METHODS

Measurement of inflammatory mediators

A multiplex cytokine analysis was carried out with a FACscan (Becton-Dickinson, Franklin Lakes, NJ) with a cytometric bead array system (Becton-Dickinson). In brief, the system uses 5 separate bead populations of varying intensity fluorescence, each coated by antibodies recognizing the mediator of interest. These are mixed together with phycoerythrin-conjugated detection antibodies to which the samples or standards are added and then incubated. The subsequent signal generated is read on the FL-3 channel (Becton-Dickinson). Two separate kits were used: the chemokine kit for detection of IL-8 (sensitivity, 0.2 pg/mL), RANTES (sensitivity, 1 pg/mL), IP-10 (sensitivity, 2.8 pg/mL), IL-6 (sensitivity, 2.5 pg/mL), IL-10 (sensitivity, 3.3 pg/mL), TNF-α (sensitivity, 3.7 pg/mL), IL-12 (sensitivity, 1.9 pg/mL), and IL-8 (sensitivity, 3.6 pg/mL). The accuracy and precision of the bead array was confirmed by means of ELISA. Other mediators of interest not included above were also assessed by means of ELISA. ELISAs were used according to the manufacturer's instructions: IL-8 (sensitivity, 5 pg/mL), IL-6 (sensitivity, <3 pg/mL), IL-13 (sensitivity, <1.5 pg/mL), IL-4 (sensitivity, <2 pg/mL), IFN-γ (sensitivity, <4 pg/mL), and TNF-α (sensitivity, <0.09 pg/mL), all by Biosource International (Camarillo, Calif), and thymus and activation-regulated chemokine (sensitivity, <10 pg/mL) by R&D Systems (Minneapolis, Minn). IP-10 and IL-6 levels were measured in sera from acute asthmatic subjects (R&D Systems).

Blood was collected at presentation and 4 to 6 weeks later. Sera were assayed for IL-6, IL-8, RANTES, and IP-10 by means of ELISA, with the same sensitivities described above.

