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# Serum periostin across asthma severity strata in Indian children: a single-center exploratory cross-sectional study

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## Abstract

# Background

Objective markers of asthma severity are limited in children, in whom spirometry is often unreliable or unavailable, and pediatric data on serum periostin from India are scarce.

# Methods

In this single-center, cross-sectional study in southern India, 80 children aged 6–18 years were enrolled by purposive sampling into four equal groups (control, mild, moderate, and severe asthma; 20 each). Severity followed the Global Initiative for Asthma 2024 classification, assigned before assay, with the laboratory blinded to group. Serum periostin was measured by enzyme-linked immunosorbent assay. Groups were compared using non-parametric tests with effect sizes; associations, adjusted analysis, and exploratory internally validated discrimination were examined.

# Results

Serum periostin rose stepwise across groups (medians 38.8, 53.2, 75.2, and 95.2 pg/mL; $p < 0.001$) with a very large effect size and significant differences between all adjacent groups. Periostin correlated strongly with an ordinal severity rank (rho = 0.95) and symptom control, and remained independently associated with severity after adjustment for age, sex, body-mass index, eosinophil count, and atopy. Routine blood counts, including eosinophil indices, did not differ between groups. Discrimination estimates were high but internally derived only.

# Conclusion

In this exploratory single-center pediatric sample, serum periostin showed a graded association with clinically classified asthma severity and outperformed routine eosinophil indices. The findings are preliminary; multicenter external validation is required before clinical use.

## Introduction

Asthma is the most common chronic respiratory disease of childhood and a major cause of morbidity, school absence, and impaired quality of life [31]. Accurate assessment of severity guides therapy, but in children it relies heavily on symptom history and on spirometry, which is effort-dependent, frequently unreliable in younger children, and often unavailable in primary- and secondary-care settings in low- and middle-income countries. Minimally invasive biomarkers reflecting the underlying disease biology could therefore strengthen severity assessment where objective lung-function testing is impractical.

Periostin is a matricellular extracellular-matrix protein induced in bronchial epithelium and fibroblasts by the type-2 cytokines interleukin-4 and interleukin-13 [1, 2]. It promotes eosinophil recruitment, subepithelial fibrosis, and airway remodeling, and in adults circulating periostin has emerged as a surrogate of type-2-high airway inflammation, with reported associations with severity, lung-function decline, and response to biologic therapy [3–5]. Pediatric evidence is comparatively limited and dominated by small studies using heterogeneous assays and units [6–10]. Data from Indian and South Asian children—who differ in genetic background, nutritional status, and environmental exposure—are particularly sparse: the few available Indian pediatric reports are small, single-center, and discordant, with periostin linked to poorer control in one cohort [11] but showing no association with severity or eosinophil indices in another [12].

Against this background we compared serum periostin across healthy controls and children with mild, moderate, and severe asthma, and, as a secondary and explicitly exploratory aim, examined how well periostin separated asthmatic from non-asthmatic children and severe from non-severe disease relative to routinely available eosinophil indices. The study was designed to generate preliminary South Asian pediatric evidence rather than to establish clinical cut-offs.

# Methods

*Study design and setting.* This single-center, cross-sectional, observational study was conducted in the department of pediatrics of a tertiary-care teaching hospital in southern India between August 2024 and February 2025. Reporting follows the STROBE statement for cross-sectional studies [30]; the exploratory diagnostic component is reported in keeping with STARD principles and is labeled accordingly.

*Participants.* Eighty children aged 6–18 years were enrolled by purposive sampling into four equal groups (n = 20 each): healthy controls and children with mild, moderate, or severe asthma. Asthmatic children were established cases who had been receiving inhaled-corticosteroid-containing therapy for at least 12 weeks; severity was therefore assigned at enrollment from the treatment intensity required to maintain control, not by prospective follow-up. Controls were children attending for non-respiratory complaints, with no asthma and no respiratory illness in the preceding four weeks. Children were excluded if they had conditions known to raise periostin independently of asthma (atopic dermatitis, recent wound or fracture healing). The deliberately balanced design was chosen for an exploratory comparison and is not representative of the source population (see Limitations).

