the B cells, monocytes, eosinophils, and Langerhans cells and plays an important role in the regulation of IgE production by B cells. This molecule is a transmembrane glycoprotein (type 2) of approximately 45 kDa and can be cleaved to yield a range of freely soluble CD23 (sCD23) fragments by the metalloprotease ADAM10. The best characterized ligands of CD23 are IgE and CD21, respectively. It was shown that antigen-IgE complexes are captured and internalized by CD23, which facilitate the antigen processing and presentation with major histocompatibility complex class II proteins. In addition, CD23 serves as a negative regulator of IgE production, which has been demonstrated through experimental animal studies in vivo. Increased production of IgE and defective antigen presentation were found in mice deficient in CD23. Accordingly, our case has defective expression of CD23 on B cells. Because of technical and financial reasons, we could analyze neither mutation of this molecule nor its soluble fragment in serum. We suggest that future studies investigating the role of CD23 (both its soluble fragments and expression on inflammatory cells, including B cells and eosinophils) and its mutation may provide some information about allergic and hypereosinophilic disorders and the mechanism of action of omalizumab on them.

The optimal duration of omalizumab treatment in allergic diseases is not known. It has been reported that lowering the recommended omalizumab dose may likely result in a clinical deterioration. On the other hand, however, after the discontinuation of the drug some effect of the therapy may continue up to 3 years. In our chronic eosinophilic pneumonia case, we plan to taper and discontinue omalizumab after completing at least 3 years of therapy.

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Weekend Effect for Pulmonary Embolism and Other Acute Cardiovascular Diseases

To the Editor:

In an issue of CHEST (September 2012), Nanchal et al presented interesting data about the existence of a “weekend effect” for pulmonary embolism. Patients admitted to the hospital on a weekend, in fact, had a 19% increased risk of death. These results represent further confirmation of previous reports from Canada (cited by the authors) and from the Emilia-Romagna region of Italy by our group that both found a 17% increased risk of death.

We have extensively explored the possible presence of a different mortality rate between weekdays and weekends and confirmed an even greater increased risk of death for other acute cardiovascular diseases as well (ie, acute heart failure [OR, 1.33]; and aortic aneurysm rupture or dissection [OR, 1.31]). Medical and nursing understaffing, shortage of diagnostic or procedural services, and presence of inexperienced residents have been suggested as possible causes. However, the data collected in Italy (and in the Emilia-Romagna region in particular) do not support such interpretations, since that health system and the hospital service organization in Italy are not comparable with that of countries like the United States or the United Kingdom.

Temporal aspects of onset of acute cardiovascular and cerebrovascular diseases might play a role as well, and patterns of circadian and seasonal times of onset of certain diseases are known. It is possible that acute diseases do not present with equal severity relative to time, that is, day of the week or hour of the day. A single-center study on acute coronary syndromes (ACSs) explored this possibility and showed that, although there were fewer ACS admissions than expected on nights and weekends, the proportion of patients with ACS presenting with ST-elevation myocardial infarctions was 64% higher on weekends. A higher severity might be linked with higher risk of mortality, and several parameters of severity collected by Nanchal et al (eg, need for mechanical ventilation, thrombolytic therapy use, or use of vasopressors) are in agreement with this. Further studies are needed to explore this intriguing relationship between time of presentation and clinical outcome of acute cardiovascular diseases.

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Response

To the Editor:

We thank Dr Manfredini and colleagues for their interest in our work. 1 We agree that from the analysis of the administrative data, we are unable to determine a cause-and-effect relationship for the weekend effect. As mentioned in their letter, we did find that fewer people than expected with pulmonary embolism (PE) were admitted over the weekend than during the week. Weekend admissions for PE also had higher severity of illness as arbitrated by the need for mechanical ventilation, thrombolysis, and vaso-pressors. However, the weekend effect persisted when we controlled for all these factors, demographics, and random hospital effects, suggesting that perhaps other variables than those mentioned are responsible. We are also in agreement that carefully planned studies should be devoted to investigate the weekend effect in PE and other acute medical conditions.

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Does Spirometry Still Measure Up in the Diagnosis of COPD?

To the Editor:

An adequate and early diagnosis of COPD is essential for an appropriate and efficient treatment of this debilitating disease. Currently, spirometry is required to establish a diagnosis of COPD in patients with chronic respiratory symptoms or in those at risk.1 According to the results reported in CHEST (December 2012) by Mohamed Hoesein et al,2 this approach would misdiagnose an important group of patients who do not (yet) fulfill the spirometry diagnostic criteria; that is, among male heavy smokers, those with higher FEV1/FVC ratios may be the ones with the fastest FEV1 decline, a hallmark of COPD. However, from their regression model that exposed potential confounders, it appears that a higher level of FEV1, actually preserved airflow.3 Adjustments for this confounder would have subsequently disfavored individuals with higher FEV1/FVC ratios. I wonder how the different groups would compare without these adjustments. Moreover, a recent article by Akkermans et al revealed a faster FEV1 decline for patients with lower FEV1/FVC ratios in both smokers and nonsmokers. Although the ECLIPSE (Evaluation of COPD Longitudinally to Identify Predictive Surrogate Endpoints), UPLIFT (Understanding Potential Long-term Impacts on Function With Tiotropium), and TORCH (Towards a Revolution in COPD Health) trials indeed found FEV1 decline to be inversely related to GOLD (Global Initiative for Chronic Obstructive Lung Disease) stage, as recognized by Mohamed Hoesein et al,3 this effect may as well be explained by the inverse relation across all studies between GOLD stage and the prevalence of current smoking, the most important factor for FEV1 decline.4,5

Whether a horse-racing effect should be acknowledged in the progression of COPD remains undecided. As yet, the current spirometry diagnostic criteria appear to remain crucial in the diagnosis of COPD. In the end, one of the striking results from this cohort appears to be the relatively high FEV1 decline in heavy smokers. Apart from an adequate diagnosis of COPD, smoking may still be the most important and practical tool to predict future disease and may as well be the most important feature at which to direct intervention.

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