
Introduction

The mucosal layer in the nose is constantly exposed to viruses, bacteria, parasites, and harmless allergens. It is crucial that a correct immune response is initiated to all these environmental factors. When harmless allergens are mistaken for dangerous pathogens the immune system will mount an unwanted inflammatory response to the allergens, resulting in allergic inflammation. An important player in the initiation of immune responses is the antigen presenting cell that resides in the mucosal tissue; the dendritic cell (DC). In recent years it has become increasingly clear that the peripheral DC initiates the immune response within an active local tissue environment, and that epithelial cells can play a role in this initiation process. Epithelial cells are more than a physical barrier and are themselves able to detect and respond to environmental signals. Epithelial cells can produce mediators that affect recruitment of immunocompetent cells to the local tissue and help create a microenvironment where these cells function ¹,²

In relation to the initiation of the immune response it is very interesting that epithelial cells produce mediators that can influence DCs. An example of such a mediator is MIP-3α, the chemokine for CCR-6 positive Langerhans cells (LCs), which is produced by bronchial epithelial cells after a variety of stimuli ³,⁴. Not only recruitment is affected by epithelial mediators, but epithelial expressed GM-CSF and TGF-β guide the differentiation of respectively, myeloid DCs and LCs from their precursors ⁵,⁶. Activation of tissue resident LCs is partly dependent on locally produced TNF-α and IL-1 ⁷,⁸, and recently TSLP (thymic stromal lymphopoietin) produced by epithelial cells was shown to be important in the activation of DC mediated allergic inflammation ⁹-¹¹. Currently, there are just a few players for which the effect on DC function has been documented. Even less is known on potential differences in mediators produced by epithelial cells from healthy or allergic individuals or if any of these differences contribute to the expression or development of allergic
In previous experiments we have investigated the epithelial response of a bronchial epithelial cell line H292 to house dust mite allergen. There we could show that epithelial cells display a broad and diverse expression of genes in response to exposure to allergen and that a substantial number of these regulated genes have a function in cell communication based on their gene ontology classification. Moreover we identified a potential regulatory network centered around TNF-α and NF-κB.\textsuperscript{12}

In this research we wanted to expand on these observations and investigate the response induced by house dust mite allergen in primary epithelium from healthy and house dust mite allergic individuals. Cultures of primary epithelial cells obtained from nasal biopsies were exposed to house dust mite extract diluted in culture medium, or with culture medium alone. RNA from this experiment was used for microarray analysis and the resulting expression profiles were subjected to bioinformatical, biostatistical, and interaction network analysis. Characterization of potential differences in the expression pattern at baseline or in response to allergen exposure will provide valuable insight into the role of the epithelium in the allergic response. Identification of the mechanism by which nasal epithelium influences the allergic response can lead to development of new therapies that target the epithelial cells.

Materials and Methods

Patient characteristics.

This study was reviewed and approved by the medical ethical committee of the Amsterdam Medical Center and all participants read and signed an informed consent. Five allergic volunteers (age 19-55) and five healthy non-smoking volunteers (age 21-32) were selected based on skin prick test for house dust mite (HDM) and other common allergens, and a nasal allergen
provocation to assess their response. Only mono-typically HDM allergic and non-allergic volunteers were included. Allergic individuals had refrained from using any medication for their allergy in the four weeks prior to the visit when biopsies were taken. Biopsies were taken from the lower edge of the inferior turbinate, 1 and 2 cm from the anterior end, using Fokkens’ forceps with a cup diameter of 2.5 mm. Local anesthesia was achieved by application of adrenalin and cocaine under the inferior turbinate without touching the biopsy site, during 10 minutes.

**Primary epithelial cell culture.**

Primary cells were obtained by digesting nasal biopsies of volunteers with 0.5 mg/mL collagenase 4 (Worthington Biochemical Corp., Lakewood, NJ, USA) for 1 hour in Hanks’ balanced salt solution (Sigma-Aldrich, Zwijndrecht, the Netherlands). Subsequently cells were washed with Hanks’ balanced salt solution (HBSS) and resuspended in BEGM (Lonza Clonetics, Breda, the Netherlands) and seeded in two wells of a 6 wells plate. Culture medium was replaced every other day. Cells were grown in fully humidified air containing 5% CO₂ at 37°C.

