Nasal obstruction as a risk factor for sleep-disordered breathing

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Nasal obstruction frequently has been associated with sleep-disordered breathing as a potential etiologic factor. Nasal obstruction results in pathologic changes in airflow velocity and resistance. Experimentally produced nasal obstruction increases resistance and leads to sleep-disordered breathing events, including apnea, hypopnea, and snoring. Clinical research examining the correlation between nasal obstruction and sleep-disordered breathing is limited, especially in regard to patients with conditions that increase nasal resistance, such as rhinitis and sinusitis. To further identify risk factors for sleep-disordered breathing, the role of chronic and acute nasal congestion was investigated in a population-based sample. Data on nasal congestion history and sleep problems were obtained by questionnaire (n = 4927) *and by objective in-laboratory measurement* (n = 911). *Participants who often or almost always experienced nighttime symptoms of rhinitis (5 or more nights a month) were significantly* ($p < 0.0001$) *more likely to report habitual snoring (3 to 7 nights a week), chronic excessive daytime sleepiness, or chronic nonrestorative sleep than were those who rarely or never had symptoms. Habitual snorers had significantly* ($p < 0.02$) *lower air flow than nonsnorers, although a linear relation between decreased airflow and sleep-disordered breathing severity did not exist. Participants who reported nasal congestion due to allergy were 1.8 times more likely to have moderate to severe sleep-disordered breathing than were those without nasal congestion due to allergy. Men and women with nasal obstruction, especially chronic nighttime symptoms of rhinitis, are significantly more likely to be habitual snorers, and a proportion also may have frequent episodes of apnea and hypopnea, indicative of severe sleep-disordered breathing. Because allergic rhinitis is a common cause of nasal obstruction and it is a modifiable risk factor, further study of this association is warranted. (J Allergy Clin Immunol 1997;99:S757-62.)*

***Key words:** Sleep-disordered breathing, sleep apnea, nasal congestion, rhinitis*

Sleep-disordered breathing has been recognized as a serious disorder for a century; however, it is only within the past few decades that advances have been made in identifying risk factors, understanding the pathophysiologic process and adverse health sequelae, and in improving diagnosis and treatment. The condition comprises a range of abnormal breathing events that include frequent episodes of apnea, hypopnea, and snores or breaths with high airway resistance.[1]

Sleep-disordered breathing once was thought to be a disease of morbidly obese, middle-aged men. Epidemiologic studies have now established that sleep-disordered breathing occurs among women and those who are not extremely overweight.[2] Population studies have begun to identify other risk factors and to quantify the strengths of their associations with sleep-disordered breathing. Understanding risk factors for a prevalent disorder is important for two reasons. First, risk factors can be indicative of causal factors. Elimination or reduction of risk factors with a high suspicion for causality is an important preventive strategy. Second, knowledge of risk factors assists in efficient screening by targeting persons most likely to have a particular disorder. For chronic conditions that often go unrecognized, such as sleep-disordered breathing, this strategy may facilitate care of patients whose condition would otherwise go undiagnosed and untreated.

Overweight and central ponderosity, aging, male sex, and severe (but relatively rare) craniofacial abnormalities such as acromegaly have been identified as strong risk factors for sleep-disordered breathing. Studies now are focused on understanding causal mechanisms for these factors. Hypothesized but untested risk factors include problems originating in the nose, including physical obstruction, allergic rhinitis, and chronic sinusitis. Other risk factors under investigation include familial predisposition, smoking, alcohol use, menopause, and ethnicity. The risk factors of particular importance in

Abbreviations used

AHI:	Apnea-hypopnea index
BMI:	Body mass index

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terms of prevention strategies are those that are prevalent and modifiable.

