Decongestant Activity of Desloratadine in Controlled-Allergen-Exposure Trials

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Abstract

Nasal obstruction, which many patients consider to be the most bothersome symptom of seasonal allergic rhinitis (SAR), is generally refractory to oral antihistamine therapy. Effective resolution of nasal obstruction associated with SAR may help to prevent lower-airway disorders and other adverse sequela (e.g. otitis media with effusion). Desloratadine, a non-sedating antihistamine with marked inhibitory effects on the early- and late-phase allergic responses, affords significant relief of sneezing, pruritus and rhinorrhea, as well as nasal congestion. Using the Vienna Challenge Chamber, a closed system that enables rigorously controlled allergen exposure, we observed that a single 5mg dose of desloratadine rapidly and markedly reduced postexposure nasal obstruction in a pilot study. Separately, three randomised, double-blind, placebo-controlled trials demonstrated that desloratadine significantly reduced nasal blockage, as well as acute SAR symptoms, from baseline as compared with placebo over a 5-hour interval in the pollen chamber. The favourable effects of desloratadine on early-phase symptoms were consistent with evidence from controlled-allergen-exposure trials involving other antihistamines (cetirizine, fexofenadine). However, desloratadine also significantly protected against allergen-induced declines in nasal airflow (as assessed by active anterior rhinomanometry) and reduced nasal secretion weights compared with placebo in a controlled-allergen-exposure paradigm. The consistent decongestant effects of desloratadine in pollen-chamber trials were also concordant with data from clinical trials conducted under natural, ambient exposure conditions. Taken together, these findings support the clinical utility of desloratadine, a non-sedating, long-acting, high-affinity H1 receptor antagonist with decongestant properties.

Seasonal allergic rhinitis (SAR) affects up to one in six persons, and many patients consider nasal stuffiness to be the most bothersome symptom of this condition. Nasal blockage occurs in nearly 70% of patients with allergic rhinitis, not only in perennial allergic rhinitis but also in SAR. Nasal obstruction compromises nasal airflow velocity, potentially causing hypopnoea, apnoea and/or nonrestorative sleep. Some patients with nasal inflammation also develop acute sinusitis, otitis media, nasal polyps or orthodontic malocclusions.

A complex vascular, secretory and neural process that results from nasal mucosal inflammation, nasal obstruction is largely refractory to therapy with oral histamine H1 receptor antagonists. The resulting retention of secretions and tissue swelling may give way to more serious sequelae, partly by increasing the propensity for mouth breathing, which in turn facilitates the introduction of aller-
gens, cold air and/or pollutants to the lower airways.[6-8]

Desloratadine, a long-acting, non-sedating anti-histamine, has been shown to have therapeutic effects on nasal obstruction in 2-week SAR clinical trials. The significant first-dose relief of obstruction afforded by desloratadine in patients with SAR was recently demonstrated in trials conducted under conditions of controlled allergen exposure. Each of these trials was conducted in accordance with ethical principles established in the Declaration of Helsinki. The study protocol and informed-consent document were reviewed and approved by institutional review boards.

The aim of this report is 3-fold. First, we communicate previously unreported findings from a pilot, open-label trial[9] assessing the decongestant effects of desloratadine in patients with SAR who were exposed to allergen under controlled conditions. This study was presented at the XIXth Congress of the European Academy of Allergology and Clinical Immunology (EAACI) in Lisbon but has not been published previously in a peer-reviewed publication. Second, we report other previously unpublished findings from three randomised, double-blind, controlled-allergen-exposure trials conducted to assess the decongestant effects of desloratadine compared with placebo: two studies with parallel-group designs and one with a crossover design. Findings from these trials[10] were also presented, at the XXth Congress of EAACI, in Berlin. Third, we place the heretofore unpublished controlled-pollen-exposure data in the context of the clinical literature.

**Decongestant Activity of Desloratadine in Controlled-Allergen-Exposure Trials**

**Pilot Study**

**Methods**

In a Vienna Challenge Chamber (VCC), a closed, controlled environment, 28 patients (aged 20 to 40 years) with a minimum 2-year SAR history were exposed to grass pollen under reproducible climatic conditions outside the pollen season (December). Entry criteria included a positive case history of SAR, positive skin prick test and positive RAST using grass pollen allergens (*Dactylis glomerata*).

