

# Nasal polyposis in patients in the Severe Asthma Registry of The German Asthma Net

C. Bal, S. Stoshikj, K. Milger, D. Skowasch, M. Gappa, C. Koerner-Rettberg, M. Jandl, O. Schmidt, R. Ehmann, C. Taube, E. Hamelmann, R. Buhl, S. Korn<sup>1</sup>, M. Idzko<sup>1</sup>.

CB, SS and MI: Department of Pneumology, University Hospital Vienna AKH, Medical University of Vienna, Vienna, Austria, KM: Department of Medicine V, Ludwig-Maximilians-University (LMU) of Munich; Comprehensive Pneumology Center (CPC-M) German Center for Lung Research (DZL), Munich, Germany, DS: Department of Internal Medicine II - Pneumology, University Hospital Bonn, Bonn, Germany, MG: Evangelisches Krankenhaus Düsseldorf, Children's Hospital, Düsseldorf, Germany, CKR: Department of Pediatrics, Research Institute, Marien-Hospital Wesel, Wesel, Germany, MJ: Hamburger Institut für Therapieforschung GmbH, Hamburg, Germany, OS: Pneumologische Gemeinschaftspraxis und Studienzentrum KPPK, Koblenz, Germany, RE: Ambulante Pneumologie mit Allergiezentrum, Stuttgart, Germany, CT: Department of Pulmonary Medicine, University Hospital Essen - Ruhrlandklinik, Essen, Germany, EH: Kinderzentrum Bethel, Evangelisches Klinikum Bethel, University Bielefeld, Bielefeld, Germany, RB: Mainz University Hospital, Pulmonary Department, Mainz, Germany, SK: Thoraxklinik Heidelberg und IKF Pneumologie Mainz, Mainz, Germany.



German Asthma Net e.V.

Acknowledgements: The German Asthma Net is supported by scientific grants from AstraZeneca, Chiesi, GSK, Sanofi. None of the supporting parties had any participation in the data, nor did they contribute to the design or the content of the present poster.

## Background and methods

Severe asthma often co-occurs with Chronic Rhinosinusitis with nasal polyps (CRSwNP), characterized by increased type 2 inflammation. The concept of united airway disease arose due to the shared presence of type 2 cytokines, local IgE production, and eosinophil infiltration.

We aimed to characterize patients with severe asthma and comorbid CRSwNP in a real-life setting. We assessed epidemiologic, inflammation, disease control, and lung function parameters in patients from the German Asthma Net (GAN), an international, multi-center registry that records long-term comprehensive data.

## Cohort

**Results:** This cross-sectional study analyzed 1135 patients, 39% of who had CRSwNP, 58% were female, and in the mean 53±16 years, with 59% frequent exacerbations (≥2/year)).

