breast cancer tissue microarray data, where only a minority of tumors overexpress the oncogene *HER2*, and where tumor aggressiveness is independent of HER2 protein levels[2]. The centrality of oncogenes is further called into question by data from Hanafusa[3], who highlights the ongoing failure to repeat Weinberg's oncogene-mediated transformation of normal diploid human cells. Furthermore, Klein and Riethmuller's[4] single cell analyses demonstrate that mutations in the *p53* tumor suppressor gene generally occur after aneuploidogenic changes, molecularly buttressing the pioneering work of Harris[5] on the contributions of aneuploidy to the etiology of tumor suppression. In the best-studied clinical case of cervical carcinoma, it is well known that aneuploidy precedes the development of cancer. Given this abundance of high-profile clinical and molecular literature, it is amusing that Gordon leaves "it to others" to refute the aneuploidy-cancer link.

Gordon's simplistic portrayal of AIDS as a single disease, however, is not amusing, but a cause for serious concern. AIDS is a clinical conglomerate of very different diseases, operationally defined by the US Centers for Disease Control as 26 necrogenic and neoplastic diseases, including Kaposi's sarcoma, dementia, pneumonia, chronic fevers, lymphoma, herpes, tuberculosis, diarrhea, lymphadenopathy and fungal candidiasis. Is Gordon seriously suggesting that HIV is the cause of dementia via immune collapse? The CDC classifies individuals with any of these diseases as AIDS if antibody to HIV is present. However, there is no detectable infectious HIV in most patients, only antibodies[6]. Thus, the 100% correlation between HIV and AIDS is not one of natural coincidence but of semantic contrivance. Furthermore, the mortality of HIV-antibody positive individuals treated with anti-HIV drugs is greater than that of mostly untreated HIV-antibody positive individuals, a disturbing finding in regard to current therapies[6]. For these and numerous other reasons Duesberg has carefully documented[6], Gordon's manifesto of HIV causality is unpersuasive, and his use of metaphors, such as "the tug of war between the virus and the immune system," reveals that he has nothing of substance to offer. Therapeutic progress in human disease requires solid clinical data, not scare mongering. It is the former that characterizes Bialy's remarkable book.

Even a cursory Pub Med search will reveal that my track record is in data evaluation in the academic, biotechnological and pharmaceutical areas with particular expertise in ultrasensitive technologies for characterizing complex human diseases via microarrays, bioinformatics and multiphoton detection platforms. Contributions to the human, *Drosophila* and mouse genome projects, functional proteomics and clinical medicine, segmental aneuploidy, and mutational and phenotypic analyses in eukaryotes are also easily found.

I do not resile from my glowing evaluation of Bialy's book, especially when confronted with impoverished rhetoric, *ad hominem* attacks, veiled accusations of genocide and the parroting of old and well-refuted arguments.

1. Zhang, L. *et al. Science* **276**,1268–1272 (1997).
2. Camp, R.L. *et al. Cancer Res.* **63**, 1445–1448 (2003).
3. Akagi, T. *et al. Proc. Natl. Acad. Sci. USA* **100**, 13567–13572 (2003).
4. Klein, C.A. *et al. The Lancet* **360**, 683–689 (2002).
5. Harris, H. *Proc. R. Soc. Lond. B.* **179**, 1–20 (1971).
6. Duesberg, P. *et al. J. Biosci.* **28**, 383–412 (2003).

Nature Biotechnology responds: The editors seek to publish book reviews of broad interest to the scientific community. The opinions expressed by the authors of these reviews do not necessarily reflect those of the journal. Given his long and distinguished publication record in genetics research and interest in epigenetics, George Miklos was deemed an appropriate reviewer to discuss the book and the impact of Duesberg's more recent research in aneuploidy, oncogenes and cancer.

Genomic islands in *Rhodopseudomonas palustris*

To the editor:

In their report of the complete genome sequence of the metabolically versatile photosynthetic bacterium *Rhodopseudomonas palustris* in the January issue, Larimer *et al.*[1] conclude that "no horizontally transferred islands of DNA are apparent based on anomalous G + C content." Using a newly developed method[2,3] that is more sensitive than the traditional window-based method for detecting GC content change, we have identified three horizontally transferred genomic islands. These genomic islands appear to encode functions that expand the environmental niches accessible to this bacterium.

Horizontal gene transfer has been recognized as a universal event throughout bacterial evolution[4]. Genomic islands contain clusters of horizontally transferred genes. It is believed that most genomic islands have many conserved features, such as an abrupt change in GC content compared with that of the rest of the genome, the presence of direct repeats flanking the genomic island, the presence of an integrase gene at the junction and use of tRNA genes as the integration sites[5]. Among the methods to detect genomic islands, assessing the change in GC content is well established. A routinely used method to assess the distribution of GC content is to count G and C residues within a window

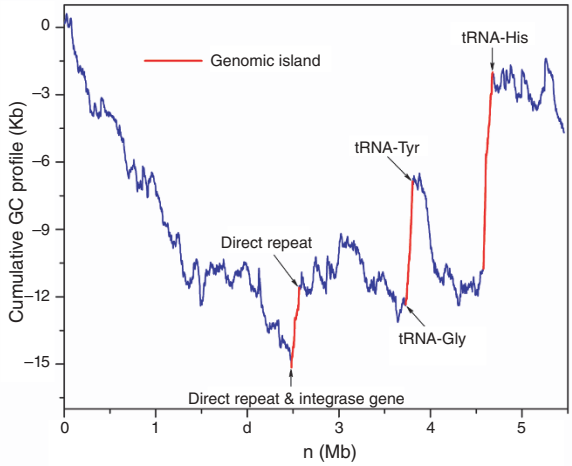

Figure 1 The cumulative GC profile for the *R. palustris* genome. Some characteristics of the cumulative GC profiles are: an up jump (drop) in the curve indicates an abrupt decrease (increase) of GC content; an approximately straight line indicates that the GC content is approximately constant within this region (see **Supplementary Methods** online for details). The cumulative GC profile for the *R. palustris* genome has three abrupt jumps in the curve, indicating that the GC content has an abrupt decrease in these regions. In addition, these three regions have many conserved features of genomic islands. For example, RPAGI-1 is flanked by two directly repeated sequences. An integrase gene immediately follows the repeat at the 5′ junction. RPAGI-2 is flanked by two t-RNA genes, whereas RPAGI-3 has a t-RNA gene at its 3′ junction. These features strongly suggest that the three regions are horizontally transferred genomic islands.