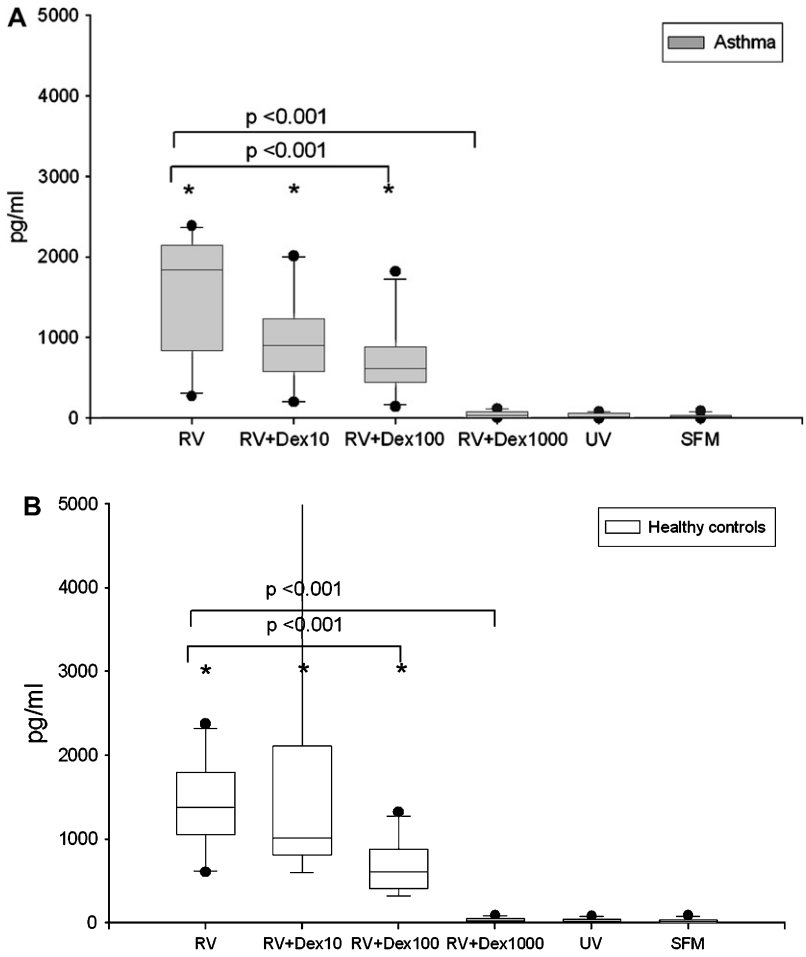


FIG E1. Induction of inflammatory mediators after rhinovirus infection in BECs from asthmatic and healthy control subjects. Subjects with asthma are represented by *hatched boxes*, and healthy control subjects are represented by *open boxes*. Data are summarized by box plots: *boxes* represent the 25th and 75th percentiles, the *central line* represents the median, and *solid circles* represent outliers. Differences between the groups were determined by using the Wilcoxon rank sum test. All responses are 48 hours after infection. All data are given in picograms per milliliter. Comparison between asthmatic and healthy control subjects was analyzed by using the Mann-Whitney *U* test. Within-group comparisons of results were analyzed by using the Wilcoxon rank sum test. *RV*, RV-16 infection at a multiplicity of infection of 2; *UV*, RV-16 inactivated by exposure to UV irradiation; *RV+Dex*, cells treated with dexamethasone at either 10, 100, or 1000 nM for 24 hours before infection with RV-16; *SFM*, cells treated with medium alone. *Significantly different from medium-only control. **A**, Induction of IP-10 after rhinovirus infection in primary BECs from asthmatic patients. **B**, Induction of IP-10 after rhinovirus infection in primary BECs from healthy control subjects. **C**, Induction of RANTES after rhinovirus infection in primary BECs from asthmatic patients. **D**, Induction of RANTES after rhinovirus infection in primary BECs from healthy control subjects. **E**, Induction of IL-6 after rhinovirus infection in primary BECs from asthmatic subjects. **F**, Induction of IL-6 after rhinovirus infection in primary BECs from healthy control subjects. **G**, Induction of IL-8 after rhinovirus infection in primary BECs from asthmatic subjects. **H**, Induction of IL-8 after rhinovirus infection in primary BECs from healthy control subjects. **I**, Induction of TNF-α after rhinovirus infection in primary BECs from asthmatic subjects. **J**, Induction of TNF-α after rhinovirus infection in primary BECs from healthy control subjects.