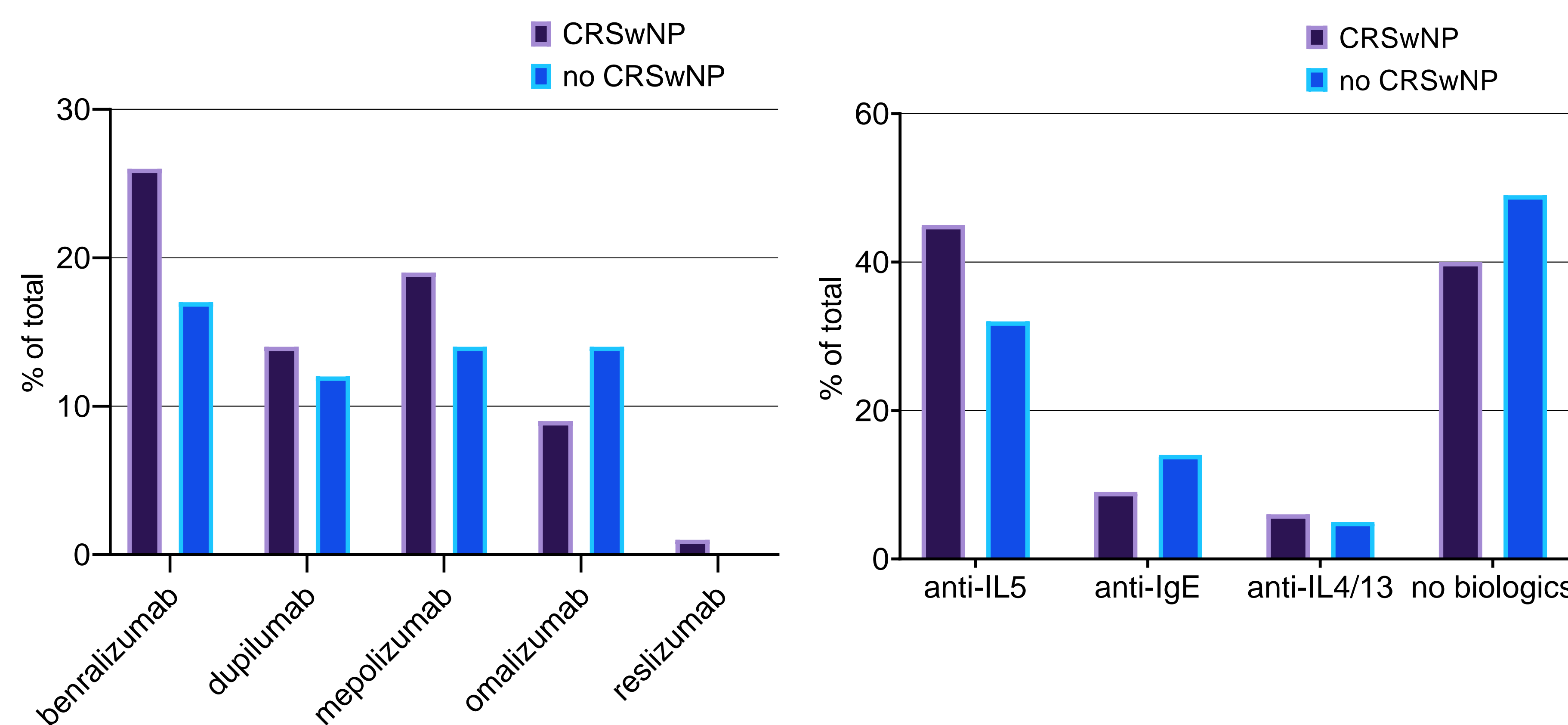
## Conclusion

In patients with severe asthma from the German Asthma Net Registry, the history of nasal polyps marks pronounced type 2 inflammation, suggesting possible benefit from targeted treatments.

There was no measured association with asthma control, exacerbation rates, and systemic corticosteroid treatments in this cross-sectional cohort with ongoing treatments.

## Biologics use

CRSwNP and severe asthma are independent indications for biologics, with increased use in this cohort. Corticosteroid use was irrespective of CRSwNP:



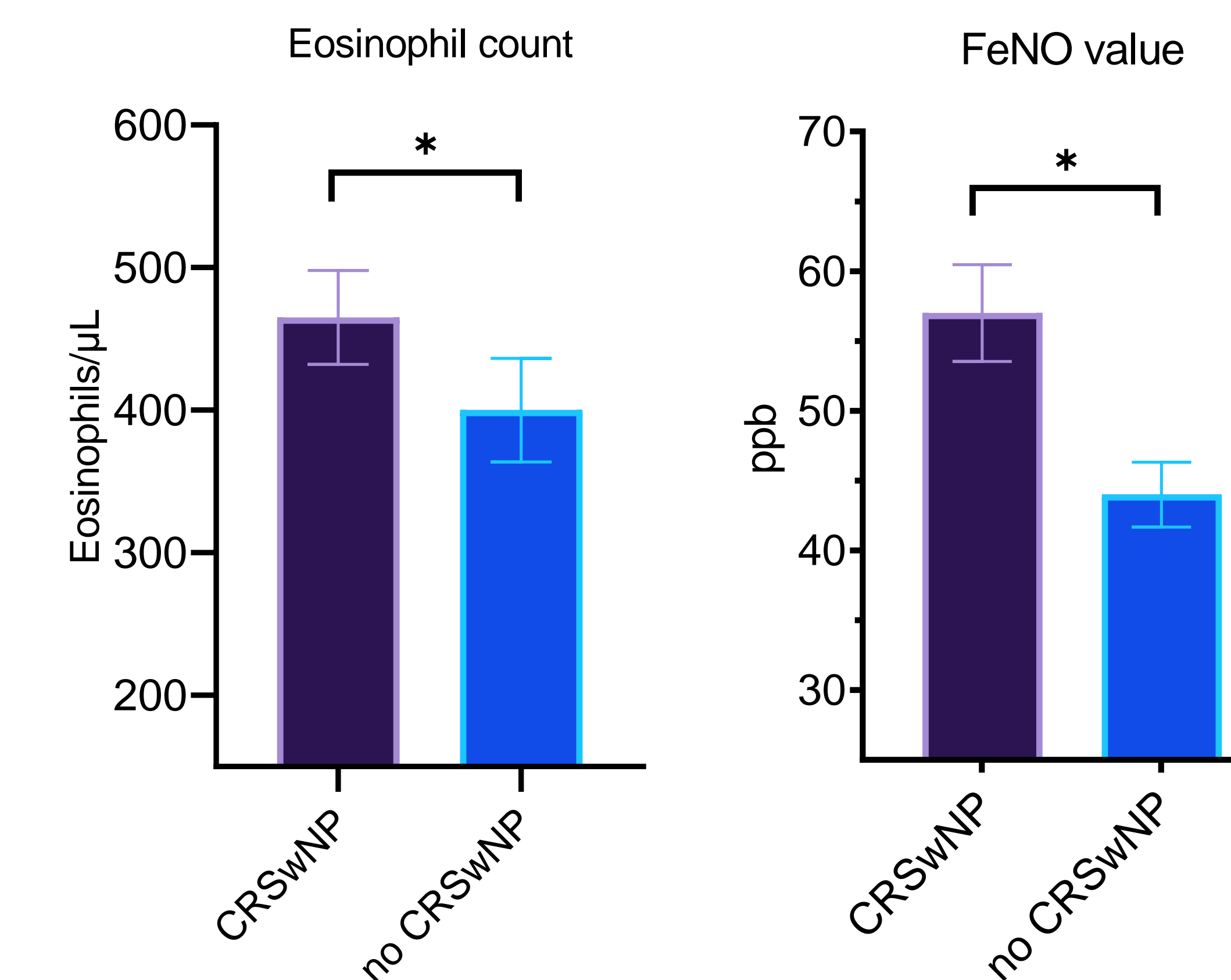
Variables, % of total (n):	CRSwNP, n=438	No CRSwNP, n=697	p-value
Biologic therapy	60% (262)	51% (353)	0.003
Anti-IL5R (benralizumab)	26% (110)	17% (117)	0.001
Anti-IL5 (mepolizumab)	19% (81)	14% (100)	0.06
Anti-IL5 (reslizumab)	1% (5)	0% (4)	0.3
Anti-IL4/13 (dupilumab)	14% (28)	12% (37)	0.5
Anti-IgE (omalizumab)	9% (38)	14% (95)	0.012
Corticosteroid maintenance therapy	34.0%	32.6%	0.6
Any corticosteroid adverse effects	58% (252)	51% (356)	0.03