Patients were also required to be essentially asymptomatic prior to controlled allergen exposure. Recent treatment with glucocorticosteroids, other asthma medications, antihistamines and/or antibiotics for respiratory tract infections without adequate drug washout were grounds for exclusion. Also ineligible were individuals with chronic upper respiratory disorders.

Moderate to severe nasal symptoms within 2 hours after initial exposure to allergen in the VCC were also required. Design of the VCC was based on guidelines established by the Subcommittee on Bronchoprovocation for Occupational Asthma.[11] These guidelines specified that an exposure chamber should be a ventilated closed system with a documented concentration of a provocation substance.

The volume of the VCC was approximately 37.2 m³, and the chamber accommodated as many as 14 patients at once. Patients were observed through four windows during continuous allergen exposure, and supervising personnel were accessible to patients by use of an intercom system. Use of antistatic measures and frequent cleaning between challenge sessions decreased the possibility of allergen adhesion with irregular exposure concentrations.

Air in the chamber was cleaned and both the temperature and humidity maintained within pre-specified ranges of 24 to 26°C (75 to 79°F) and 30 to 35%, respectively. The pollen challenge was conducted at an airborne concentration of 1500 g/m³, a level that is consistent with everyday ambient allergen exposure during the hay fever season. Distribution of allergen within the VCC was held constant through the use of an automatic supply unit delivering a constant turbulent airflow and ongoing monitoring of airborne particle concentrations. All patients thus inhaled similar amounts of allergen during each challenge session.
During the continuous allergen exposure inside the VCC, participants recorded SAR symptoms electronically every 10 minutes, using a 4-point Likert scale where 0 = no symptoms, 1 = mild symptoms, 2 = moderate symptoms and 3 = severe symptoms. Nasal symptoms comprised anterior discharge, stuffiness, sneezing and nasal pruritus, whereas non-nasal symptoms were ocular pruritus, redness and tearing, as well as itching of the ears and palate. Minimum requirements for entry were a total symptom score (TSS) of 9; a combined nasal symptom score of 6, including a nasal discharge score of at least 2; and a non-nasal symptom score of at least 3. The mean entry TSS was 12.3.

Once their symptoms met the foregoing criteria, patients received a 5mg dose of desloratadine. Allergen challenge and monitoring of patients’ signs and symptoms continued for the next 3 hours. The non-sedating antihistamine was then administered once daily at the same dose in the morning of days 2 and 3, and a second allergen challenge (with computerised symptom assessment) was conducted on day 4, within 24 hours of the previous dose. Symptoms were assessed at baseline and during allergen exposure on days 1 and 4 (figure 1).

**Results**

All patients reported significant resolution of obstruction after desloratadine administration on day 1. Within 3 hours, all 28 patients showed declines from baseline. Among the patients who experienced at least a 2-point reduction from baseline TSS, nasal stuffiness scores were reduced by more than 20% at 20 to 30 minutes and by approximately 50% at 60 to 90 minutes (figure 2a).⁹

During allergen challenges conducted on day 4, 21 to 24 hours after the preceding desloratadine dose (*'trough' drug concentration), mean nasal stuffiness scores remained well below 2 (i.e. mild) for 120 minutes after exposure (figure 2b).⁹ Coupled with evidence that the mean area under the TSS versus time curve decreased by nearly 40% from day 1 to 4, this finding indicated that desloratadine’s decongestant effects were sustained over the 24-hour dosing interval. The chief limitation of this trial was its open-label design.

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Fig. 1. Design of the pilot allergen-chamber trial, an open-label trial in 28 patients.⁹ DL = desloratadine; od = once daily.

Fig. 2. Change in nasal obstruction after allergen challenge: (a) percentage changes in mean nasal stuffiness scores on day 1 among desloratadine 2-point respondents during controlled allergen exposure; (b) mean nasal stuffiness scores following allergen challenge on day 4 (21 to 24 hours after the preceding dose) among desloratadine 2-point responders.⁹
Therefore, the study was repeated as three double-blind, placebo-controlled trials.

