Table 1 Clinical characteristics and microbiological findings of 231 patients with community acquired pneumonia

	Enterovirus (n = 12)*	Rhinovirus (n = 7)	Influenza A (n = 17)*	Other respiratory viruses (n = 12)	Other or undetermined aetiology (n = 184)
Age (y) (mean (SD))	45.9 (18.5)	44.6 (19.9)	64.2 (16.7)	50.3 (23.4)	48.8 (17.7)
Males (n (%))	6 (50)	6 (86)	5 (29)	7 (58)	103 (56)
Underlying disease (n (%))	4 (33)	4 (57)	9 (53)	3 (25)	73 (40)
COPD or asthma (n (%))	2 (17)	3 (43)	4 (24)	1 (8)	23 (13)
Cardiovascular disease (n (%))	2 (17)	1 (14)	3 (18)	2 (17)	29 (16)
Smoker (n (%))	4 (33)	4 (57)	3 (18)	1 (8)	59 (32)
PSI class IV–V (n (%))	2 (17)	2 (29)	8 (47)	2 (17)	35 (19)
Died (n (%))	0	1 (14)	3 (18)	0	1 (5)
Streptococcus pneumoniae (n (%))	3 (25)	4 (57)	6 (35)	2 (17)	50 (27)

COPD, chronic obstructive pulmonary disease; PSI, Pneumonia Severity Index.
*Two patients with enterovirus and influenza A.

important viruses causing this disease. Collectively, our findings corroborate those of Jennings and colleagues[1] and support their conclusion that the importance of both viral pneumonia and mixed viral/bacterial pneumonia may be greater than previously realised.

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Leptin and regulatory T cells in obese patients with asthma

Taylor and colleagues[1] demonstrated a significant association between asthma severity and obesity. However, the mechanisms underlying this association are not fully understood. We suggest that the increase in asthma severity in obese patients might also be related to a defective function of regulatory T cells (Tregs).

Tregs play an essential role in immune homeostasis and protection against autoimmunity, and it has been suggested that the function of Tregs may be defective in patients with asthma.[2] On the other hand, leptin, a known hormone marker for obesity, exerts actions on multiple organ systems, including the immune system. Indeed, it has been shown that leptin signalling negatively modulates Treg function.[3]

Therefore, the increase in asthma severity observed in obese patients might by caused, in part, by a decreased immunological tolerance induced by a decreased function of Tregs mediated by leptin. Moreover, it has been suggested that induction of Treg development might be a useful tool for asthma treatment.[2] However, Treg increases might also increase cancer risk by impairing host antitumor immune response.[4] Thus the safest way to improve asthma in obese patients is to lose weight.

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Thunderstorm associated asthma in Atlanta, Georgia

Associations between thunderstorm activity and asthma morbidity have been reported in numerous locations around the world.[1] The most prominent hypotheses explaining the associations are that pollen grains rupture by osmotic shock in rainwater, releasing allergens, and that gusty winds from thunderstorm downdrafts spread particles and/or aeroallergens, which may ultimately increase the risk of asthma attacks. A full understanding of "thunderstorm asthma" is crucial, especially with projections of increases in heavy rainfall, thunderstorm events and aeroallergen concentrations as the climate system warms.[2 3] Many existing studies of this phenomenon have been limited in power and scope.[1] Our study seeks to conduct the most extensive investigation of thunderstorm occurrence and asthma morbidity to date in a region, the Southeast US, that has not previously been examined but where thunderstorms are highly prevalent.

We capitalised on the availability of an extensive emergency department (ED) visit database, consisting of data on over 10 million ED visits collected from 41 of 42 hospitals in 20 county Atlanta, Georgia, between 1993 and 2004. We selected visits for asthma (identified using the primary International Classification of Disease, 9th revision diagnosis codes 493, 786.07) by patients residing in zip codes located wholly or partially in the study area. Thunderstorm occurrence data were obtained from the automated surface observing system station at the Atlanta Hartsfield–Jackson airport, which recorded 564 thunderstorm days (12.9% of 4383 total study days). In order to test the mechanistic hypotheses of thunderstorm asthma, we also obtained total daily rainfall and maximum 5 s wind gust data. The wind gust data were used as a surrogate for thunderstorm downdrafts and to indicate the maximum wind speed of the storm. We assessed the association between thunderstorms and next day asthma ED visits using Poisson generalised linear models.[4] We controlled for long term temporal and seasonal trends and meteorological conditions with cubic splines,[5] which allow for flexible control of temporally varying confounding factors. We examined effect modification by levels of rainfall and wind speed, defined a priori by quartiles of their respective distributions.

We observed 215 832 asthma ED visits during the study period; 24 350 of these visits occurred on days following thunderstorms. In our epidemiological models, we observed an association between daily counts of asthma ED visits and thunderstorm occurrence ($p<0.001$, fig 1). Overall, asthma visits were 3% higher on days following thunderstorms. When thunderstorms were stratified by rainfall amount, associations with asthma were observed for thunderstorms with rainfall but not for thunderstorms with no recorded rainfall.