**House dust mite extract and exposure experiment.**

House dust mite extract containing biologically non-relevant trace amounts of LPS was kindly provided by Prof. Dr. M. L. Kapsenberg (AMC, Netherlands) as a lyophilized powder. It was dissolved in phosphate buffered saline (PBS), and then dialyzed against PBS and diluted to a stock concentration of 8 μg/μL. Cells were cultured for two weeks to 80% confluence and were subsequently pre-incubated with HBSS for 8 hours prior to exposure to HDM. Pre-incubation medium was removed and cells were exposed to HBSS containing a previously determined optimal concentration of house dust mite extract (2 μg/mL) or with HBSS alone for 24 hours. Supernatants were removed and stored for further analysis; cells were used for RNA extraction.
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RNA extraction.

Total RNA from each sample was extracted using Trizol (Life Technologies, Inc., Gaitersburg, MD, USA) according to manufacturer’s protocol, followed by purification by nucleospin RNA II (Machery-Nagel, Düren, Germany). The RNA concentration was measured on the nanodrop ND-1000 (NanoDrop Technologies inc., Wilmington, DE, USA) and RNA quality was checked on the Agilent 2100 bio-analyzer (Agilent Technologies, Palo Alto, CA, USA).

Microarray Affymetrix u133 plus 2.0.

Human Genome U133 Plus 2.0 Genechip Array (Affymetrix inc., Santa Clara, CA, USA) representing 47,000 transcripts, including 38,500 well-characterized genes was used in the analysis of HDM-induced genes. Technical handling of microarray experiments was performed at the MicroArray Department (MAD) of the University of Amsterdam (Amsterdam, The Netherlands), a fully licensed microarray technology centre for Affymetrix Genechip® platforms and official Dutch Affymetrix Service Provider. In short, biotin-labeled cDNA samples were prepared as described in the Affymetrix expression analysis technical manual (Affymetrix) using 3 μg of purified total RNA as template for the reaction. For this the One-Cycle cDNA Synthesis Kit (Affymetrix) was used. The Array images were acquired using a GeneChip Scanner 3000 (Affymetrix) and analyzed with Affymetrix GeneChip® Operating Software (Affymetrix).

Microarray data analysis and statistics.

The quality of the images was checked by visual inspection, and all raw data passed the quality criteria for average background, scale factors, percentage present calls, 3'/5' ratios GAPDH, 3'/5' ratios beta-actin, hybridization spike-in controls, poly-A spike-in controls. The data also passed a set of quality control checks provided by the affy, affyPLM and affyQCreport packages from Bioconductor (http://www.bioconductor.org/). Expression
values were calculated using the robust multi-array average (RMA) algorithm\textsuperscript{13}, and statistically analyzed for differential gene expression using ANOVA (MAANOVA package, version 0.98.8\textsuperscript{14}). The permutation based F$s$ test was used for hypothesis testing\textsuperscript{15}, and all p-values were adjusted for false discovery rate correction\textsuperscript{16}. In order to quantify the effect of HDM extract on gene expression, pair-wise statistical tests were performed to analyze: 1) the effect of HDM on epithelial cells from healthy individuals separately, 2) the effect of HDM on epithelial cells from allergic individuals separately, and 3) the difference in response to HDM in allergic compared to healthy individuals.

\textit{Ontology-, cluster-, and network analysis.}

Gene ontology was done using the online gene ontology program GOstat (http://gostat.wehi.edu.au/) where we used curated datasets and subsets to investigate the overrepresentation of gene ontology groups\textsuperscript{17}. Cluster analysis on all significantly affected genes was done by transforming the means of the expression values for a gene in the four groups (healthy or allergic after control exposure, or after HDM exposure) to Z-scores and using unsupervised K-means clustering based on correlation, for this we used Spotfire DecisionSite Functional Genomics. Network analysis was performed using Pathway Architect software (Stratagene, La Jolla, CA, USA), here we used the curated dataset to build a regulation interaction network. We overlaid the colors used in our K-means cluster analysis to clarify the relative expression changes of the genes in our network.