Placebo-Controlled Trials

Three larger, single-dose, randomised, double-blind, placebo-controlled trials of pollen exposure were carried out under laboratory conditions. Two crossover trials were performed in a VCC out of the allergy season: study A (n = 52) evaluated the effects of a single oral dose of desloratadine (5mg), whereas study B (n = 53) assessed the effects of two single doses of desloratadine (5 or 7.5mg), compared with placebo. In the third trial (study C), 360 patients were randomly allocated to a single dose of desloratadine 5mg (n = 120), desloratadine 7.5mg (n = 120), or placebo (n = 120). Study C was conducted in the Environmental Exposure Unit (EEU; Kingston, Ontario, Canada) with allergen priming.[10]

Methods

Volunteers at least 19 years of age with a minimum 2-year history of SAR and positive immunoglobulin E-mediated skin test responses to allergen were eligible for inclusion in the three studies (studies A, B and C). Other entry criteria included a minimum baseline TSS value of 10; nasal symptom score above 6, including a nasal discharge score of at least 2; and a combined non-nasal symptom score of 4 or greater. Obstruction was moderate to severe at the baseline evaluation, with nasal congestion scores ranging from approximately 2.3 to 2.8 in the three studies.

In trials A and B, patients were exposed to allergen in the chamber for 2 hours before the baseline assessment, whereas those evaluated in study C were primed with allergen for up to six 3-hour intervals over 2 weeks before baseline. Using the 4-point Likert scale, where 0 = no symptoms, 1 = mild, 2 = moderate and 3 = severe, patients recorded the severity of nasal stuffiness and other symptoms every 15 minutes for 2 hours, then every 30 minutes for the remaining 3 hours (total, 5 hours). The observation period was 5 hours to permit assessment of the effects of desloratadine on the late-phase allergic response.

The effects of active treatment or placebo on other SAR symptoms following controlled allergen exposure in either the VCC or EEU included sneezing, nasal discharge, itchy eyes, tearing and itchy ears/palate. In the crossover studies (A and B), treatments and allergen exposure were separated by a washout period of at least 10 days.

Results

Administration of a single desloratadine dose (5mg) conferred significant relief of nasal blockage as compared with placebo at 5 hours in each study. Indeed, compared with placebo, desloratadine elicited an approximately 1.6- to nearly 4-fold greater decline in nasal stuffiness score at 5 hours (p < 0.05). Single-dose desloratadine reduced the nasal stuffiness score from baseline by approximately 20% to nearly 40% at hour 5 (p ≤ 0.02 vs placebo in each trial). Tabulated data for nasal congestion scores over each 15-minute interval in the VCC or EEU by treatment groups in studies A, B and C are presented in table I, table II and table III.[10] In the two trials that also evaluated a desloratadine dose of 7.5mg, no significant differences in decongestant effects were evident as compared with the 5mg dose.[10]

Compared with placebo, single-dose desloratadine (5mg) also significantly improved patient ratings of other symptoms, including nasal discharge, sneezing, itchy nose, itchy eyes, tearing and itchy ears/palate across studies A, B and C.

Discussion

Nasal symptoms such as sneezing and pruritus, as well as itchy, teary eyes, are induced primarily by release of mediators (e.g. histamine) in the early-phase response. According to the Allergic Rhinitis and Its Impact on Asthma (ARIA) report issued by the World Health Organization,[12] certain rhinitis symptoms tend to aggregate in two different patient subgroups, although these are not mutually exclusive; both symptom categories often coexist in a single patient. ‘Sneezers and runners’, who tend to have watery mucus and nasal itch, tend to experience particularly severe daytime symptoms, including conjunctivitis, nasal itching.
and often paroxysmal sneezing.\textsuperscript{12} Relief of these acute symptoms is a common benchmark for the clinical efficacy of conventional, second-generation antihistamines (e.g. loratadine, cetirizine, fexofenadine).

‘Blockers’, on the other hand, tend to have thick nasal mucus, little or no sneezing, itch or ophthalmic manifestations, and particularly severe evening symptoms,\textsuperscript{12} which can lead to sleep-disordered breathing with daytime fatigue.\textsuperscript{3,13} Because nasal obstruction is a potentially chronic manifestation of the late-phase response, resolution of SAR requires balanced therapeutic attack on effector pathways in both phases of the allergic cascade.