*Severity classification and assessment.* Asthma was classified as mild, moderate, or severe by the treating pediatric team according to the Global Initiative for Asthma 2024 strategy [14], based on the treatment step required for control; classification was completed before periostin assay. A symptom-control score (higher scores indicating poorer control), anthropometry with age-referenced body-mass-index percentiles, and reported allergen exposure (clinical history of dust or pollen sensitivity) were recorded.

*Laboratory methods.* Five milliliters of venous blood were collected, allowed to clot, centrifuged, and the serum stored at -80 degrees C until assay. Serum periostin was measured by a quantitative double-antibody-sandwich enzyme-linked immunosorbent assay for human periostin (osteoblast-specific factor 2) (Shanghai Coon Koon Biotech, Cat. No. CK-bio-12826) per the manufacturer's instructions, with a standard-curve range of 1-120 pg/mL, sensitivity 1.0 pg/mL, and intra- and inter-assay coefficients of variation below 7% and 10%, respectively. Laboratory personnel were blinded to clinical group. A complete blood count with total leukocyte count, differential, absolute eosinophil count, and absolute neutrophil count was obtained.

*Statistical analysis.* Continuous variables were summarized as median (interquartile range) or mean +/- standard deviation according to distribution (Shapiro-Wilk test). Serum periostin was non-normally distributed across the pooled sample; groups were compared by the Kruskal-Wallis test with Dunn post-hoc comparisons (Holm correction) and the rank-based epsilon-squared effect size. Normally distributed blood counts were compared by one-way analysis of variance and categorical variables by the chi-square test. Associations used Spearman correlation with bootstrap 95% confidence intervals. The independent association of severity with periostin was assessed by multivariable linear regression (periostin as outcome; predictors severity step, age, sex, body-mass index, absolute eosinophil count, and atopy), which was the primary adjusted analysis; an ordinal model was unstable because periostin almost completely separated the strata. As a secondary, exploratory analysis, discrimination was assessed by receiver-operating-characteristic analysis, reporting the apparent area under the curve with bootstrap confidence intervals, an optimism-corrected estimate (.632 bootstrap and repeated five-fold cross-validation), and the Youden cut-off; no external validation was performed. Given the modest, artificially balanced sample, these estimates are regarded as hypothesis-generating and the study was not formally powered. A sensitivity analysis excluded controls reporting atopic exposure. Records were de-identified before analysis. Two-sided $p < 0.05$ was significant. Analyses used Python 3.12.

# Results

*Participant characteristics.* All 80 enrolled children were analyzed (Fig. 1). Groups were balanced for age (median 10-13.5 years; $p = 0.28$) and sex (47 boys, 33 girls; $p = 0.61$). Body-mass-index-for-age differed only borderline ($p = 0.06$). Reported atopic exposure was more frequent with greater severity ($p < 0.001$). Routine blood counts—total leukocyte count, neutrophil and eosinophil percentages, absolute eosinophil count, and absolute neutrophil count—did not differ across groups (all $p > 0.10$); the absolute eosinophil count showed a slight, non-significant decline toward severe asthma rather than the expected rise (Table 1).

Table 1
Baseline characteristics and peripheral blood counts by study group (n = 80)

| Variable | Control (n = 20) | Mild (n = 20) | Moderate (n = 20) | Severe (n = 20) | p |
|---|---|---|---|---|---|
| Age, y, median | 11.5 | 13.5 | 11.0 | 10.0 | 0.28 |
| Male, n (%) | 12 (60) | 14 (70) | 10 (50) | 11 (55) | 0.61 |
| BMI, kg/m2, median | 17.3 | 17.5 | 17.2 | 18.9 | 0.06 |
| Atopic exposure, n | 7 | 4 | 11 | 17 | < 0.001 |
| Symptom-control score, mean | 0.0 | 0.25 | 0.50 | 2.10 | < 0.001 |
| TLC, /mm3 | 8613 +/- 946 | 8676 +/- 986 | 8612 +/- 704 | 8503 +/- 523 | 0.93 |
| Eosinophils, % | 6.1 +/- 1.5 | 6.1 +/- 2.8 | 5.6 +/- 1.9 | 5.8 +/- 2.1 | 0.82 |
| AEC, /uL | 545 +/- 133 | 542 +/- 143 | 513 +/- 89 | 474 +/- 93 | 0.19 |
| ANC, /uL | 4786 +/- 585 | 4614 +/- 417 | 4836 +/- 695 | 4886 +/- 420 | 0.42 |

Continuous variables compared by Kruskal-Wallis test (medians) or one-way ANOVA (mean +/- SD); categorical by chi-square. AEC absolute eosinophil count; ANC absolute neutrophil count; BMI body-mass index; TLC total leukocyte count.