\textit{Quantitative PCR.}

Quantitative PCR was used to validate the differential expression of selected genes. Isolated RNA from control treated and HDM treated cells was used to synthesize cDNA using the MBI Fermentas first strand cDNA synthesis kit (Fermentas GmbH, St. Leon-Rot, Germany). PCR was performed...
on Bio-Rad iCycler (Bio-Rad, Veenendaal, The Netherlands). mRNA specific TaqMan® gene expression assays for ACTB, ATF3, CCL20, CNFN, EGR1, FLG, GAPDH, GATA3, GCSF, GROA, IL1RA, IL12B, IL13Rα2, IL8, KRT4, MCP1, MIP1B, PLAU, SPINK7, TNFA, TNFR1, TNFIP3 were ordered from Applied Biosystems (Nieuwerkerk a/d IJssel, The Netherlands). We performed all PCR assays three times, on all samples. Fold changes were calculated using the comparative Ct method.

Results

Validation of the microarray results.

After incubation with HDM extract, we identified 555 probe sets that were statistically differentially expressed in primary epithelium from healthy individuals and 301 probe sets that were statistically differentially expressed in primary epithelium from allergic patients. This collection of probe sets was first curated by, when possible, discarding the less specific x_at probe sets and the splice variant specific s_at probe sets. For analysis we further selected only those genes for which the expression level of their probe sets change by more than 1.5-fold (see supplemental table E1). In healthy epithelium the original 555 probe sets correspond to 209 uniquely annotated genes and 19 unannotated and/or hypothetical sequences in our curated dataset. Of these genes, 206 were up-regulated and 22 were down-regulated. In allergic epithelium the original 301 probe sets correspond to 87 uniquely annotated genes and 12 unannotated and/or hypothetical sequences in our curated dataset. Here 62 genes showed increased and 37 decreased expression.

The results of this microarray experiment were validated by independent real time PCR on the same starting material used for the microarray analysis. A selection of 20 genes that had revealed an increased, decreased, or unchanged expression level in either the healthy or allergic curated gene set was used for this validation experiment. After normalization for three
household genes (GAPDH, β-actin and β-2-microglobulin), the relative change in expression of these genes measured by PCR was directly comparable to the relative change obtained from our microarray experiment, both for the healthy epithelium \( (r = 0.621, p = 0.006) \) and for the allergic epithelium \( (r = 0.735, p < 0.001) \) (data is shown in supplementary table E2).

*Global analysis reveals an activated state in primary allergic epithelium.*

Within our curated dataset the expression of only a few genes (9/311) shows identical statistically significant changes upon HDM exposure in healthy and allergic epithelium. The expression of the other genes either change in healthy or in allergic epithelium, and in many cases shows a differential response in both groups (Figure 1).

![Figure 1](image_url)

The absence of an overlap in the response could be due to a differential change in the expression profile by the HDM extract, to intrinsic differences between healthy and allergic epithelium at baseline, or a combination of both. To investigate the relative contribution of these factors we first used Principal Components Analysis (PCA) on the whole data collection of microarray chips. Most of the variation observed for the chips (57 %, Figure 2) lies in the difference between healthy and allergic epithelium (PC1). What was unexpected that the next largest contribution (12 %) comes from the variability
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of the individuals within the healthy epithelium group (PC2), with only a very limited variation in the allergic epithelium group. Exposure to HDM extract only has third largest contribution (7 %) to the observed variation between all the genes in the healthy and allergic group.

Figure 2: Principal components analysis. A (left): showing the relative contribution to variance for the first 10 principal components. B (right): showing a scatter plot of all 20 arrays and their individual relative contribution to principal component 1 (PC1) and principal component 2 (PC2). Array ID’s are indicated in the graph.

