

Understanding planet Academia

To the editor—In his thoughtful review of Donald Kennedy's book *Academic Duty* (*Nature Med.* 4, 241–2, 1998), Michael Zigmond expresses the wish that "Kennedy had devoted more space to a discussion of why there is a relative lack of attention to duty on the part of the professoriate. For example, he [Kennedy] asks faculty to devote more effort to their local institution, but does not discuss the relatively low percentage of faculty salaries that are provided by many of those institutions. Should we make faculty salaries a larger portion of university budgets?"

As a former dean of two universities who recently left academia after 30 years, I winced at that statement and question. Who usually covers the salary for tenured faculty when the grant runs out or the outside support otherwise is not renewed? And as for the suggestion that universities pick up a greater proportion of salaries, what planet does Dr. Zigmond live on? Has he not observed the steep climb in tuition at virtually all public and private research institutions in the past decade? Sophistry about bloated administrations or unappreciative legislatures (anyone but the faculty themselves) won't help move the discussion of faculty 'loyalty' along. I agree with Kennedy: too many faculty are more loyal to their 'guild' of fellow specialists than to their employer. Interestingly, as they age, and the hope or likelihood of a 'better offer' from another institution (often prompted by a fellow guild member) decreases, they tend to undergo a religious conversion to the hometown team. And in the era of uncapped retirement age, it appears they're sticking around at the expense of slots for younger colleagues.

It's hard to know what the cure for self-absorption is! I often paraphrase Henry Rosovsky, former long-time dean of Harvard College, and his *The University, An Owner's Manual* (Norton, 1990), by saying that I knew I'd been in academic administration long enough when, while watching *Amadeus*, I found myself rooting for Salieri!

FRANKLIN M. LOEW

Medical Foods Inc.

5 Cambridge Center, 8th Floor

Cambridge, Massachusetts 02142 USA

Zigmond replies—I found much to praise in my review of Kennedy's book, including the central premise—that faculty should devote more attention to the academic duty that accompanies their academic freedom. However, I also noted that Kennedy ignored many of the bases of the conditions he describes. As an example of the need for a deeper analysis, I commented on his criticism of faculty for attending to national and international professional responsibilities at the cost of their local responsibilities.

In his discussion of this issue, Kennedy failed to note that universities often pay only a fraction of their faculty's salaries. When I asked "should we make faculty salaries a larger portion of university budgets?" I was being rhetorical, for I realize that universities choose not to cover those salaries and are not likely to change their practice. This need not be seen as a criticism. By encouraging faculty to pay significant portions of their salaries, universities save money that then can be used for other needs, such as staff salaries, libraries and renovations. But in so doing, universities also must give up some of their claim on faculty attention.

Kennedy also should have acknowledged that most universities explicitly

reward faculty for their extra-institutional activities. Editorships in professional journals, service as officers in professional societies, participation in research review committees—these and other 'outside' activities, when accompanied by research and publication, are precisely what drive promotions, appointments and prizes. And this, too, need not be interpreted as a criticism. Scholarship thrives on the existence of a community of scholars that transcends geographical boundaries.

In sum, what Kennedy describes and Loew acknowledges is not so much a problem with the faculty as an emergent property of the university. And that property cannot easily be altered without structural changes in the way universities, as well as faculty, operate—changes that might well lead to less desirable outcomes than the ones that Kennedy decries. That is the reality of the academic planet that Kennedy, Loew and I share.

MICHAEL J. ZIGMOND

Department of Neurology

University of Pittsburgh

S-517 Biomedical Science Tower

Pittsburgh, PA 15213 USA

email: zigmond+@pitt.edu

GLI gene and rhabdomyosarcoma

To the editor—Your interesting News & Views item about the etiology of rhabdomyosarcoma¹ repeats a common misinterpretation of the cause and effect of gene defects in cancer development. The GLI gene was originally cloned because of its amplification in a glioma cell line² and was subsequently shown to have oncogenic potential. However, it has long since been shown that this gene is only occasionally coamplified with the CDK4 gene in sarcomas^{3,4} and gliomas⁵, and this amplification probably does not significantly contribute to the etiology of these tumors. By incorrectly citing the excellent paper characterizing *GLI* and its corresponding protein⁶, this misconception is perpetuated.

OLA MYKLEBOST

Department of Tumor Biology

Norwegian Radium Hospital

N-0310 OSLO, Norway; email: olam@radium.uio.no

- Zhan, S. & Helman, L.J. Glimpsing the cause of rhabdomyosarcoma. *Nature Med.* 4, 559–60 (1998).
- Kinzel, K.W., et al. Identification of an amplified, highly expressed gene in a human glioma. *Science* 236, 70–73 (1987).
- Berner, J.-M., et al. Separate amplified regions encompassing CDK4 and MDM2 in human sarcomas. *Genes Chromosomes & Cancer* 17, 254–259 (1996).
- Forus, A., et al. Mapping of amplification units in the q13-14 region of chromosome 12 in human sarcomas: Some amplicons do not include MDM2. *Cell Growth & Differentiation* 4, 1065–1070 (1993).
- Reifenberger, G., Reifenberger, J., Ichimura, K., Meltzer, P.S. & Collins, V.P. Amplification of multiple genes from chromosomal region 12q13-14 in human malignant gliomas: Preliminary mapping of the amplicons shows preferential involvement of CDK4, SAS, and MDM2. *Cancer Res.* 54, 4299–4303 (1994).
- Kinzel, K.W. & Vogelstein, B. The GLI gene encodes a nuclear protein which binds specific sequences in the human genome. *Mol. Cell Biol.* 10, 634–42 (1990).