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Effects of seasonal smog on asthma and COPD exacerbations requiring emergency visits in Chiang Mai, Thailand

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Abstract

Background: Seasonal smog produces particulate matters that are less than 10 microns in diameter (PM_{10}), which are known to have several impacts on the respiratory system.

Objective: This study was to determine the association of an increased PM_{10} level due to seasonal smog in Chiang Mai and emergency visits for asthma and chronic obstructive pulmonary disease (COPD) exacerbations.

Method: A retrospective cross-sectional study was conducted between the months of January and March from 2006 until 2009. The association of an increased PM_{10} level and the daily number of asthma and COPD exacerbations were analyzed using a generalized linear model; a Poisson regression model was fit to the number of daily emergency visits using predictor variables: lags of PM_{10}, day of the week, and time.

Results: There were a total of 917 emergency visits for acute exacerbations of asthma and COPD, with a median of 2 visits per day (range 0-10). The median PM_{10} level during the same interval was 64.5 microgram per cubic meter ($\mu g/m^3$) (16-304). For every 10 $\mu g/m^3$ rise in PM_{10} concentration, there was a lag time of 6 days for asthma exacerbations [Adjusted relative risk (RR)=1.020; 95% confident interval (CI), 1.001-1.040; (p=0.014)], 7 days for COPD exacerbations [RR=1.030; 95%CI, 1.010-1.050 (p=0.024)] and 7 days for all exacerbations [RR=1.030 95%CI, 1.010-1.040 (p<0.001)].

Conclusions: This study confirms the effect of increasing PM_{10} concentrations from seasonal smog on asthma and COPD exacerbations. However, there was an approximately 1 week lag time between the elevated PM_{10} levels and time to emergency visits due to disease exacerbation.

Key words: asthma, chronic obstructive pulmonary disease, pollution, exacerbation, emergency

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Introduction

Chiang Mai, with an altitude of approximately 310 meters above sea level, is situated approximately 700 kilometers from Bangkok, and is one of the largest cities in Thailand covering an area of approximately 20,107 km^2. Its population is around 1.7 million people, distributed among 24 administrative districts. It is surrounded by high mountain ranges. Due to its geographical features, Chiang Mai, as well as some provinces in the northern part of Thailand, has been annually facing air pollution during the dry season. The air pollution in northern Thailand has been recognized as seasonal smog crisis from January to April every year since 2006.[1] The severe air pollution from hazes of northern Thailand has been empirical from the air quality data of the Pollution Control Department's monitoring stations in the northern areas.[2] For example, particulate matter with a diameter of less than 10 microns (PM_{10}) reached a peak concentration on March 14th 2007, at 383 micrograms per cubic meter ($\mu g/m^3$), which was three times higher than Thailand's acceptable safety concentration (120 $\mu g/m^3$)[2] and seven times higher than that of World Health Organization (WHO)(50$\mu g/m^3$).[3] The major sources of hazes during crisis were forest fires; open burnings in the agricultural settings; and garbage burnings.

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Asthma is among the chronic diseases that affect people worldwide. The prevalence of asthma in adults aged 20-44 years of northern Thailand in 2001-2002 was 3.01%.[4] Chronic obstructive pulmonary disease (COPD) is also among the chronic diseases distributed worldwide. The prevalence of COPD in adults aged over 40 years in Chiang Mai was 5.4% in urban areas.[5] Several studies have been well documented showing the adverse effects of air pollution linked with respiratory and cardiovascular morbidity and mortality.[6-16] The effect of PM_{10} from traffic pollutants on lung function was also demonstrated in Bangkok, Thailand.[17]

In this study, our aim was to determine the association of an increased PM_{10} level and emergency visits for asthma and COPD exacerbations of patients residing in municipal areas of Chiang Mai, Thailand.