The PCA showed that not only differences in response to allergen, but also differences at baseline between healthy and allergic contribute to variation; therefore we used K-means clustering on the expression levels in the four groups. This allows us to compare groups of genes that share specific expression patterns. The 311 genes that change their expression in healthy and/or allergic epithelium can be effectively separated into 12 clusters (Figure 3). Baseline expression of a given gene in 5 out of 12 clusters is substantially higher in allergic epithelium than in healthy epithelium. Together these clusters (number 7, 9, 10, 11, and 12) represent 74 % of all regulated genes. The K-mean cluster analysis shows that for some of these clusters expression after HDM exposure is high and remains largely unchanged in the allergic group and is up-regulated to a certain degree in the healthy group. For cluster 9 (8 genes) the expression level in healthy epithelium after HDM
exposure is even higher than in the allergic group, in cluster 11 (46 genes) the expression level is similar, while in cluster 12 (129 genes) the expression level in the healthy group does not quite reach the expression level of the allergic group. Evidently, a substantial group of genes in the epithelium from allergic individuals already displays an activated state at baseline. Interestingly, some clusters (number 3, 4, 7, 10) show changes in the allergic group while the expression in the healthy group remains unchanged, while two genes (cluster 5) even show an opposite response to allergen exposure.

Figure 3: K-means clusters. Here all 311 genes from curated dataset (see results section) were transformed to Z-scores, and were organized in 12 clusters using K-means clustering. Supplemental table 1 gives all 311 genes and their respective cluster number. AB = allergic at baseline, AH = allergic after HDM exposure, CB = healthy control at baseline, CH = healthy control after HDM exposure.
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The activated state in allergic epithelium is reflected by only a few ontology classes.

With only a limited number of expression profiles describing the effect of HDM exposure in healthy and allergic epithelium we wondered whether these patterns could be linked to specific functions of these genes.

Ontology analysis for genes that change their expression due to exposure to HDM extract in the primary epithelium from healthy individuals shows that these genes are principally involved in cell-to-cell contact. Cell communication (GO:7154) is by far the biggest group (66 genes) that is significantly overrepresented in the curated healthy data set \((p=2.4\times10^{-5})\). Related ontology groups are also overrepresented: signal transduction (GO:7165, \(p=4.6\times10^{-4}\)), receptor binding (GO:5102, \(p=2.8\times10^{-6}\)), extra-cellular space (GO:5615, \(p=8.9\times10^{-5}\)), and transcription factor activity (GO:3700, \(p=2.9\times10^{-6}\)). These changes seem to be a consequence of a general response of healthy epithelium to environmental factors as the ontology group response to external stimulus (GO:9605, \(p=4.4\times10^{-7}\)) and the related groups response to wounding (GO:9611, \(p=5.8\times10^{-6}\)), response to abiotic factors (GO:9628, \(p=1\times10^{-4}\)), and response to stress (GO:6950, \(p=2.4\times10^{-5}\)) are all significantly overrepresented in healthy gene set. Other processes that are affected are cell proliferation (GO:8283, \(p=3.3\times10^{-9}\)), (negative regulation) of cell death (GO:43069 \(p=3.5\times10^{-4}\) and GO:8219 \(p=1.9\times10^{-4}\) respectively), and intracellular junctions (GO:5911, \(p=3\times10^{-4}\)). A similar picture emerges when we combine the K-mean cluster analysis with the gene ontology class analysis. As clusters 9, 11, and 12 represent 58% of all genes that behave differently between healthy and allergic epithelium with expression in healthy epithelium going up by HDM exposure and expression remaining high in allergic epithelium, the activated status of allergic epithelium is best described by genes involved in cell communication and cell proliferation.

Although HDM extract also affects the expression pattern in the primary epithelium of allergic individuals, these genes do not seem to
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represent a specific gene ontology class. The only class that is significantly overrepresented is protease inhibitor activity (GO:0030414, $p=8.9 \times 10^{-3}$), but of this group only a few genes are statistically altered in allergic epithelium (SPINK-5, SPINK-6, SPINK-7, SERPIN-B3, C3, and WFDC-5).

Involvement of the NF-κB and AP-1 transcription factor complexes in the differential gene expression of healthy and allergic epithelium.