## Comorbidities and age

Patients with and without CRSwNP differ in age and eosinophilic comorbidity prevalence:

Variables, % of total (n):	CRSwNP, n=438	No CRSwNP, n=697	p-value
Current age ≥ 18 years	98% (430)	95% (659)	0.003
Asthma onset ≥ 12 years	87% (381)	70% (488)	<0.0001
Surgical treatment of CRSwNP	76% (336)	n/a	n/a
Chronic sinusitis	80% (350)	30% (211)	<0.0001
NSAID-exacerbated disease	28% (121)	16% (110)	<0.0001
Acetylsalicylic acid deactivation	2% (10)	0% (0)	<0.0001
Eosinophilic granulomatosis with polyangiitis	6% (27)	2% (16)	0.001
Hypereosinophilic syndrome	5% (22)	3% (19)	0.04
Eosinophilic pneumonia	3% (11)	1% (6)	0.03

## Type 2 inflammation parameters



Variables, mean (SD) / % of total (n):	CRSwNP, n=438	No CRSwNP, n=697	p-value
FeNO, ppb	57 ± 52	44 ± 44	<0.0001
Blood eosinophils/μL	465 ± 556	400 ± 797	0.03
Predominantly allergic asthma form	42% (184)	49% (339)	0.008
Seasonal sensitisation (Prick test)	50% (219)	58% (403)	0.009
Seasonal sensitisation (Specific IgE)	16% (70)	24% (166)	0.002
Perennial sensitisation (Prick Test)	55% (239)	61% (427)	0.02
Perennial sensitisation (Specific IgE)	18% (78)	28% (192)	0.000

**Type 2 inflammation markers are significantly higher in CRSwNP patients:** they exhibited higher levels of exhaled nitric oxide (FeNO, p<0.001) and blood eosino-phil counts (p=0.03).

CRSwNP was also **more common in adults** than in children (p=0.003) and **inversely associated with allergic diatheses** (p<0.05).

## Asthma control and lung function

Mean disease control and lung function parameters were clinically similar:

Variables, mean (SD):	CRSwNP, n=438	No CRSwNP, n=697	p-value
Asthma onset, years	36 ± 17	32 ± 21	0.004
ACQ-5 score (MCID: 0.5 points)	2.6 ± 1.6	2.8 ± 1.4	0.06
ACT score (MCID: 3 points)	16 ± 6	14 ± 6	<0.001
mAQLQ score (MCID: 0.5 points)	4.1 ± 1.4	3.9 ± 1.3	0.04
FEV1 in % of predicted (MCID: 5-15%)	71 ± 21	66 ± 23	0.001
Worst FEV1 (% pred.), historical	61 ± 19	58 ± 21	0.007
FVC, % of predicted (MCID: 5-15%)	83 ± 20	79 ± 20	0.001
FEV1/FVC, in %	70 ± 15	67 ± 18	0.005