Methods

Design and study participants

A retrospective cross-sectional study was conducted between the months of January and March from 2006 until 2009 in Chiang Mai, Thailand. Since Chiang Mai has been annually affected by the seasonal smog crisis and has an air quality monitoring station located at the center of the city, we recruited COPD and asthmatic patients with the following eligibility criteria: (1) had COPD or asthma diagnosed by physicians (2) experienced COPD or asthma symptoms requiring medical treatment during the past year, (3) aged greater than 40 years old in COPD and greater than 15 years old in asthma, and (4) living in municipal areas of Chiang Mai for more than 3 years. Records of daily emergency visits due to acute exacerbations of COPD and asthma were collected from Chiang Mai University and Chiang Mai Ram hospitals which are the only two tertiary care hospitals located in municipal areas of Chiang Mai district. The daily number of emergency visits due to acute exacerbations of COPD and asthma were recorded by the emergency physicians with the primary diagnoses based on the International Classification of Diseases (ICD) version 10 (ICD-10 J44.1, and J45.901 for COPD and asthma exacerbation, respectively). The study was approved by the Ethics Committee of the Faculty of Medicine, Chiang Mai University (Study code: 09DEC241266, Date approval: 4th January 2010).

Measurements of air pollutants (PM_{10}) and meteorological parameters

Sampling station located in municipal areas of Chiang Mai district, Chiang Mai. Ambient air concentrations of pollutants were measured by the Pollution Control Department, Ministry of National Resources and Environment with the continuous automated air sampling monitoring station located at the center of the city. The analysis method for carbon monoxide (CO) concentrations was non-dispersive infrared detection; for sulfur dioxide (SO_2) concentrations it was the pararosaniline technique; for nitrogen dioxide (NO_2) and ozone (O_3) concentrations it was the chemiluminescence technique; and for PM_{10} and $PM_{2.5}$ it was the gravimetric technique.[18] The data reported were daily average concentrations for all parameters. We also obtained the meteorological data, temperature and relative humidity from the Northern Meteorology Center, Chiang Mai province on a daily basis.

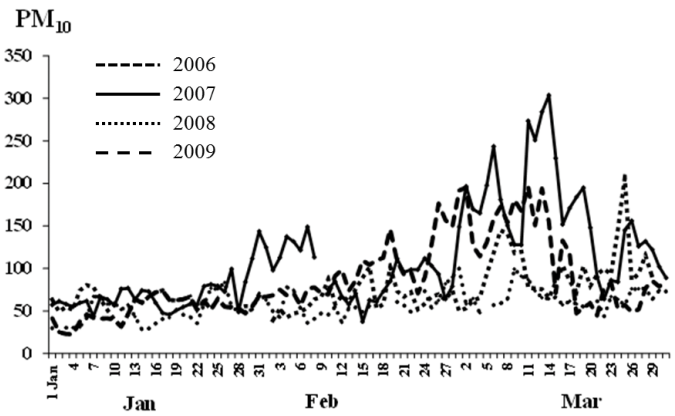
Statistical analysis

Results for numerical values were expressed as means±SD or median, IQR (Interquartile range) and those for categorical data were expressed as absolute frequencies and percentages. The association between daily number of asthma and COPD exacerbations and PM_{10} concentrations was analyzed by the application of general linear models (GLM) with Poisson distribution,[19] a method of analysis which has been found to perform satisfactorily in previous studies.[16,20,21] Poisson models with log links are often called log-linear models and are used for frequency data. To determine the association between effects of PM_{10} on disease exacerbation, Poisson regression was used for analysis after adjustment for SO_2, NO_2, CO, O_3, temperature, and humidity. To assess the lag structure between concentration of PM_{10} level and emergency department visits, we initially examined separate models for each lag from 0 to 7 days before the emergency visit. The lag time zero (lag0) is the day of PM_{10} measurement. Finally, risk regression analysis is applied to the data in order to estimate risk ratios (RR) with 95% confidence intervals (CI) of the independent variables in the constructed model. All analyses were carried out with the SPSS statistical package, version 16 for Windows (SPSS Inc. IL, USA).

Results

A total of 917 emergency department visits were made for 740 patients (223 asthma and 517 COPD), with 389 males and 351 females. Their mean age was 64.9 years with a standard deviation of 18.8 years. The crisis period of PM_{10} during January to March from 2006 to 2009 is shown in Figure 1. The daily pollutant data included: PM_{10}, SO_2, NO_2, CO, and O_3, and are also shown in Table 1.

Figure 1. The crisis period of PM_{10} during January to March from 2006 to 2009.



