

## LETTERS

## Multi-professional lung cancer disclosure to change anxiety and depression: an exploratory study

The physician's communication style when disclosing bad news about cancer can reportedly affect the patient's psychological adjustment.<sup>1,2</sup> Little is known about changes in anxiety and depression during the initial period of lung cancer diagnosis disclosure. The primary goal of this study was to analyse the impact of a multi-professional interview for diagnosis disclosure (MIDD) on anxiety and depression in patients with newly diagnosed non-small cell lung cancer (NSCLC).

Data were obtained from a prospective study in ambulatory adults with histologically confirmed NSCLC. Depression and anxiety were assessed by the Hospitalised Anxiety and Depression Scale (HADS) twice: at admission (time 1), when the diagnosis was unknown, and after diagnosis disclosure (time 2). Median time between times 1 and 2 was 13 days. During this period, the mean length of hospitalisation was 3 days. The HADS is a 14 item scale measuring anxiety and depression.<sup>3</sup> Each subscale is scored from 0 to 21, with higher scores indicating greater distress. French validation of this tool has been conducted by Razavi and colleagues.<sup>4</sup>

The MIDD was structured in two steps:

1. Medical interview: the physician disclosed the diagnosis and the proposed treatment course according to medical guidelines about breaking bad news.<sup>5</sup> During this interview, the referent nurse observed the patient's and physician's reactions, collected data to identify themes for determining a nurse-led intervention adapted to the patient's needs and expectations (supportive communication).
2. Referent nurse interview without the physician: she asked the patient if he/she understood the information given by the physician and reformulated the physician's key words. The nurse then explained the details of the treatment procedures, checked how much more information the patient wished to know and responded to his/her reactions and questions.

Sixty-five patients were recruited. Twenty-four patients were excluded for the following reasons: histological diagnosis ( $n = 14$ ), cerebral metastasis diagnosed between times 1 and 2 ( $n = 1$ ), disclosure of lung cancer diagnosis outside of MIDD ( $n = 6$ ) and refusal to answer at time 2 ( $n = 3$ ). A subset of 41 patients was available for analysis: subjects were 33 men and eight women, aged 29–85 years (mean 61), performance status (PS) 0–1. There was no significant difference in distribution of

gender and PS between included and excluded subjects but a significant difference in marital status (patients living alone more often in excluded subjects).

Before diagnosis, the overall prevalence rates of anxiety and depression were 51% and 19.5%, respectively. After MIDD, the prevalence rates were 44% and 27%, respectively. Mean anxiety score decreased over time ( $p < 0.01$ ) although depression remained stable.

The reduction in anxiety after MIDD could be due to the fact that patients often experience anticipatory anxiety before their consultation with a physician and that, after the consultation, their anxiety generally decreases. Alternatively, there is the issue of the adequacy of the information provided by physicians. In our study, effective reformulation by the nurse of the information provided by the physician probably impacted favourably on the reduction in anxiety.

Thus in the absence of a control group, we could not conclude a real benefit of MIDD although it might be plausible. This is the first time the impact of MIDD on psychological and QOL measures has been evaluated in France.

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## The role of rhinoviruses and enteroviruses in community acquired pneumonia in adults

The article by Jennings and colleagues<sup>1</sup> described interesting findings regarding the common nature of mixed viral/bacterial aetiology in patients with community acquired pneumonia (CAP) and the association between mixed rhinovirus/pneumococcal infection and severe disease. We have also examined the role of respiratory picornaviruses as causative agents of CAP in adults and their contribution to disease severity. As part of a larger prospective clinical study<sup>2</sup> of the aetiology of CAP, the occurrence of rhinoviruses and enteroviruses was analysed in 231 patients. Detailed information on the study design has been reported previously.<sup>2</sup> In addition, throat swab specimens were examined for the presence of rhinoviruses and enteroviruses using previously described reverse transcriptase (RT)-PCR assays.<sup>3</sup>

The characteristics of the patients and microbiological findings are described in table 1. Viruses were detected in 46 (20%) patients, of whom 19 (41%) were positive for respiratory picornaviruses by RT-PCR. Among the 12 patients with enteroviruses, additional aetiological agents were identified in seven (58%), including three (25%) *Streptococcus pneumoniae*. Among the seven patients with rhinoviruses, a concomitant *S pneumoniae* infection was detected in four (57%).

It has been shown in an experimental model that adherence of *S pneumoniae* to human tracheal epithelial cells is increased in the presence of rhinovirus.<sup>4</sup> The results of Jennings and colleagues<sup>1</sup> prove this association in vivo by showing that 39% of their patients with rhinovirus identified from a nasopharyngeal sample had concurrent *S pneumoniae* infection. Consistently, as many as 57% of our patients with rhinovirus also had *S pneumoniae* infection. Rhinovirus was associated with severe disease (Pneumonia Severity Index IV-V) in 29% of cases, the percentage being somewhat lower than the 39% of severe rhinovirus associated infections reported by Jennings and colleagues.<sup>1</sup> One of our patients with mixed rhinovirus/pneumococcal infection died.

To date, only limited data exist on the role of enteroviruses in lower respiratory tract infections in non-immunocompromised adults. To our knowledge, only one previous study included the enterovirus PCR test in the diagnostic array of CAP.<sup>5</sup> Moreover, only one (0.5%) of the 198 patients in that study had enterovirus infection. Here, enterovirus was the second most common viral agent after influenza A virus, being detected in 5% of our patients. This percentage is similar to that observed in association with lower respiratory tract infection in children<sup>6</sup> in whom enteroviruses are among the most

**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)
<i>Streptococcus pneumoniae</i> (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<sup>1</sup> 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<sup>1</sup> 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.<sup>2</sup> 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.<sup>3</sup>

Therefore, the increase in asthma severity observed in obese patients might be 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.<sup>2</sup> However, Treg increases might also increase cancer risk by impairing host antitumor immune response.<sup>4</sup> 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.<sup>1</sup> 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.<sup>2,3</sup> Many existing studies of this phenomenon have been limited in power and scope.<sup>1</sup> 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.<sup>4</sup> We controlled for long term temporal and seasonal trends and meteorological conditions with cubic splines,<sup>5</sup> 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.