*Serum periostin across severity.* Serum periostin increased monotonically with severity: control 38.8 (interquartile range 36.8–42.5), mild 53.2 (47.4–56.4), moderate 75.2 (71.4–79.5), and severe 95.2 (90.8-101.5) pg/mL (Kruskal-Wallis H = 71.4, p < 0.001; epsilon-squared = 0.90). All adjacent pairwise contrasts were significant after Holm correction, and all non-adjacent contrasts had p < 0.001 (Table 2, Fig. 2).

Table 2
Serum periostin by asthma severity and its associations

| Parameter | Value | 95% CI |
|---|---|---|
| Serum periostin, median (IQR), pg/mL | | |
| Control | 38.8 (36.8–42.5) | – |
| Mild | 53.2 (47.4–56.4) | – |
| Moderate | 75.2 (71.4–79.5) | – |
| Severe | 95.2 (90.8-101.5) | – |
| Correlation with periostin (Spearman rho) | | |
| Ordinal severity rank | 0.95 | 0.92 to 0.96 |
| Symptom-control score | 0.70 | 0.56 to 0.80 |
| Absolute eosinophil count | -0.29 | -0.48 to -0.08 |
| Body-mass index | 0.25 | 0.03 to 0.43 |
| Kruskal-Wallis H = 71.4, p < 0.001, epsilon-squared = 0.90; all adjacent post-hoc comparisons p < 0.05 (Dunn-Holm). Multivariable linear regression: +18.0 pg/mL periostin per severity step (95% CI 16.5–19.6), model R2 = 0.92. CI confidence interval; IQR interquartile range. | | |

*Associations and adjusted analysis.* Periostin correlated very strongly with the ordinal severity rank (Spearman rho = 0.95, 95% confidence interval 0.92–0.96) and moderately-to-strongly with symptom control (rho = 0.70), showed a weak inverse correlation with absolute eosinophil count (rho = -0.29) and a weak positive correlation with body-mass index (rho = 0.25), and was unrelated to eosinophil percentage (Table 2). In multivariable linear regression, each one-step increase in severity was independently associated with an 18.0 pg/mL rise in periostin (95% confidence interval 16.5–19.6, p < 0.001); age, sex, body-mass index, and atopy were non-significant, and the model explained 92% of periostin variance.

*Exploratory discrimination.* For asthma versus control, the apparent area under the curve for periostin was 0.99, with an essentially identical optimism-corrected estimate; the internally derived Youden cut-off (approximately 51 pg/mL) gave 90% sensitivity and 100% specificity (Table 3, Fig. 3). For severe versus non-severe asthma the apparent area under the curve was 0.98. By contrast, the absolute eosinophil count discriminated at little better than chance (area under the curve approximately 0.60). These estimates are apparent and internally validated only and are not a substitute for external validation.

Table 3
Exploratory diagnostic accuracy of serum periostin (internally derived)

| Contrast | AUC (95% CI) | Cut-off, pg/mL | Sensitivity/Specificity |
|---|---|---|---|
| Asthma vs control | 0.99 (0.96-1.00) | approx. 51 | 90% / 100% |
| Severe vs non-severe (asthmatics) | 0.98 (0.93-1.00) | approx. 85 | 90% / 100% |
| Absolute eosinophil count (asthma vs control) | approx. 0.60 | – | poor |

Internally validated (.632 bootstrap and repeated five-fold cross-validation). Youden cut-offs derived in the same sample (apparent; external validation required). AUC area under the curve; CI confidence interval.

*Sensitivity analysis.* Excluding the seven controls who reported atopic exposure (n = 73) left the gradient and discrimination essentially unchanged, indicating the findings were not driven by atopic mislabeling of controls.

# Discussion

In Indian children aged 6–18 years, serum periostin rose in a clear, stepwise, and statistically robust manner from controls through mild, moderate, and severe asthma, correlated strongly with an ordinal severity rank and with symptom control, and remained independently associated with severity after adjustment for age, sex, body-mass index, eosinophil count, and atopy. Periostin tracked clinically classified severity far more closely than routine eosinophil indices did.