Nasal obstruction has been mentioned frequently in reviews and case reports as a potential etiologic factor for sleep apnea.[3-5] A biologic basis for nasal obstruction as a cause of sleep-disordered breathing lies in the effect of nasal breathing on resistance and flow velocity, which affects the pressure differential between the atmosphere and the intrathoracic space. Partial or complete obstruction can occur when the intrathoracic negative pressure generated by the inspiratory muscles pulls on the compliant soft tissue in the upper airway, sucking the airway closed. Moreover, the nose accounts for half of total respiratory system resistance.[6] In this regard, the nose has been described as a variable resistor with a collapsible segment, such that flow limitation in the nasopharynx results in conditions favorable to downstream pharyngeal collapse.[7]

The importance of effectual nasal breathing in maintaining the automatic respiratory rhythms in sleep has long been recognized. Airflow through the nose, however, is a formidable task, given the physics of airflow through the complex nasal structure of compliant nares, a mucosal lining that is greatly affected by vasodilation and constriction, secretions of variable viscosity, and sinus pockets where inspired air is detained by turbulence for warming and humidifying. Several conditions affect nasal resistance, including temperature and humidity of the air supply, posture, nasal vasoconstriction, and mucosal changes.[5] The nasal mucosa, responsive to both internal and external stimuli, appears to play a large role in modulating resistance. Experimentally produced local nasal vasoconstriction produces a decrease in resistance in sleeping snorers.[8] White et al. showed that repeated episodes of apnea and hypopnea during sleep result from chronic stimulation of the mucosa with an irritant.[9] An interaction between sleep-disordered breathing and upper airway mucosa also may occur whereby snoring vibrations injure the mucosa, contributing to increased resistance and snoring. In summary, nasal breathing problems, including obstruction and irritation, are plausible risk factors for sleep-disordered breathing.

A number of experimental studies have evaluated changes in nasal obstruction with the use of nasal packing. Zwillich et al. compared apnea during sleep (measured by means of polysomnography) experienced by adults with and without artificial nasal obstruction (by means of balloon canula). The investigators found that obstruction was associated with a statistically significant increase in number of episodes of apnea and cortical arousal.[10] Several other studies of artificial obstruction also showed a positive association between obstruction and sleep-disordered breathing events, including snoring, apnea, and hypopnea. A 1996 study involving children and young adults added experimental support to a role of nasal obstruction in sleep-disordered breathing. Millman et al. found that although there was individual variability in response, on average nasal occlusion induced by packing led to an increase in the total number of episodes of apnea or hypopnea during 1 hour of sleep (AHI), apnea duration, and sleep fragmentation.[11] The investigators concluded that nasal obstruction has a clinically significant impact on breathing during sleep and speculated that nasal obstruction over several days could, by means of increased sleep-disordered breathing, affect daytime performance by children and young adults if there was no habituation.

The physiologic tasks of the nose and experimental alteration of nasal resistance support the hypothesis that chronic conditions that increase nasal resistance, including permanent physical obstruction and the congestion and irritation of rhinitis and sinusitis, contribute to sleep-disordered breathing. In spite of a strong rationale for the hypothesis, few clinical or epidemiologic studies have investigated the association. Such studies are important to determine whether the specific mechanisms identified during basic experimental studies are not overcome with habituation and actually translate to measurable health outcomes that persist in daily life.

Several studies have been conducted among patients without allergies with sleep-disordered breathing. Blakely and Mahowald[12] used rhinometry to measure awake nasal resistance for 53 patients with sleep apnea and 37 healthy persons. The investigators hypothesized that mean total nasal resistance for patients with apnea would be greater than for those without apnea and that apnea severity would be associated with increased nasal resistance. The results indicated that although patients with sleep apnea had elevated nasal resistance, a linear relation between resistance and apnea severity measured by means of oxygen desaturation did not exist. Other studies also have shown that nasal resistance during waking hours is only weakly related to obstructed breathing during sleep. Metes et al. analyzed records of 370 patients at a sleep clinic who snored and had been examined by means of polysomnography and rhinometry.[13] Nasal resistance was not related to severity of sleep-disordered breathing, indicated by the frequency of episodes of apnea and hypopnea, but nasal resistance was a significant predictor of snoring frequency. Miljeteig et al. measured awake nasal resistance and performed overnight polysomnography on 683 patients.[14] They found no statistically significant correlation between nasal resistance and either snoring or sleep apnea. Miljeteig et al. furthered this work by studying breath-by-breath nasal resistance during sleep for eight patients. The investigators used a sealed mask system with a pneumotachograph and a nasopharyngeal catheter with pressure transducers.[15] The resistance measures were recorded simultaneously with polysomnography. Analysis of the data indicated that nasal resistance fluctuated markedly throughout the night, but there was no correlation between resistance and snoring. Limitations in the study method, including elimination of any oral snoring by means of taping the mouth shut, did not appear to explain the negative findings, and the authors concluded that nasal resistance and snoring severity were not correlated.