The association between emergency visits of all exacerbations of COPD and asthma and the concentration of PM_{10} after adjustment with SO_2, NO_2, CO, O_3 and humidity is shown in Table 2 (RR 1.030; 95%CI 1.010-1.040).

Table 1. Daily pollutants data (between the months of January and March of each year from 2006 until 2009)

Time in month	PM_{10} (μg/m³)	SO_2 (ppb)	NO_2 (ppb)	CO (ppm)	O_3 (ppb)
2006					
January	47.3 (34.2-58.4)***	1.3 (1.1-1.5)***	12.8 (10.8-14.6)	0.6 (0.3-0.7)***	20.3 (15.8-22.9)***
February	53.6 (47.6-62.7)***	1.3 (1.1-1.4)***	10.8 (4.5-15.6)	0.6 (0.5-0.7)**	26.1 (22.9-31.6)
March	83.8 (63.9-95.3)	0.5 (0.3-0.8)	11.0 (8.8-14.7)	0.9 (0.6-1.1)	30.0 (26.1-33.6)
2007					
January	58.0 (52.1-72.5)***	0.5 (0.3-0.7)	17.1 (15.1-18.4)***	1.1 (0.9-1.1)***	19.5 (16.8-22.6)***
February	93.3 (68.2-112.9)***	0.2 (0.0-1.0)	17.9 (14.1-21.4)**	0.9 (0.7-1.1)***	26.8 (20.2-29.4)***
March	151.8 (124.5-196.6)	0.4 (0.1-1.2)	23.6 (16.9-28.0)	1.5 (1.3-1.8)	37.2 (34.3-40.1)
2008					
January	52.7 (43.3-70.0)***	0.1 (0.0-0.3)**	13.0 (11.4-15.2)	0.7 (0.5-0.8)***	25.0 (23.6-29.0)***
February	55.5 (43.9-68.9)***	0.2 (0.0-0.8)*	7.3 (6.0-8.7)***	0.6 (0.5-0.7)***	29.2 (24.9-34.7)**
March	75.2 (66.8-94.7)	0.6 (0.2-1.0)	14.1 (10.0-15.4)	0.8 (0.7-0.9)	34.6 (30.3-41.2)
2009					
January	35.2 (27.5-43.3)***	0.5 (0.2-0.7)**	13.9 (13.4-15.6)**	0.8 (0.7-0.9)	25.6 (23.9-29.7)***
February	68.2 (49.3-94.4)**	0.7 (0.1-1.1)*	16.7 (15.4-20.8)	0.7 (0.6-1.0)	35.6 (32.8-42.3)
March	100.6 (63.2-116.1)	1.6 (0.4-2.3)	19.4 (15.4-25.8)	0.8 (0.4-1.1)	38.6 (32.6-41.4)

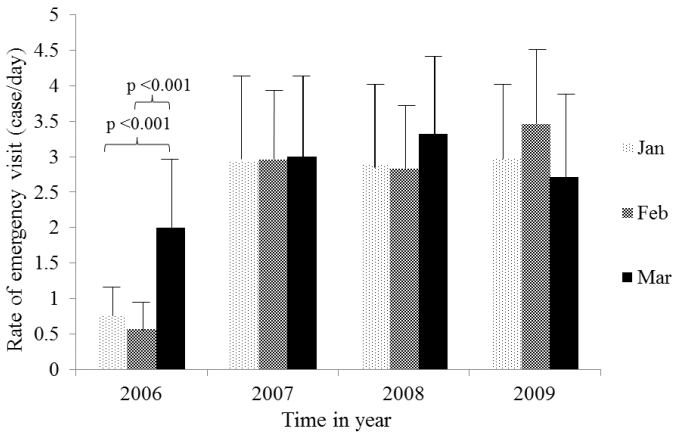
Note: Data are median and IQR. * p<0.05, ** p<0.01, and ***p<0.001 comparison with data in March of each year
Abbreviations: IQR, interquartile range; PM_{10}, Particulate matters with diameter of less than 10 micron; m^3, per cubic meter; SO_2, sulfur dioxide; NO_2, nitrogen dioxide; CO, carbon monoxide; O_3, ozone

Rate of emergency visits (cases/day) from January-March of each year from 2006 until 2009 is shown in Figure 2. Rate of emergency visits of asthma and COPD exacerbation was significant higher in March when compared to January and February only in year 2006.