\begin{table}[h]
\centering
\begin{tabular}{|c|c|c|c|c|c|}
\hline
\textbf{Time (h:min)} & \textbf{Desloratadine 5mg (n = 52) [A]} & \textbf{Placebo (n = 52) [B]} & \textbf{p value (A vs B)} \\
& \textbf{mean} & \textbf{SE} & \textbf{mean change (\%)} & \textbf{mean} & \textbf{SE} & \textbf{mean change (\%)} & \\
\hline
Baseline\textsuperscript{a} & 2.38 & 0.09 & 2.33 & 0.10 & 0.434 \\
0:15 & -0.06 & 0.07 & -1.0 & 0.02 & 0.06 & 3.6 & 0.421 \\
0:30 & -0.15 & 0.08 & -5.1 & -0.13 & 0.08 & -2.0 & 0.855 \\
0:45 & -0.33 & 0.08 & -12.5 & -0.19 & 0.08 & -6.2 & 0.278 \\
1:00 & -0.37 & 0.10 & -12.2 & -0.27 & 0.09 & -8.5 & 0.483 \\
1:15 & -0.44 & 0.10 & -15.7 & -0.27 & 0.10 & -8.2 & 0.184 \\
1:30 & -0.36 & 0.12 & -9.3 & -0.21 & 0.10 & -5.6 & 0.413 \\
1:45 & -0.58 & 0.12 & -20.5 & -0.21 & 0.10 & -6.5 & 0.026 \\
2:00 & -0.62 & 0.12 & -22.8 & -0.29 & 0.11 & -8.5 & 0.046 \\
2:30 & -0.63 & 0.12 & -23.7 & -0.29 & 0.10 & -11.1 & 0.019 \\
3:00 & -0.69 & 0.12 & -27.2 & -0.29 & 0.10 & -11.1 & <0.01 \\
3:30 & -0.62 & 0.11 & -23.4 & -0.33 & 0.11 & -13.4 & 0.048 \\
4:00 & -0.54 & 0.12 & -18.6 & -0.23 & 0.11 & -9.2 & 0.063 \\
4:30 & -0.58 & 0.11 & -21.8 & -0.29 & 0.11 & -9.5 & 0.056 \\
5:00 & -0.62 & 0.13 & -20.5 & -0.19 & 0.12 & -5.2 & 0.011 \\
\hline
\end{tabular}
\caption{Nasal stuffiness/congestion score by treatment group (intent-to-treat): study A}
\end{table}

\begin{table}[h]
\centering
\begin{tabular}{|c|c|c|c|c|c|c|c|}
\hline
\textbf{Time (h:min)} & \textbf{Desloratadine 5mg (n = 53) [A]} & \textbf{Desloratadine 7.5mg (n = 53) [B]} & \textbf{Placebo (n = 53) [C]} & \textbf{p value (A vs C)} & \textbf{p value (B vs C)} \\
& \textbf{mean} & \textbf{SE} & \textbf{mean change (\%)} & \textbf{mean} & \textbf{SE} & \textbf{mean change (\%)} & \\
\hline
Baseline\textsuperscript{a} & 2.26 & 0.10 & 2.26 & 0.09 & 2.36 & 0.10 & 0.269 & 0.251 \\
0:15 & -0.04 & 0.04 & -1.6 & -0.04 & 0.05 & 0.0 & -0.08 & 0.05 & -1.6 & 0.608 & 0.615 \\
0:30 & -0.17 & 0.08 & -3.5 & -0.17 & 0.08 & -5.1 & -0.23 & 0.07 & -7.7 & 0.616 & 0.610 \\
0:45 & -0.30 & 0.08 & -10.3 & -0.26 & 0.10 & -7.1 & -0.36 & 0.09 & -11.5 & 0.592 & 0.430 \\
1:00 & -0.42 & 0.07 & -16.7 & -0.34 & 0.10 & -11.9 & -0.55 & 0.08 & -23.4 & 0.201 & 0.067 \\
1:15 & -0.53 & 0.09 & -20.5 & -0.38 & 0.10 & -14.1 & -0.57 & 0.09 & -22.4 & 0.713 & 0.136 \\
1:30 & -0.70 & 0.09 & -28.5 & -0.49 & 0.10 & -20.2 & -0.51 & 0.09 & -20.5 & 0.128 & 0.900 \\
1:45 & -0.82 & 0.09 & -25.6 & -0.57 & 0.10 & -23.4 & -0.57 & 0.10 & -22.4 & 0.631 & 0.978 \\
2:00 & -0.88 & 0.10 & -27.9 & -0.51 & 0.10 & -19.6 & -0.62 & 0.12 & -23.7 & 0.704 & 0.430 \\
2:30 & -0.70 & 0.11 & -29.5 & -0.55 & 0.11 & -21.8 & -0.47 & 0.11 & -17.8 & 0.130 & 0.594 \\
3:00 & -0.75 & 0.11 & -31.7 & -0.62 & 0.11 & -25.3 & -0.60 & 0.11 & -23.4 & 0.362 & 0.910 \\
3:30 & -0.77 & 0.11 & -33.0 & -0.75 & 0.11 & -31.7 & -0.55 & 0.12 & -20.8 & 0.137 & 0.155 \\
4:00 & -0.75 & 0.11 & -31.7 & -0.64 & 0.11 & -27.2 & -0.42 & 0.11 & -14.1 & 0.022 & 0.112 \\
4:30 & -0.72 & 0.10 & -29.8 & -0.60 & 0.11 & -26.0 & -0.40 & 0.11 & -13.8 & 0.025 & 0.133 \\
5:00 & -0.70 & 0.11 & -29.2 & -0.60 & 0.11 & -26.3 & -0.36 & 0.10 & -12.2 & 0.017 & 0.076 \\
\hline
\end{tabular}
\caption{Nasal stuffiness/congestion score by treatment group (intent-to-treat): study B}
\end{table}
Table III. Nasal stuffiness/congestion score by treatment group (intent-to-treat): study C