TABLE I. Survey sample* characteristics (n = 4927)

Characteristic	Finding
Mean age (yr)	45 (7.8)
Mean body mass index (kg/m^2)	29 (6.4)
Male (percentage of participants)	59
Snoring status (percentage of participants)	
Occasional (1–3 nights per week)	31
Habitual (>3 nights per week)	26
Allergies as cause of nasal congestion (percentage of participants)	
Medicate	13
Do not medicate	22
Total	35
Rhinitis symptoms (percentage of participants)	
Never	30.8
Rarely	31.4
Sometimes	20.3
Often	11.7
Always or almost always	5.7
Chronic excessive daytime sleepiness (percentage of participants)	15
Chronic nonrestorative sleep (percentage of participants)	24

Values in parentheses are standard deviations.
*Includes cohort participants (n = 911).

TABLE II. Cohort sample characteristics (n = 911)

Characteristic	Finding
Self-reported nasal congestion on the day or night of study (percentage of participants)	
Due to illness	15
Due to allergy	12
Other cause	1
Total	28
Regular seasonal nasal congestion (percentage of participants)	
Due to allergy	23
Due to illness	10
Other cause	1
Total	34
Mean nasal air flow before sleep (mL/sec at −1.5 cm H_2O)	
Women	
Right nostril	236 (128)
Left nostril	240 (131)
Both nostrils	476 (216)
Men	
Right nostril	283 (156)
Left nostril	275 (151)
Both nostrils	562 (257)

Values in parentheses are standard deviations.

In one study investigators compared sleep-disordered breathing among patients with seasonal allergic rhinitis when the rhinitis was symptomatic and when it was not.[16] The results indicated higher AHI and longer apnea duration when patients had symptoms. Although the findings were statistically significant, they were based on group averages for apnea frequency and duration; intrasubject comparisons showed minimal differences. Nasal obstruction and sleep-disordered breathing also were explored in a population-based study.[17] As part of a home study of nighttime oximetry for 1001 men enrolled in an Oxford, England, medical practice, Stradling and Crosby asked subjects about whether they experienced nasal stuffiness. History of nasal congestion was not related to the frequency of desaturation events during sleep, but congestion was associated with habitual snoring. Twenty-eight percent of the men with nasal congestion and 14% of the men without nasal congestion reported that they were habitual snorers.[17]

Previous clinical studies have provided little support for a role of nasal obstruction in increasing severity of sleep-disordered breathing. However, because of methodologic limitations of these early studies, the hypothesis cannot be entirely dismissed. As a prevalent and potentially modifiable risk factor, nasal obstruction warrants attention in clinical and epidemiologic investigations of sleep-disordered breathing. Toward the goal of identifying and quantifying risk factors for sleep-disordered breathing, we investigated the role of chronic and acute nasal congestion, measured subjectively and objectively, in the ongoing Wisconsin Sleep Cohort Study.

METHODS

All men and women 30 to 60 years of age employed at one of five large state agencies in south central Wisconsin were surveyed about sociodemographic factors, sleep characteristics, brief medical history, and potential risk factors for sleep-disordered breathing. The job categories ranged from unskilled labor to professional at each agency. A total of 4927 questionnaires were completed, for a response rate of 75%. Of particular relevance to this investigation, questions were asked about the frequency of snoring and other sleep disturbances, somnolence, nasal congestion, and allergy history. Participants were asked how often they had nasal obstruction, congestion, or discharge as a sleeping problem at night. Possible responses were never, rarely (once a month), sometimes (2 to 4 times per month), often (5 to 15 times per month), or almost always (16 to 30 times per month). The respondents also were asked whether they had allergies, such as hay fever, that caused nasal congestion and whether they took medication for allergies.

With the group of survey participants as a well-defined sampling frame, a stratified random cohort of men and women was recruited for an extensive study protocol that included overnight studies every 4 years. The recruitment is ongoing, and the response rate has averaged 50% of those invited. The data for this analysis of nasal obstruction and sleep-disordered breathing included overnight polysomnography findings, measurement of nasal patency by means of rhinometry, medical history, and self-reported daytime somnolence and sleep habits. The cohort reported herein consists of 911 participants who had complete data for these items.