Figure 3 summarizes the association between PM_{10} and emergency visits due to asthma and COPD exacerbations after adjusting for SO_2, NO_2, CO, O_3, temperature, and humidity. An increase in 10 μg/m³ of PM_{10} was significantly associated with increased risks for acute exacerbation for all subjects at a lag day ranging from 0 to 7 and cumulative lag days ranging from 0-7. The strongest effect on acute asthma attacks was observed with a cumulative lag day of 6 days (Adjusted RR 1.020, 95% CI, 1.001-1.040, p=0.014), but the strongest effect on acute exacerbation of COPD was observed with a cumulative lag day of 7 days (Adjusted RR 1.030, 95% CI, 1.010-1.050, p=0.024). In all patients, the strongest effect on acute exacerbation was observed with a cumulative lag day of 7 days (Adjusted RR 1.030, 95% CI, 1.010-1.040, p<0.001).

Table 3 summarizes the results of the PM_{10} less than or higher than the standard value of the World Health Organization (50μg/m³) analysis for emergency visit due to COPD and asthma exacerbations. Asthma, COPD, and all patients were significantly associated with increased risks for exacerbation when the PM_{10} is higher than 50 μg/m³ [RR 1.356 (95% CI, 1.051-1.750 p=0.019), RR 1.293 (95% CI, 1.016-1.645 p=0.037), and RR 1.342 (95%CI, 1.148-1.568 p<0.001) respectively]. The PM_{10} higher than 50 μg/m³ was also implicated with a cumulative lag 1 day [RR 1.364 (95% CI, 1.005-1.763 p=0.018), RR 1.241 (95% CI, 1.022-1.507 p=0.029), and RR 1.290 (95%, CI 1.104-1.506 p=0.001), respectively].

Figure 2. Rate of emergency visit (case/day) from January-March of each year from 2006 until 2009.



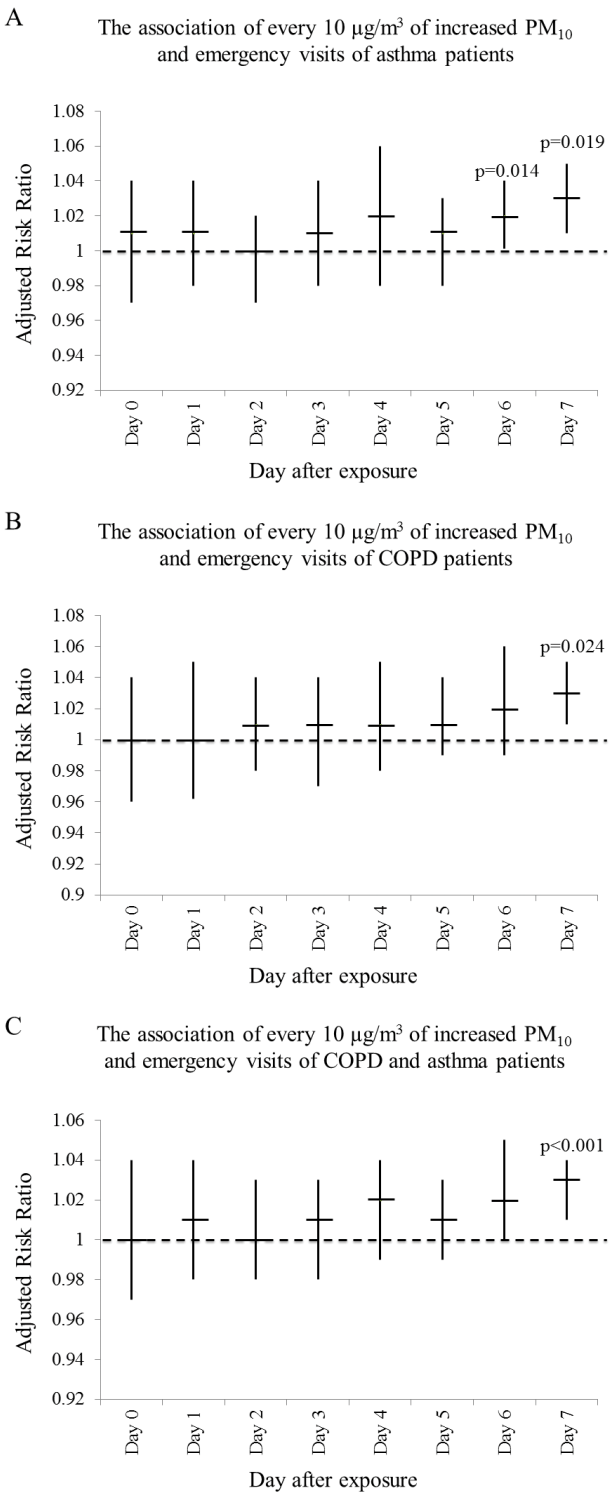
Note: Data are mean and standard deviation