The direction and gradient agree with earlier pediatric reports of higher periostin with greater severity or poorer control [6–10, 13]. The two prior Indian pediatric studies are instructive and discordant: one linked periostin to poorer control [11], while another found no association with severity or eosinophil indices [12]. Our data, spanning four clinically defined strata in a single cohort, show a clear gradient and may help reconcile this mixed picture, though differences in assay and population must be weighed. Absolute concentrations differ widely across studies because of assay, calibrator, and unit heterogeneity: notably, our values (tens of pg/mL) are about three orders of magnitude lower than the ng/mL-scale concentrations reported with other kits [11, 12], which precludes any direct comparison of absolute values or cut-offs and underscores the need for assay standardization [15]. Our contribution is therefore not the discovery of periostin as a marker but preliminary South Asian pediatric data across the full severity spectrum, benchmarked head-to-head against routine eosinophil indices.

Biologically, interleukin-4 and interleukin-13 drive epithelial and fibroblast periostin, which promotes eosinophil adhesion and transforming-growth-factor-beta-dependent subepithelial collagen deposition [1, 2, 16, 17]. Periostin therefore plausibly indexes both type-2 inflammation and the remodeling that

characterizes more severe disease, which may explain why a structural-remodeling marker tracked severity better than circulating eosinophil counts.

The absolute eosinophil count neither differed across groups nor discriminated asthma from control and correlated weakly and inversely with periostin—a dissociation reported elsewhere [18]. The most plausible explanation is treatment: all asthmatic children had received inhaled corticosteroids for at least 12 weeks, and these suppress blood eosinophils more than periostin [19]; underpowering and single-timepoint sampling may also contribute. Because individual corticosteroid dose and adherence were not recorded, residual confounding cannot be excluded and is a key target for validation studies.

The near-perfect discrimination must be read with caution. Cut-offs were chosen and tested in the same sample, the groups were artificially balanced and well separated by design, and no external testing was performed; real-world performance will be lower. These values are hypothesis-generating signals of separability, not deployable thresholds, and we make no claim of diagnostic utility. If validated, a minimally invasive serum marker that tracks severity could complement symptom-based assessment in resource-variable pediatric settings where spirometry and fractional exhaled nitric oxide are unavailable and where the affordability of a new assay will itself require evaluation—a context directly relevant to Indian practice.

Strengths include an exclusively pediatric cohort with four clearly defined, equally sized groups and a healthy control group, transparent STROBE-based reporting, an independently verified analysis, and direct comparison of periostin with routine eosinophil indices in an under-represented population. The limitations are substantial and bound the interpretation. The study was single-center with a modest sample (n = 80) recruited by purposive, non-random sampling into artificially balanced groups, so prevalence-dependent measures, cut-offs, and the population distribution of periostin cannot be inferred, and the sample was not formally powered, including for the multivariable model, whose near-complete separation makes its coefficients unstable. The cross-sectional design precludes inference about within-child change, exacerbations, or causality. Spirometry, fractional exhaled nitric oxide, and total immunoglobulin E were unavailable, limiting comparison with established type-2 markers. Individual corticosteroid dose, adherence, disease duration, and sampling state were not captured, and allergic rhinitis—which can raise periostin—was not excluded. The receiver-operating-characteristic cut-offs were internally derived and not externally validated, so the reported accuracy is optimistic. Periostin is not asthma-specific and is influenced by age, growth, and puberty, and a single assay platform was used without inter-laboratory standardization.

In conclusion, in this single-center exploratory pediatric study serum periostin showed a graded association with clinically classified asthma severity and performed better than routine eosinophil indices in separating enriched severity groups. These findings support further multicenter, longitudinal, externally validated study in Indian and other resource-variable pediatric populations before periostin can be recommended for routine severity classification or diagnostic decision-making.

## Declarations

# Competing interests

The authors declare no competing interests.

### Ethics approval

The study was approved by the Institutional Ethics Committee, JSS Medical College, JSS Academy of Higher Education & Research, Mysuru (dated 23 June 2023), and performed in accordance with the Declaration of Helsinki.

### Consent to participate

Written informed consent was obtained from a parent or guardian, with age-appropriate assent from each child.

### Consent to publish

Not applicable; no individually identifiable data are included.