Polysomnography included recording of sleep-state data

TABLE III. Prevalence of habitual snoring and hypersomnolence by frequency of nighttime symptoms of rhinitis (n = 4927)

	Frequency of nighttime symptoms of rhinitis (nights/month)*				
Sleep problem	**Never**	**Rarely (1)**	**Sometimes (2–4)**	**Often (5–15)**	**Always or almost always (>15)**
Habitual snoring	23	25	32	36	47
Chronic excessive daytime sleepiness	10	12	17	25	30
Chronic nonrestorative sleep	17	20	27	37	37

Values are percentage of participants who experienced the sleep problem. Overall χ square $p < 0.0001$.
*Self-reported occurrence of nighttime nasal congestion or discharge.

TABLE IV. Relation between chronic symptoms of rhinitis* and snoring and hypersomnolence (n = 4927)

Sleep problem	Odds ratio† for chronic nighttime rhinitis symptoms	p Value
Snoring		
Never or rarely	1.0‡	
Occasional (1–3 nights/week)	1.3	0.02
Habitual (>3 nights/week)	2.0	<0.0001
Chronic excessive daytime sleepiness	2.4	0.001
Chronic nonrestorative sleep	2.2	<0.0001

*Frequency of rhinitis symptoms 5 nights or more per month.
†Adjusted for age, sex, and body mass index.
‡Reference category.

TABLE V. Nasal air flow by snoring status for the cohort sample (n = 911)

Snoring frequency	Total nasal airflow* (mL/sec at -1.5 cm H_2O) Mean	Standard error
Never or rarely	547	17.3
Occasional (1–3 nights/week)	527	18.5
Habitual (>3 nights/week)	498†	11.4

*Measured by means of rhinometry before polysomnography; adjusted for age, sex, and body mass index.
†p = 0.02 in comparison with nonsnorers.

(electroencephalographic, -oculographic, and -myelographic findings), breathing patterns (oral airflow, nasal airflow, rib cage and abdominal excursions), heart rate (electrocardiogram), and oximetry data (Young et al.[18]). All records were scored by trained technicians and reviewed by one of two sleep clinicians. Events with no air flow for 10 seconds or more were scored as apnea, and events with a 40% or more reduction in respiratory effort accompanied by 4% or more desaturation were scored as hypopnea.

To describe sleep-disordered breathing, AHI (total number of episodes of apnea or hypopnea divided by hours of sleep) was calculated for each participant. Snoring status was based on the participant's responses to a question on snoring frequency according to what bedpartners and others had told them and on AHI from polysomnography. Participants were coded as *habitual snorers* if they reported a snoring frequency of 3 to 7 nights per week regardless of AHI, *occasional snorers* if they reported snoring 1 to 3 nights per week regardless of AHI, and *simple snorers* if they were coded as habitual snorers and their AHI was less than 5.

Each subject was interviewed regarding health history, life style, and other factors. The following questions regarding nasal obstruction were asked: (1) Have you had any nasal congestion or stuffiness today or tonight (or both)? (2) If yes, do you know what caused the stuffiness? The interviewer specifically asked whether the cause was a cold, allergy, or other (which was specified). If the answer was allergy, the cause of the allergy was discussed. (3) Are there times during the year when you regularly experience nasal congestion or stuffiness at night? If yes, the interviewer asked whether this was seasonal or year-round; if seasonal, the seasons were specified. The cause of chronic stuffiness was discussed. (4) Do you have any other problems that cause nasal stuffiness at night? If yes, the causes were specified.

Nasal patency was measured by means of the technique of single nostril anterior rhinometry.[19] The equipment used for this technique consisted of plastic tubes held at the nares, a pneumotachometer, and pressure transducers. Airflow velocity (mL/sec) was measured at a pressure differential of -1.5 cm H_2O. The device was calibrated before use with a standard resistor at a reading of 250 mL/sec. Measurements were made with the participant in a seated position; readings were taken in the right nostril and then the left nostril. The validity of this technique has been assessed in clinical trials of patients with seasonal allergic rhinitis and acute coryza. Limited normative data, by age and sex, describing this technique are available.[20] Body mass index (BMI, weight/height^2) was used to describe body habitus.