<table>
<thead>
<tr>
<th>Time (h:min)</th>
<th>Desloratadine 5mg (n = 120) [A]</th>
<th>Desloratadine 7.5mg (n = 120) [B]</th>
<th>Placebo (n = 120) [C]</th>
<th>p value (A vs C)</th>
<th>p value (B vs C)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean SE</td>
<td>mean change (%)</td>
<td>mean SE</td>
<td>mean change (%)</td>
<td>mean SE</td>
</tr>
<tr>
<td>Baseline†</td>
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<td>-0.32 0.06</td>
<td>-0.51 0.07</td>
<td>2.83 0.04</td>
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<tr>
<td>0:15</td>
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<td>-2.60</td>
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<td>-13.30</td>
<td>-0.05 0.06</td>
</tr>
<tr>
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<td>-19.40</td>
<td>-20.70</td>
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<td>-23.30</td>
</tr>
<tr>
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<td>-31.70</td>
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<td>-25.70</td>
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<td>-37.10</td>
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</tr>
<tr>
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<td>-23.90</td>
</tr>
</tbody>
</table>

† Baseline mean is based on the actual scores, whereas all other means are based on the changes from baseline. SE = standard error.

Desloratadine exerts dual inhibitory effects on both the early-phase and late-phase response. Such balanced therapeutic attack is consistent with clinical benefits for both subpopulations of rhinitis sufferers.

In the placebo-controlled pollen-chamber studies,[10] desloratadine significantly alleviated rhinorrhea, sneezing and itchy nose, as well as a number of non-nasal symptoms (i.e. itchy eyes, ears and palate, tearing), with all of these being of comparable clinical relevance.

Taken individually, these data were in line with expectations for antihistamines. However, the consistency of improvement across all symptoms, including nasal congestion, is different from certain experiences with existing antihistamines. For example, a single-dose 5-hour EEU study of similar design conducted by Day and colleagues[14] showed that, compared with placebo, fexofenadine (60 to 120mg) significantly relieved sneezing (p = 0.0094), itchy nose, palate and/or throat (p = 0.0004), and itchy, watery, red eyes (p = 0.0083), but not rhinorrhea (p = 0.0607) or nasal congestion (p = 0.3683). In another study,[15] conducted in the VCC, a single dose of cetirizine 10mg or fexofenadine 120mg significantly reduced all symptoms of SAR, but no significant differences were observed in nasal flow as measured by active anterior rhinomanometry.

In addition, Day’s group[16] reported that cetirizine and placebo reduced patient ratings of a composite total symptom complex score by 36.7% and 12.0%, respectively (p < 0.01). However, the effects of cetirizine on nasal congestion alone were not reported as a distinct variable.

The pollen-chamber data in support of the decongestant efficacy of desloratadine were also concordant with changes in patient ratings of nasal congestion under conditions of ambient allergen exposure. The significant effect of desloratadine in reducing obstruction relative to placebo has been observed in randomised, double-blind, placebo-controlled trials involving patients with SAR, either alone[17] or in concert with bronchial asthma.[18]

In these studies, which were presented at the 57th annual meeting of the American Academy of Allergy Asthma & Immunology,[17,18] deslorata-
dine 5mg conferred significant first-dose relief of nasal congestion, and this significant relief of obstruction (versus placebo) endured for up to 4 weeks with once-daily administration (5 mg/day).