# Funding

No funding was received for conducting this study.

# Author Contribution

N.D. conceived and designed the study, supervised its conduct, and critically revised the manuscript for important intellectual content. A.R.M. recruited the participants, collected the clinical and laboratory data, and drafted the manuscript. S.N. contributed to data interpretation and critically revised the manuscript for important intellectual content. D.S.K. contributed to the study design, performed and interpreted the statistical analysis, and critically revised the manuscript; D.S.K. is the corresponding author. All authors read and approved the final manuscript and agree to be accountable for all aspects of the work.

# Data Availability

Available on request.

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# Figures

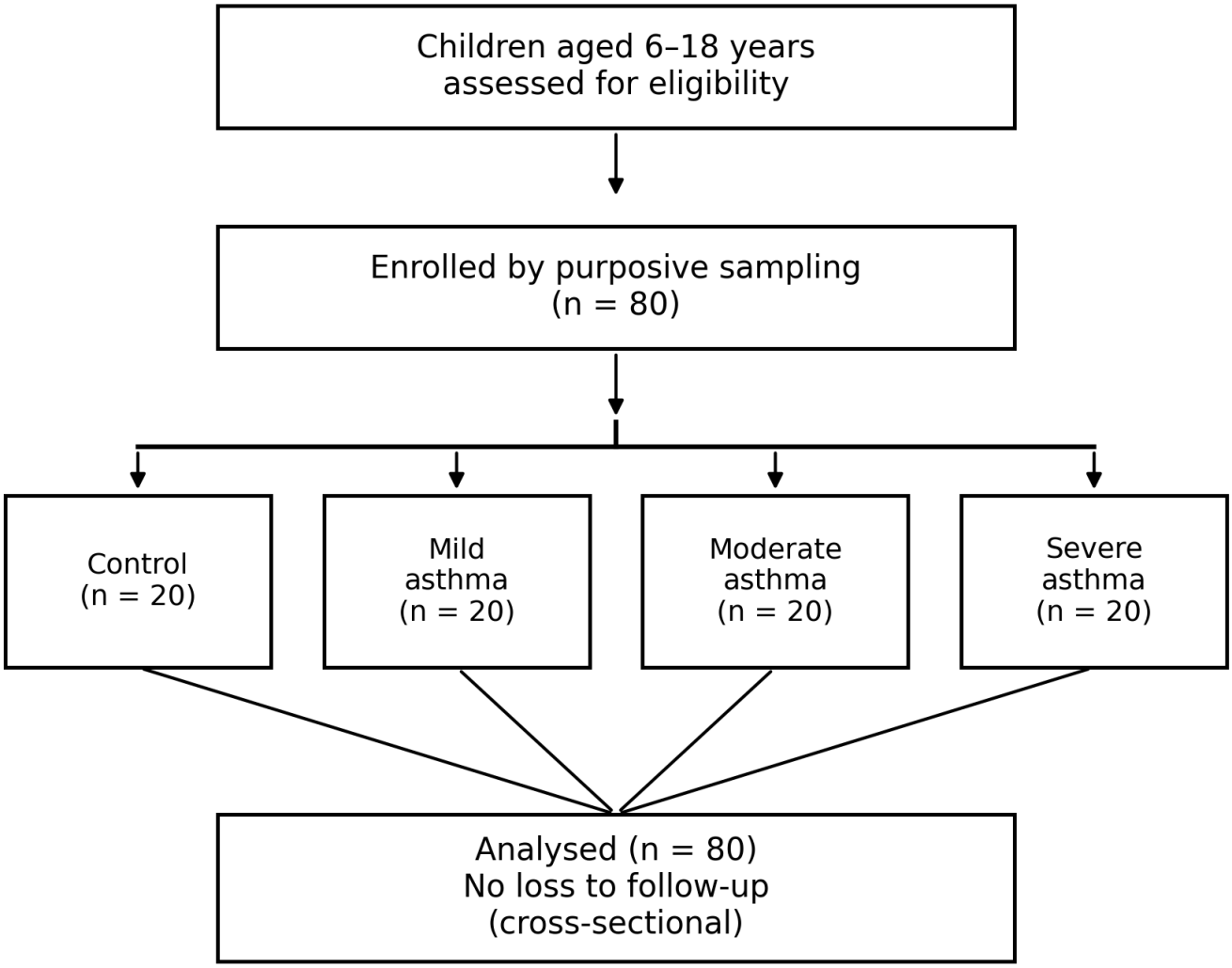



**Figure 1**

Participant flow. Children aged 6-18 years were assessed for eligibility and 80 were enrolled by purposive sampling into control, mild, moderate, and severe asthma (n = 20 each); all 80 were analyzed with no loss to follow-up