Now we found cell communication and cell proliferation activated in epithelial cells of allergic individuals, we wanted to find regulators that could be responsible for this activated state. To find these regulators we used regulation network analysis. In our network analysis (see Figure 4) we saw that the expected players involved in cell communication: inflammatory markers (IL-1α, IL-1β, and TNF-α), chemokines (IL-8, GRO-α, GRO-β, and IP-10), growth factors (EREG, AREG, and HBEGF), and receptors (TLR-3, PLAUR, PTGER-4, and IL-7R). More interestingly, this regulation seems to be mediated by proteins from the transcription factor complexes NF-κB (NFKB1, NFKB2, NFKBIA, and NFKBIZ), and AP-1 (FOS, JUN, JUNB, FOSL1, and ATF-3). When we use a color to identify in which cluster the genes from figure 3 are in the regulation network analysis we see that these transcription factor complexes also share a similar expression pattern. The activated state we identified above is also reflected at the transcription factor level, with the NF-κB and the non-canonical AP-1 transcription factors (JUNB and FOSL1) mapping to cluster 11. Interestingly, the canonical AP-1 transcription factors (FOS and JUN) behave differently. FOS and ATF-3 are expressed at similar levels at baseline in healthy and allergic epithelium (cluster 8) and expression goes up in healthy epithelium, but goes down in allergic epithelium. For JUN the baseline expression is higher in allergic (cluster 9), but again expression in allergic epithelium is down-regulated and in healthy epithelium is up-regulated upon allergen exposure.
Figure 4: Regulation Interaction Network. We used the 311 genes in the curated data set to create a regulatory network, showing known regulatory interactions between these genes. Colours correspond with the colours used in figure 3.
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Discussion

The first important observation coming from our data is that for a selective group of genes the allergic epithelium already is in an activated state. Baseline expression levels for these genes are higher in allergic individuals than corresponding levels in healthy individuals. Moreover for these genes, allergen exposure in healthy individuals leads to an increase in their expression levels, while the levels in allergic individuals remain largely unchanged. In our analysis of which processes are affected by the allergic status we find the genes belonging to the ontology classes cell communication and proliferation. In cell communication we find chemokines (IL-8, GRO-α, GRO-β, and IP-10) and cytokines (IL-1α, IL-1β, IL-1ε, and TNF-α) which have a known and well documented functions in the recruitment and activation of cells of the immune system. In cell communication we also find genes for growth factors (CTGF, HBEGF, AREG, EREG, and FGF-5) and intercellular junction (TJP-1, -2, CLDN-1, -4, and OCLN). These groups are known to be involved in the repair of damaged epithelium that, in this case, could be the consequence of cleavage of intracellular junction proteins by the protease activity contained within the HDM allergen extract. Where a substantial number of the regulated genes in epithelium from healthy individuals can be categorized in well-defined gene ontology classes, this does not hold true for the regulated genes in allergic epithelium. Only a single class of protease inhibitors is significantly enriched. Most likely this reflects a protective mechanism in allergic epithelium to counteract the activity of the protease activities contained within the allergen extract or those released by tissue resident mast cells (tryptase, chymase). Although relatively little attention has been given to the role of proteases in health and disease it is important to note that mutations in one of the protease inhibitors SPINK-5, as well as the protease ADAM-33, have been associated to asthma \(^{18,19}\). Other proteases like the matrix metallo-proteinases play a crucial role in the local
tissue remodeling associated with asthma \textsuperscript{20,21}.

In this research we set out to characterize the response of epithelial cells; in order to do so we chose to culture cells taken from a biopsy for two weeks, after which the cell cultures contain only epithelial cells, thus eliminating any contamination of gene expression by other cell-types. One of our concerns was that culturing the cells would alter the baseline expression, or the ability to respond, in a way that would prevent us from detecting differences between healthy and allergic individuals. Given that we detect so many well-established mediators that have been shown prior both \textit{in vitro} and \textit{in vivo} gives us confidence that the differences in the expression profiles are likely to reflect to situation \textit{in situ}. Another deliberate choice in our model was to stimulate the cells for 24 hours and then investigate the mRNA profile. We wanted to mimic the continuous exposure that the nasal epithelium is subject to \textit{in vivo}. As a consequence we were able to determine the relevant expression patterns of the NF-κB and AP-1 transcription factor families. However, our data do not reveal how this state is achieved after allergen exposure. For this purpose, earlier time points after induction should be investigated and this could well have consequence for the overall outcome of our experiment, with other genes and/or regulatory pathways playing a more prominent role.