Table 2. The association between emergency visits of all exacerbations of COPD and asthma and concentration of PM_{10} after adjusted with SO_2, NO_2, CO, O_3 and humidity

Parameters	Adjusted RR	95% Confidence Interval
PM_{10} lag day 7	1.030	1.010-1.040
SO_2	.904	.814-1.004
NO_2	.997	.978–1.017
CO	1.152	.747–1.774
O_3	1.018	.996–1.039
Humidity	1.007	.979–1.035

Abbreviations: COPD, chronic obstructive pulmonary disease; RR, risk ratio; PM_{10}, Particulate matters with diameter of less than 10 micron; SO_2, sulfur dioxide; NO_2, nitrogen dioxide; CO, carbon monoxide; O_3, ozone

Figure 3. Correlation of every 10 μg/m³ of increased PM_{10} and emergency visits after adjusted for SO_2, NO_2, CO, O_3 and humidity.



Note: Horizontal lines represent adjusted risk ratio; error bars represent 95% confidence intervals (CI), figure 2A, 2B, and 2C for asthma, COPD, and COPD and asthma respectively

Abbreviations: COPD, chronic obstructive pulmonary disease

Table 3. Risk of asthma and COPD exacerbations required emergency visits compare between days with PM_{10} below and above WHO cut off values (PM_{10}>50μg/m³)

Disease	WHO PM_{10} cut off	RR	95%CI	p-value
Asthma	PM_{10}≤50μg/m³	1		
	PM_{10}>50μg/m³	1.356	1.051-1.750	0.019
COPD	PM_{10}≤50μg/m³	1		
	PM_{10}>50μg/m³	1.293	1.016-1.645	0.037
Total	PM_{10}≤50μg/m³	1		
	PM_{10}>50μg/m³	1.342	1.148-1.568	<0.001

Abbreviations: COPD, chronic obstructive pulmonary disease; WHO, World Health Organization; RR, risk ratio; CI, confidence interval; PM_{10}, Particulate matters with diameter of less than 10 micron

Discussion

The air pollution in northern Thailand has been recognized as a seasonal smog crisis which has increased to the peak level of PM_{10} during January to April every year since 2007.[1] This time-series study aimed to determine the association of an increased PM_{10} level and COPD and asthma exacerbations requiring emergency visits. The scope of this study is focused on the data obtained from two tertiary care hospitals located in the municipal areas of Chiang Mai; Chiang Mai University Hospital which represents the government hospital; and Chiang Mai Ram hospital which represents the private hospital. Our results showed that a total of 917 emergency visits were made for 740 patients (517 COPD and 223 asthma). Interestingly, the rate of acute exacerbation did not increase immediately on the day of increase level of PM_{10} but several days after exposure to PM_{10}. Our study found associations between increased levels of PM_{10} in every 10 μg/m³ and emergency visits with the following periods: lag time 7 days for COPD, lag time 6 days for asthma, and lag time 7 day for both diseases. It could be further explained that the rate of acute exacerbation requiring emergency visits for COPD increased the relative risk by 1.03 or 3% in 7 days, while for asthma increased the relative risk by 1.02 or 2% in 6 days after the PM_{10} level increased by 10 μg/m³ from the previous day. For both diseases, the emergency visits increased relative risk 1.03 or 3% in 7 days after the PM_{10} level increased every 10 μg/m³ from the previous day. Previous studies reported an association between an increase of 10 μg/m³ for PM_{10} and increase in respiratory admissions within the range of 0.8%-3.4%.[11,15] Our result for asthma was similar to that of the previous study in which the lag structure between pollutant levels and emergency visits (separate models for each lag) and the risk ratios for asthma visits were generally positive and strongest with a lag of 5 to 8 days, but in COPD the lag period was shorter, the associations for emergency visits were generally positive and strongest for same-day pollutant levels and for levels lagged by 1 day.[22] A previous study in Brazil found that asthma attacks increased shortly after the level of TSP (total suspended particle) generated from pre-harvest sugar cane burning was increased for 1-5 days RR=11.6% (95% CI, 5.4-17.7).[23] These mechanisms in COPD and asthma could be explained by the activation mechanism of inflammation causing tissue damage and subsequently increasing the