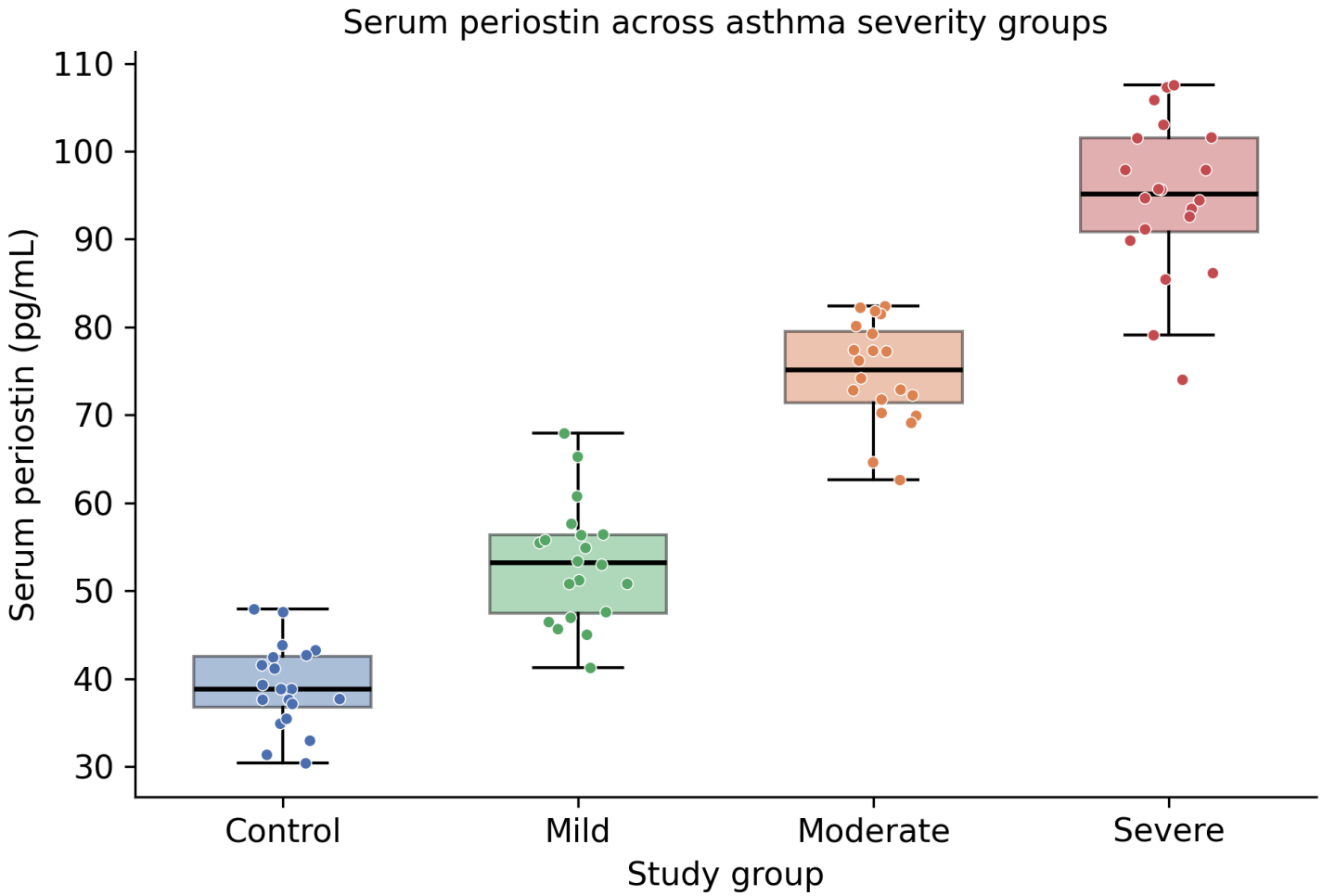



**Figure 2**

Serum periostin across asthma severity groups. Box-and-scatter plot of serum periostin (pg/mL) by group; boxes show median and interquartile range and points are individual children