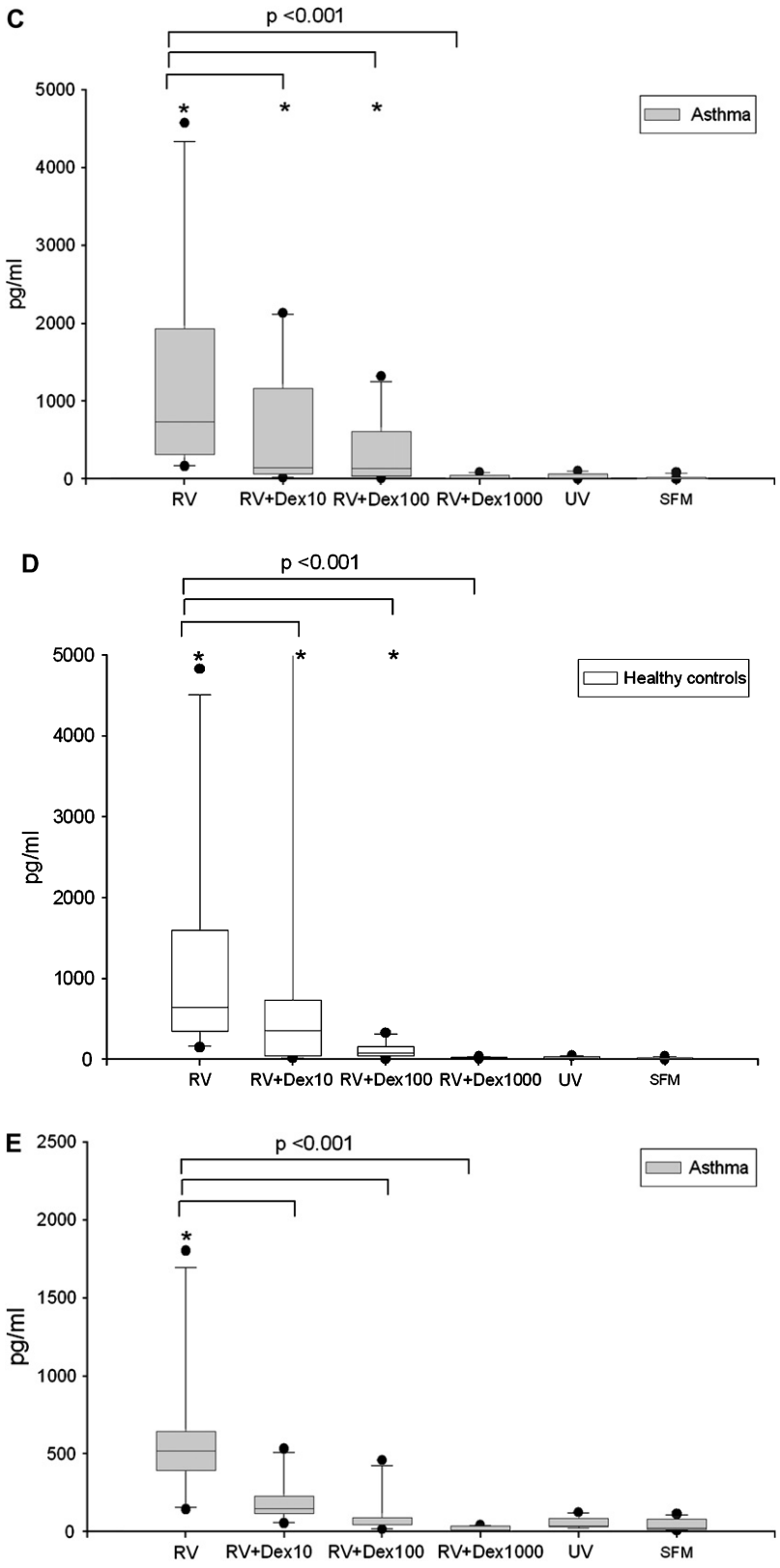
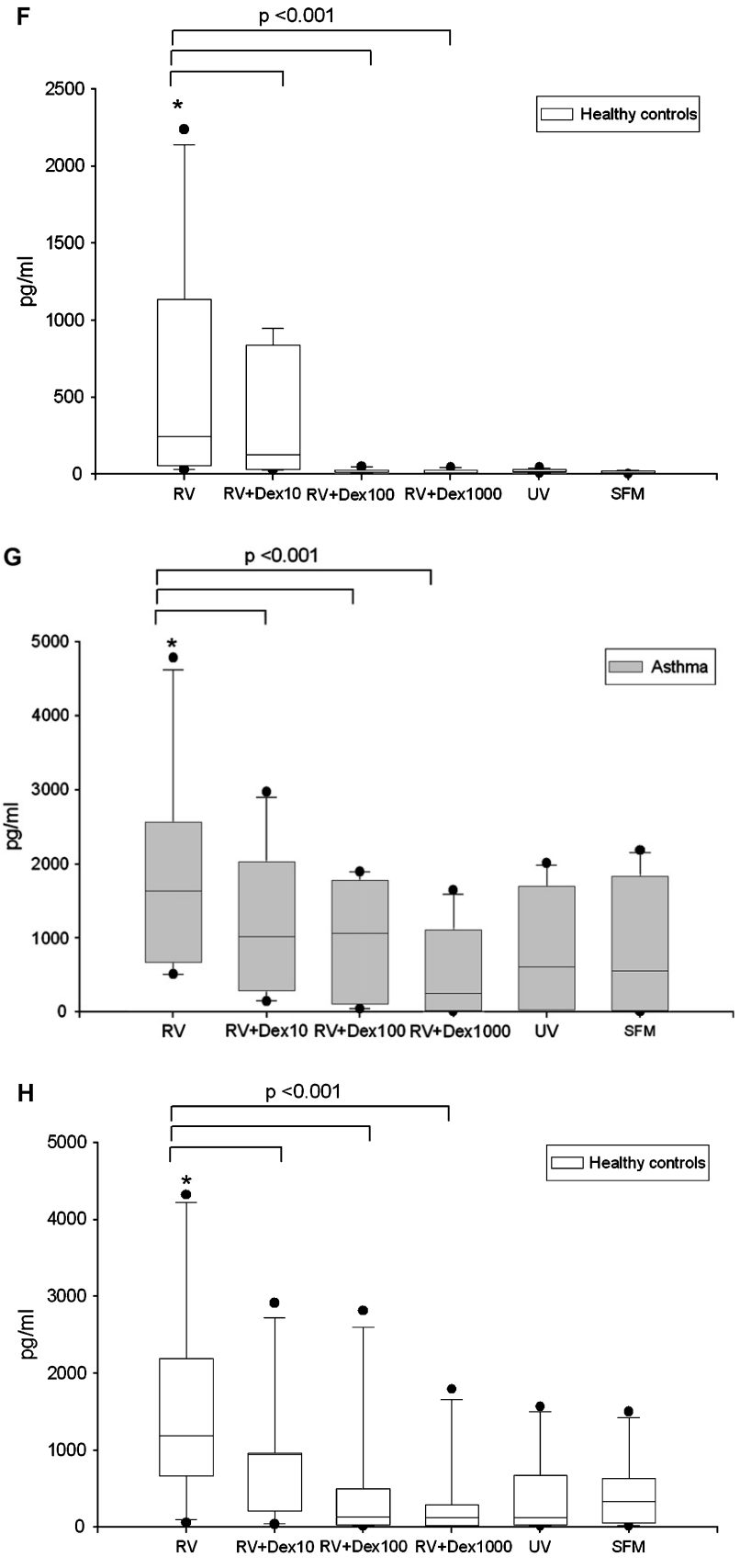