There is little overlap in the response to HDM extract in primary epithelium from healthy and allergic individuals. As described above a substantial group of genes that are up-regulated in healthy individuals are already expressed at high levels in allergic epithelium even without exposure to allergen. To expand on this observation it would seem that the induced expression of these “activated” genes is part of the normal response in healthy primary epithelium. How is this activated status in allergic epithelium induced or maintained? Both healthy and allergic individuals are constantly exposed to house dust mite allergen in every day life. Epithelial cells of healthy individuals do express PAR-2 (Protease Activated Receptor 2), though at lower levels than in allergic individuals, so \textit{in vivo} activation of epithelial cells
by protease containing HDM extract prior to isolation could occur in both \(^2^2\). However, *in vitro* experiments with the epithelial cell line H292 have shown that the effect of PAR2 activation normally is transient, with expression levels of the responding genes returning to baseline levels 72 hours after the initial exposure to HDM extract. Therefore it does not seem likely that the activated state we see at baseline can be directly maintained during the two weeks of culturing that precedes our HDM-induction experiment, not without a positive feedback loop.

In previous work we have suggested that TNF-α, induced by HDM exposure, could be a central player both in mediating the HDM effect, as well as in restimulating epithelial cells. No matter whether we assume a direct effect of HDM or an indirect effect through a HDM-induced feedback loop, the outcome is different for healthy and allergic epithelium. This would lead us to conclude that the allergic status could be aggravated in allergic individuals because of an inability to shut this response down. This would explain the high level of correspondence between genes activated in healthy epithelium and those already activated in allergic epithelium. Alternatively, the “activated” status could be not related to the *in vivo* action of HDM on epithelium directly, but to the indirect effect that allergen-induced mediators from other tissue resident cells like mast cells have on the epithelium. However, it is not entirely clear how this would explain the correspondence between the HDM-induced genes in our *in vitro* experiment and the “activated” state in allergic epithelium.

When we assume a differential regulation of the HDM-response in epithelium of healthy and allergic individuals, this seems also reflected in the response of two important transcription factor complexes. The transcription factor NF-κB is a family of (hetero)dimeric proteins that has been linked to inflammation in many cell types, and is known to regulate (and to be regulated by) many different mediators \(^2^3;2^4\). That we find genes belonging to the NF-κB family up-regulated in response to allergen in epithelium from healthy individuals
is perhaps not surprising, but given the complex interactions between the family members it is hard to firmly establish NF-κB as the responsible factor for the observed induction of genes. The NF-κB proteins NFKB1/p105 and NFKB2/p100 are the cytoplasmic precursors of respectively the nuclear factors p50 and p52. Whereas homodimers of p50 or p52 are linked to transcriptional repression, the formation of heterodimers with the NF-κB REL-family members is linked to transcriptional activation. In our microarray experiments RELB is expressed at a considerably higher level in allergic compared to healthy epithelium and the expression levels are unaffected by allergen exposure. Interestingly, it has been described that knock-out mice for NFKB1 show a reduced stress response with lower IL-6 and COX-2/PTGS-2 levels, suggesting that at least in the overall outcome NFKB1 is required for a correct response to environmental signals. Given that the expression of these genes remains high when allergic epithelial cells are cultured for two weeks in absence of HDM suggests that NF-κB in allergic individuals could be (partly) responsible for maintaining the activated state. Although these observations could well explain the activated state in allergic epithelium or the induction of genes in healthy epithelium it fails to account for how this activated state is maintained or why this does not occur in epithelium from healthy individuals. Further experiments are needed to investigate whether epigenetic modification of key-regulators differs between healthy and allergic epithelium, or if the presence of an activating auto-feedback loop in allergic epithelium could be responsible for the maintenance of the allergic phenotype in tissue culture. Also, one might wonder whether the observed effect can be observed for other allergens and whether allergen-specific PAMP receptors could be involved in the response of epithelial cells to allergens.