DATA ANALYSIS

Data were analyzed with software modules for descriptive statistics, contingency tables, multiple linear regression, and logistic regression. Mean airflow was adjusted for confounding factors, including age, sex, and BMI, by means of the general linear models procedure. Multiple regression modeling was used to estimate differences in AHI which may be due to nasal congestion and decreased airflow. For categorical outcome variables of hypersomnolence, snoring, and sleep-disordered breathing categories based on AHI cutpoints, logistic modeling was used.

Age, sex, and body habitus (including measurements of height, weight, BMI, skin folds, waist, hip and neck

TABLE VI. Nasal airflow by AHI severity category for the cohort sample (n = 911)

AHI Category	Total nasal air flow* (mL/sec at −1.5 cm H_2O) Mean	Standard error
<5, nonsnorer, no SDB	542	13.7
<5, habitual snorer†	491	13.6‡
= 5-15	510	22.0
>15	530	28.2

AHI, Total number of apneas or hypopneas during one hour of sleep; *SDB,* sleep-disordered breathing.
*Measured by means of rhinometry before polysomnography.
†Defined as simple snorers
‡p = 0.01 in comparison with nonsnorers with no sleep-disordered breathing.

TABLE VII. Association between nasal obstruction and severity category of sleep-disordered breathing* (n = 911)

AHI	Chronic nighttime symptoms of rhinitis: Odds ratio	p Value‡	Nasal congestion due to allergy†: Odds ratio	p Value‡
<5, nonsnorer, no SDB	1.0		1.0	
<5, habitual snorer§	2.1	0.0001	1.5	0.04
5–15	1.6	0.1	1.0	0.9
>15	1.5	0.3	1.8	0.04

AHI, Total number of episodes of apneas or hypopneas during one hour of sleep; *SDB,* sleep-disordered breathing.
*Adjusted for age, sex, and body mass index.
†Self-reported condition before polysomnography.
‡Comparison with no SDB.
§Defined as simple snorers.

circumferences) were investigated as confounding factors. The statistical significance of linear regression coefficients was assessed by means of t tests, and that of logistic regression coefficients was assessed by means of Wald χ-square tests. Two-tailed p values less than 0.05 were considered to indicate statistical significance.

RESULTS

Characteristics of the survey sample (n = 4927) are shown in Table I and those of the cohort sample (n = 911) in Table II. The prevalence of allergic and acute rhinitis was comparable with population estimates at 23% to 35%.[21] Airflow measurements (mL/sec at −1.5 cm H_2O) were in the range of limited normative data available.[20] Results of analyses of the survey sample are provided in Tables III and IV. Table III shows the prevalence of habitual snoring, chronic excessive daytime sleepiness, and chronic nonrestorative sleep (i.e., not feeling rested regardless of sleep amount) for each frequency category of nighttime rhinitis symptoms. Participants who often or almost always experienced nighttime symptoms of rhinitis (defined as those with chronic symptoms of rhinitis), compared with those who rarely or never had symptoms, were significantly ($p < 0.0001$) more likely to report habitual snoring, chronic excessive daytime sleepiness, or chronic nonrestorative sleep. The independent association of nighttime symptoms of rhinitis with snoring and sleepiness is shown in Table IV. Odds ratios, adjusted for sex, age, and BMI, indicated that participants with symptoms of rhinitis often or almost always, compared with those without symptoms, were 1.3 times as likely to be occasional snorers, twice as likely to be habitual snorers, and more than twice as likely to be hypersomnolent or feel unrested habitually. All associations were statistically significant.

Nasal airflow, measured in the cohort sample by means of rhinometry at −1.5 cm H_2O pressure differential, is shown by snoring status in Table V and by AHI in Table VI. All air flow values are adjusted for age, sex, and BMI. Habitual snorers, compared with nonsnorers, had significantly lower airflow ($p < 0.02$). There was a suggestion of a trend toward decreased airflow with increased snoring frequency. However, as indicated in Table VI, a linear relation between decreasing airflow and increasing AHI does not exist. Although participants in all AHI categories that indicated the presence of sleep-disordered breathing, including snoring with AHI less than 5, had lower airflow than those without sleep-disordered breathing, there appeared to be no difference between simple snorers and those with a high AHI. Similarly, linear regression analysis of AHI and airflow as a continuous variable showed no significant association between AHI and airflow. The regression coefficients for airflow in each nostril were very small (beta = .001 and .005, respectively); a decrease in flow of 100 ml/sec was associated with an increase of only one apnea or hypopnea over the entire sleep time.