sensitivity of the trachea. A previous review has summarized the role of increased particles, especially ultrafine particles, in exacerbations. Ultrafine particles and transition metals are common components of particles that cause oxidative stress, which may enhance pro-inflammatory effects in the airways of patients that are already inflamed by disease. Infection with adenovirus and other pathogens may also interact with oxidative stress and particles to promote exacerbations.[24] A longer lag period for emergency department visits observed in our study is plausible for less severe respiratory conditions for biologic reasons (an underlying distribution of sensitivity or illness severity in the population) and for behavioral reasons (the time it takes for an exacerbation to become serious enough to necessitate a visit), especially compared with outcomes such as an acute cardiac event.[22] Our findings could be helpful to hospitals located in pollution areas, enabling them to cope with the increased number of emergency visits due to increased PM_{10}. Our study found that the acute exacerbation of COPD and asthma requiring emergency visits was associated with levels of PM_{10} that were higher than the WHO standard of 50 μg/m^3. On the day when PM_{10} was higher than 50 μg/m^3, the increased rates of acute exacerbation of COPD, asthma, and total patients were 29.3%, 35.6% and 34.2%, respectively. These findings support a WHO standard level of PM_{10} less than 50 μg/m^3; however, the cut-off point standard level of PM_{10} in Thailand is still defined as 120 μg/m^3. We hope that our findings will raise awareness about pollution levels and the impact on health, especially in patients with respiratory diseases. Further studies need to be done to investigate the quality of life and health effect in a large sample size of patients with respiratory diseases, other chronic disease, and the normal population.

The strengths of this study were firstly, we used time series analysis to assess the trends and relationships using a generalized estimating equation with Poisson regression analysis, which is in the same format as the previous epidemiologic study.[6] Secondly, we also adjusted the other pollutants including SO_2, NO_2, CO, O_3, temperature, and humidity. Thirdly, we selected only Chiang Mai dwellers living in municipal areas exposed to seasonal smog during the entire study period.

This study has some limitations. Firstly, although acute exacerbations of COPD and asthma were diagnosed by responsible physicians at emergency rooms based on ICD-10 as previously reported,[25] some relevant data such as clinical disease severity, pulmonary function, history of frequency of acute exacerbation, and treatment were not usually available in routine electronic medical records. Therefore, our results were not adjusted for non-meteorological parameters. Secondly, annual data on distribution of PM_{10} concentration and emergency visits was not available as our study focused on the crisis period of PM_{10} during January to March 2006-2009. Thirdly, we have no epidemiological data for influenza infection over the study period; therefore, this may be confounded in the rate of acute exacerbation. Although we collected data on acute exacerbation over the same period each year, this confounder may potentially skew the results of this study.

In conclusion, this epidemiology study investigated the effect of pollution on the health of people in Chiang Mai, which found that the increased level of PM_{10} results in the acute exacerbation rate of COPD and asthma requiring emergency visits. These occurrences of acute exacerbations were after a period of 6-7 days. On the day when PM_{10} was higher than 50 μg/m^3, there were increased rates of acute exacerbations of COPD, asthma, and total patients.

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Author contributions

The first author developed study design and carried out acquisition and interpretation of data, statistical analysis, manuscript preparation, and critical revision of intellectual contents. The other authors conducted the acquisition and interpretation of data and critical reviews of the manuscript. All authors read and approved the final manuscript.

Conflict of interests

The authors have no conflicts of interest in connection with the work submitted.

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