Because the effects of previously available antihistamines on nasal obstruction were typically marginal, SAR patients often required adjunctive decongestants such as pseudoephedrine.\textsuperscript{[19-21]} In an abstract presented at the XXth Congress of EAACI, Lorber et al.\textsuperscript{[22]} reported that the decongestant effect of once-daily desloratadine (5 mg/day) for 15 days under ambient-allergen-exposure conditions was of similar magnitude to that observed with pseudoephedrine (120mg twice daily). Suboptimal tolerability, with insomnia and nervousness, limits the clinical value of pseudoephedrine monotherapy.\textsuperscript{[1,22]}

The open-label and placebo-controlled trials reported herein\textsuperscript{[9,10]} were also consistent with data from a separate controlled-allergen-exposure crossover study that assessed the effects of desloratadine 5mg and placebo on nasal airflow (active anterior rhinomanometry) and nasal secretion weights in 47 patients with histories of SAR; this study report has been published elsewhere.\textsuperscript{[23]} Within 30 minutes of allergen exposure, significantly less pronounced decreases in nasal airflow (p < 0.02), as well as less nasal secretion (p < 0.001) and less severe symptoms (e.g. nasal congestion, rhinorrhea) were observed with desloratadine compared with placebo.

The potential clinical implications of these findings are noteworthy. First, unmitigated nasal obstruction promotes mouth breathing, which can in turn lead to untoward sequelae through the introduction of allergens, pollutants or cold air to the lower airways.\textsuperscript{[6-8]} The conditioning effects of the nose, which serves as the first line of defence for the lungs, include warming and filtering. The tortuous, mucus-lined nasal passages trap particles larger than 10μm under normal circumstances.

Bronchial asthma is one of a number of potentially serious conditions that can be induced by, or coexist with, SAR; poorly controlled SAR can also contribute to the development of sinusitis, nasal polyp recurrence, otitis media, otitis media with effusion, hearing impairment and orthodontic malocclusion.\textsuperscript{[4,5,8,24]}

Griffin et al.\textsuperscript{[6]} reported that the severity of bronchoconstriction varied linearly (r = 0.81) with the degree of cooling in the retrotracheal oesophagus among asthmatic patients who were asymptomatic before inhaling air at subfreezing temperatures. Nasal respiration reduced the retrotracheal oesophageal temperature by 0.4°C, as against a decline of 2.7°C observed in patients using oral respiration (p < 0.0001).\textsuperscript{[6]} Further, Henriksen and Wenzel\textsuperscript{[25]} reported that effective relief of nasal obstruction correlated closely (r = 0.80; p < 0.001) with declines in mouth breathing among youngsters with asthma.

Mouth breathing can also cause dry, chapped lips, dryness of the mouth and throat, and, when chronic, more serious effects.\textsuperscript{[26]} In addition, breathing that bypasses the nose, such as after tracheostomy, is known to disturb the lower airways,\textsuperscript{[27]} lending further credence to the role of oral respiration in the potential exacerbation of asthma.

Nasal blockage, which tends to be more severe during the evening hours, nearly doubles the risk of sleep-disordered breathing.\textsuperscript{[3]} Sleep disturbances caused by nasal stuffiness, which is inherently more severe in the evening for many patients, are also associated with daytime somnolence; patients may erroneously attribute daytime somnolence to their antiallergy medications.\textsuperscript{[11]} Finally, nasal mucosal swelling can also potentially diminish access of topical medications to absorptive surfaces in the nasal mucosa.\textsuperscript{[28]}

\textbf{Conclusion}

When exposed to grass pollen under controlled-exposure conditions for 5 hours, SAR patients who received a single dose of desloratadine (5mg) experienced significant relief of nasal obstruction and significant improvement in nasal flow. These results were concordant with enhanced ratings of nasal stuffiness by patients with SAR and/or SAR-asthma who received desloratadine under condi-
tions of normal ambient exposure. On the basis of these findings, desloratadine, a long-acting agent that exhibits high binding affinity for the H1 receptor and exerts salutary effects on the late-phase allergenic response, represents a sound therapeutic alternative, conferring both consistent decongestant effects and nonsedating relief of early-phase symptoms (e.g. sneezing, rhinorrhea, pruritus) in SAR.

Acknowledgements

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