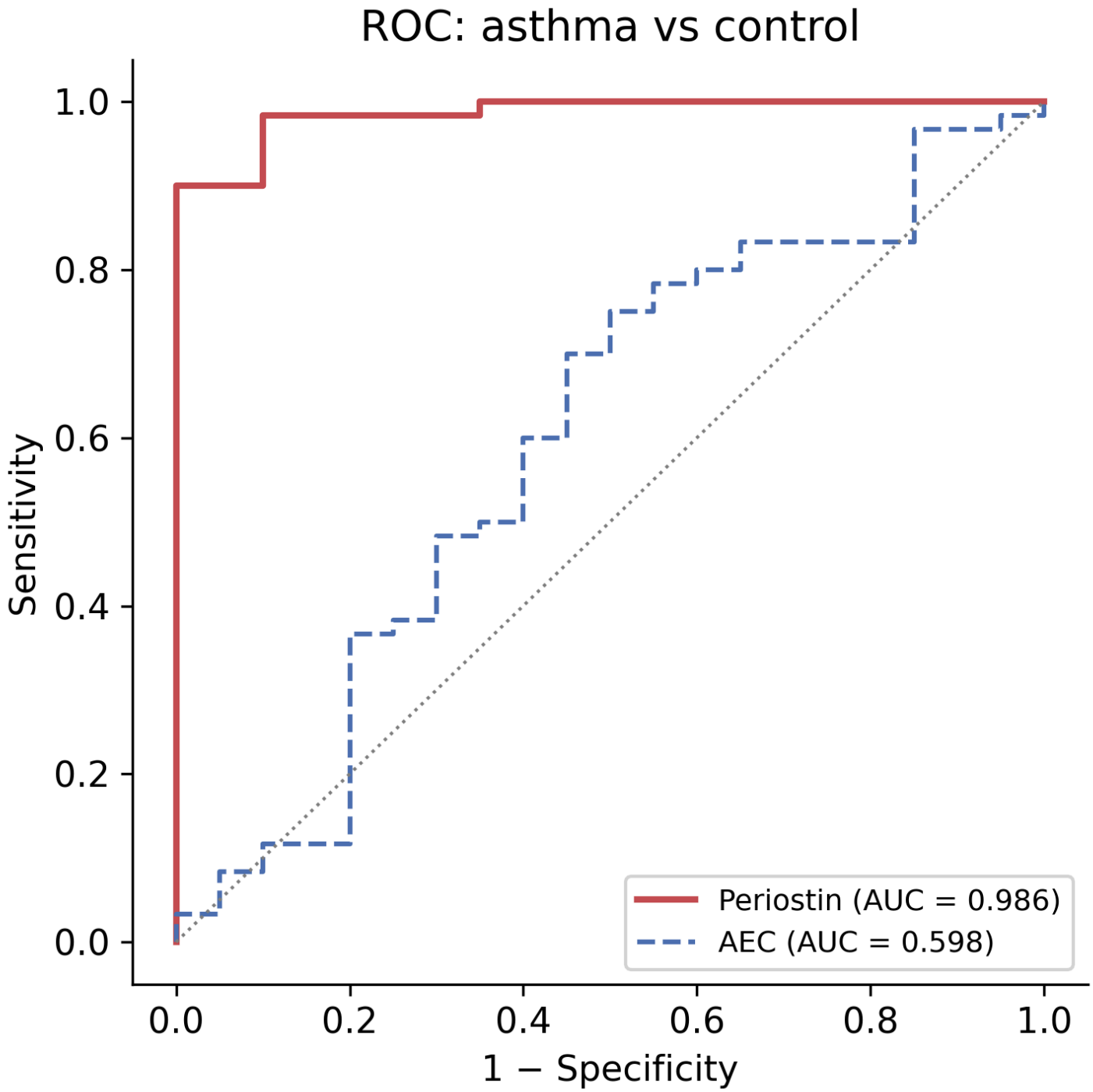



**Figure 3**

Receiver-operating-characteristic curves for asthma versus control: serum periostin versus absolute eosinophil count; the analysis is internal and exploratory

## Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Supplementarydataset.xlsx