What is interesting is that epithelial cells also show differential expression for some of the AP-1 family members. In these family members it is strange that the non-canonical members FOSL1 and JUND show the same pattern as NF-κB whereas FOS and JUN, the genes for c-FOS and c-JUN protein, do
show an increase in healthy individuals, but are actually down-regulated in allergic. If this expression could be linked to a protective mechanism against the effects of NF-κB activation, than this could explain some of the differences we see between healthy and allergic. It has been described that in *Drosophila* AP-1 is required to down-regulate NF-κB target genes by interfering with promoter binding. If this mechanism also applies in humans, then the up-regulation of FOS/JUN in healthy epithelium may contribute to the down-regulation of NF-κB regulated genes, and the absence of this response in allergic epithelium may contribute to the maintenance of the activated state seen in allergic epithelium. Interestingly, the FOS-related AP-1 family member ATF-3 displays a similar expression profile as the FOS gene itself. Identified as a stress factor, this bZIP protein in its homodimeric form acts as transcriptional repressor, but when it heterodimerizes with members of the JUN family it acts as a transcriptional activator.

Airway epithelium is becoming more widely accepted as an active player in the response to allergens, however it was unknown if the response to allergen exposure would differ between allergic and healthy individuals. We have shown that not only the response differs, but also the expression at baseline is different between healthy and allergic individuals. Linking our transcriptional observations to functional consequences is hard. Foremost, it is not clear how differences in the transcription level translate into effects on the protein level. This is further complicated by the formation of different homo- and hetero-dimers that each can have their distinct effects, by the functional interaction of the NF-κB and AP-1 transcription factors at promoter sites, and even by the formation of hetero-dimers between NF-κB, and AP-1 family members. Despite all these considerations the allergic epithelium seems an important target in the treatment, and possibly prevention, of allergic disease. Reducing the activated state in allergic epithelium may have direct consequence for the manifestation of complaints, for instance by reducing the influx of effector cells, and understanding how and why the
response in allergic epithelium differs from that in healthy epithelium may help in preventing the initiation of the allergic response.

Reference List

tests for differential gene expression by shrinking variance components estimates.
Biostat 6:59-75.
Ontologies within a group of genes. Bioinformatics. 20:1464-1465.
Experimental Allergy 34:325-327.
bronchial epithelium as a key regulator of airway inflammation and remodelling in
and K. Shirato. 1999. Inhibition of Matrix Metalloproteinases Prevents Allergen-Induced
and protein expression of protease-activated receptor 2 in structural and inflammatory
cells in the nasal mucosa in seasonal allergic rhinitis. Clinical & Experimental Allergy
36:1039-1048.
23. Cheng, D. s., W. Han, S. M. Chen, T. P. Sherrill, M. Chont, G. Y. Park, J. R. Sheller, V.
Controls Lung Inflammation and Injury through the NF-{kappa}B Pathway. J Immunol
178:6504-6513.
thymic stromal lymphopoietin in airway epithelial cells is controlled by NF{kappa}B.
PNAS 104:914-919.
2005. The role of intracellular Ca2+ in the regulation of proteinase-activated receptor-2
26. Heron, E., P. Deloukas, and A. P. van Loon. 1995. The complete exon-intron structure
of the 156-kb human gene NFKB1, which encodes the p105 and p50 proteins of
transcription factors NF-kappa B and I kappa B-gamma: implications for NF-kappa
regulates lipopolysaccharide-stimulated MAP kinase signaling by governing the stability
negative crosstalk between the AP1 and NF-kappaB signaling modules. Nat.Immunol
6:211-218.
**Supplementary table E1.** Genes that are statistically differentially expressed in healthy and/or allergic individuals after allergen exposure. Genes are given in alphabetical order, indicated are average fold changes and p-values for healthy or allergic. In addition the cluster number from the K-means clustering is also indicated.

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**Supplementary table 2.** Correlation between PCR and Microarray. All data given are fold changes given as geometric mean (range). Calculated from taqman Q-PCR (indicated as PCR) or microarray (indicated as MA) for healthy samples or allergic samples. In addition assay IDs as assigned by applied biosystems are also indicated. ND indicates no expression could be determined.

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