FIG E1. Continued.



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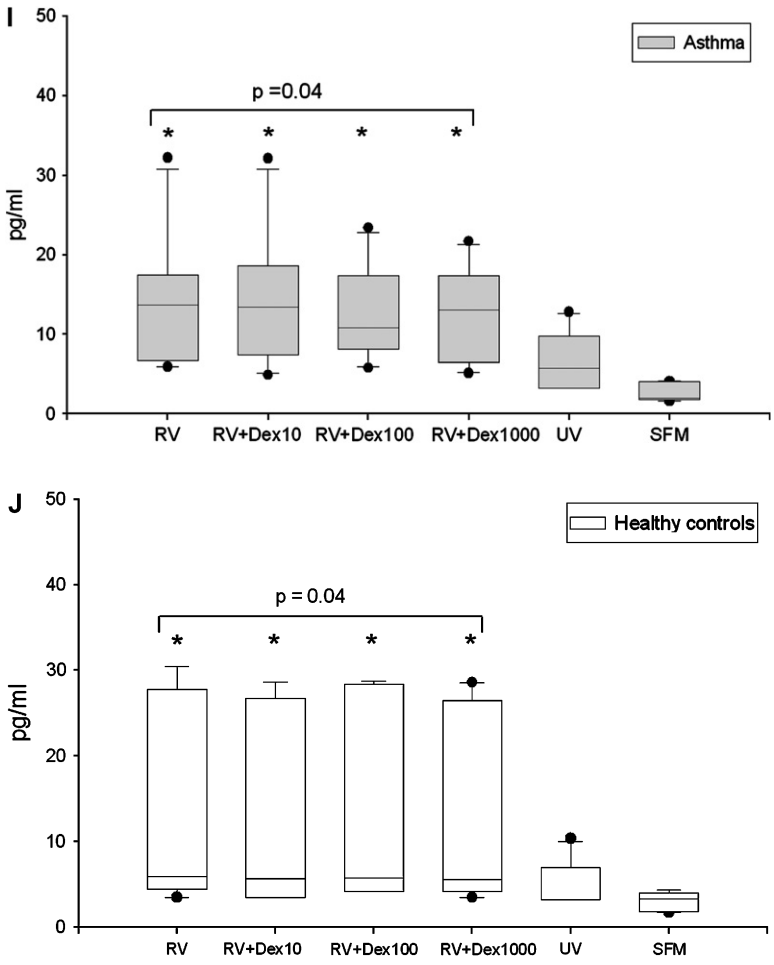