Results of self-reported nasal congestion as predictors of severity of sleep-disordered breathing are shown in Table VII. Participants with chronic nighttime rhinitis were twice as likely as participants with no nasal congestion to be simple snorers. There was no linear trend, however, and the odds ratios for nighttime symptoms of rhinitis at AHIs of 5 and 15 were not statistically significant. Participants who reported nasal congestion due to allergy were about 1.5 times more likely to be simple snorers than were those with no nasal congestion. Although AHI greater than 15 was associated with a significant odds ratio (1.8), a linear trend was not clear for the relation between nasal congestion due to allergy and increasing AHI.

DISCUSSION

Indicators of nasal obstruction, including self-reported congestion and objectively measured flow, were associated with sleep-disordered breathing as evidenced by habitual snoring or worse sleep-disordered breathing. Although a linear trend between decreased nasal airflow and greater AHI was not observed, habitual snoring was consistently associated with decreased nasal airflow, self-reported stuffiness attributed to allergy, and self-reported nighttime nasal congestion or discharge. The lack of a linear relation between nasal obstruction and severity of sleep-disordered breathing is not consistent with the physiologic hypothesis that increased nasal resistance and decreased flow increase the frequency of airway collapse. In their comprehensive review of research on nasal obstruction and sleep-disordered breathing, Olsen and Kern[5] stated that nasal obstruction is more likely to cause snoring than mild or severe obstructive sleep apnea (with frank apnea and hypopnea), and that degree of nasal obstruction and severity of sleep-disordered breathing are not directly correlated. The findings from the Oxford epidemiologic study[17] were consistent with this conclusion. Our findings at an epidemiologic level of inquiry, including the present findings, are in agreement with the overall state of current clinical research.

The possibility of a linear relation cannot be entirely dismissed at this point, however, on the basis of our findings or those of previous studies. It is possible that there is a linear trend but that it is too subtle to be detected in our study on the basis of one-time measurements of AHI and nasal airflow. Like nearly all previous investigators, we were not able to monitor airflow continuously during the study, and airflow could have changed considerably during the night. Participants may have been misclassified with regard to actual airflow when sleep-disordered breathing events occurred. However, no linear relation to sleep-disordered breathing was found in a study in which nasal resistance was continuously measured during sleep.[15] Furthermore, in that study,[15] nasal resistance during sleep was not significantly different from resistance measured during waking hours. It is possible, however, that the invasiveness of the method used, including taping the mouth closed, confounded the results. In summary, the hypothesis of a linear relation between nasal resistance and sleep-disordered breathing has not been thoroughly tested, yet it cannot be supported by the limited data currently available.

An additional finding from our analyses was that self-reported chronic symptoms of rhinitis were significantly related to excessive daytime sleepiness and not feeling rested regardless of amount of sleep. Sleep-disordered breathing, including habitual snoring, is related to hypersomnolence; therefore, associations between rhinitis and sleepiness may be explained by sleep-disordered breathing. However, it is possible that rhinitis symptoms, independent of their effect on breathing, may cause cortical arousal and fragmented sleep. This association warrants further investigation for the management of rhinitis, because somnolence due to sleep fragmentation may be compounded by sleepiness caused by daytime use of medication.

CONCLUSIONS

Sleep-disordered breathing, particularly simple snoring, which represents the mildest form, has a high prevalence among adults.[18] Our analysis showed that in a population-based sample, middle-aged men and women with nasal obstruction, particularly those with chronic nighttime symptoms of rhinitis, are significantly more likely to be habitual snorers. A proportion of these patients also may have more severe sleep-disordered breathing with frequent episodes of apnea and hypopnea. The findings suggest that nasal obstruction is a risk factor for sleep-disordered breathing but do not support a linear association between obstruction and severity of sleep-disordered breathing. Allergic rhinitis is one of the most common causes of nasal obstruction and discharge at night. As a modifiable risk factor, allergic rhinitis warrants further study into the causal nature of its association with sleep-disordered breathing.

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