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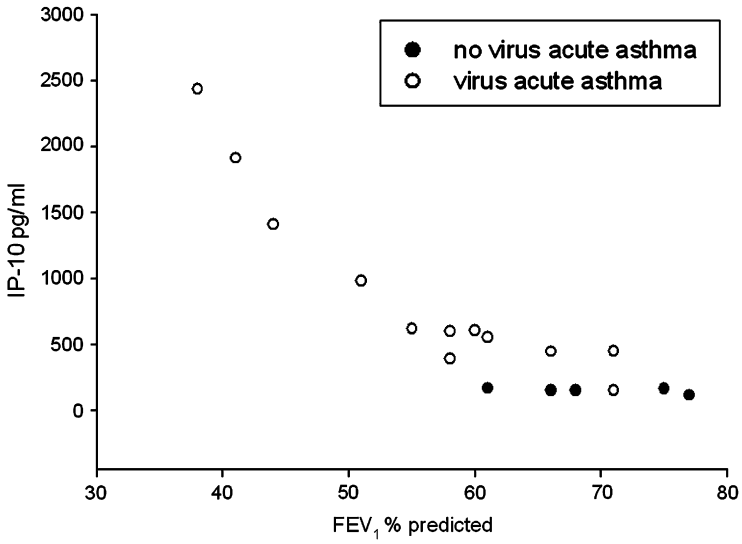

FIG E2. Relationship between serum IP-10 and FEV_1 percent predicted at acute asthma presentation. The following scatter plot compares serum IP-10 levels in individuals with their degree of airflow obstruction expressed as FEV_1 percent predicted at the time of acute presentation. Univariate correlations were analyzed by using the Spearman rank test.

TABLE E1. Subjects from whom primary BECs were cultured

	Healthy control subjects	Asthmatic subjects
Age (mean [SD])	30 (6.9)	31 (12.6)
Sex (male/female)	4/6	4/6
FEV_1 % predicted (mean [SD])	109 (7.7)	103.6 (9.8)
PC_{20} histamine (mg/mL; mean [SD])	>8	1.23 (0.43)*

*$P < .01$ compared with healthy control subjects (Mann-Whitney U test).

TABLE E2. Subjects recruited with acute asthma

	Acute virus-induced asthma	Acute non–virus-induced asthma	*P* value
N	20	10	NA
Sex (male/female)	8/12	4/6	.4
Age (mean [SD])	33 (11.2)	31 (14.1)	.7‡
ICS (BDP/d*; mean ± SD)	1075 ± 400	1080 ± 380	.8‡
Using ICS before presentation	22/24 (92%)	12/14 (86%)	.7§
FEV_1 % predicted (median [IQR])	56 (44-60)	68 (64-76)	.01‖
Change in FEV_1 after bronchodilator (%)†	0 (−4.2 to 8.2)	9.4 (5.4 to 29)	.04‖

ICS, Inhaled corticosteroid.

**BDP*/*d* is defined as the total dose of inhaled corticosteroids used expressed as a dose of beclomethasone.

†Significantly less than healthy control subjects, $P < .001$.

‡Student *t* test.

§χ^2 Test.

‖Mann-Whitney *U* test.