2.57 eV) and C_{70} (7.64 and 2.69 eV), respectively. This is consistent with the fact that La@C_{82} has low oxidation and reduction potentials relative to those of C_{60} and C_{70} (ref. 9). Even though the electron affinity of C_{82} (3.37 eV) is comparable to that of La@C_{82}, thermal addition of disilirane to C_{82} is suppressed owing to its larger ionization potential (6.96 eV)[8].

An exohedral derivative of an endohedral fullerene compound was reported previously by Saunders and co-workers[10], who allowed helium atoms to perfuse through the cage of a derivative of empty C_{60}. In contrast, the compounds reported here are made by attaching substituents to a preformed endohedral species. We anticipate that this approach may enable us to 'tune' chemically the degree of charge transfer in, and thus the properties of, the metallofullerenes.

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No UVB effect?

SIR — McMinn *et al.*[1] have examined the relative abundances of diatom taxa in three sediment cores, each taken from a different fjord in the Vestfold Hills region of eastern Antarctica. Over a 20-year period they noted little change in species composition based on relative abundances, and they thus concluded "that the enhanced ultraviolet-B levels resulting from the 'ozone hole' [have] had little effect on the diatom component of the phytoplankton community."

McMinn *et al.* acknowledge that their results probably apply only to coastal diatom communities largely shielded from ultraviolet-B radiation by thick ice cover. Unfortunately, the title of their letter, "Minimal effects of ultraviolet-B radiation on Antarctic diatoms over the past 20 years", implies something much broader for the Antarctic ecosystem. The data reported in their paper do not warrant this claim relative to Antarctic diatoms in general, or perhaps even for the diatom communities from the Vestford Hills region.

First, the diatom communities they have studied represent only a fraction of the diatom-based productivity of the Southern Ocean. It is the planktonic ice-edge and sea-ice communities of the more northern areas of the Southern Ocean that are the most productive during spring, are exposed to higher levels of ultraviolet-B, and are therefore more vulnerable to ozone depletion events. Second, the results are less compelling because the diatom counts were done on a relative rather than an absolute basis. For example, there could have been a 90% decrease (or increase) in the absolute abundance of diatom cells in Antarctic waters, without a change in the relative abundance of species. Further, large interannual variation in absolute diatom abundance could mask subtle but significant shifts in the relative proportions of the community if the counts were not statistically stratified.

Third, the potential effects of ultraviolet-B on diatom community species richness and diversity are problematic. The notion that environmental stresses such as ultraviolet-B will reduce species numbers and diversity is not necessarily correct. A long-term *in situ* study[2] showing that diatom community diversity increased under exposure to ultraviolet-B demonstrates the weakness of diversity as a measure of community response to this parameter. We believe that the absence of a decline in diatom species richness or diversity over the past 20 years in the data collected by McMinn *et al.* does not necessarily indicate that "increased UVB irradiances have had little effect on the planktonic diatom community."

Fourth, Smith *et al.*[3] showed that declines in primary productivity under the influence of elevated ultraviolet-B were less than the interannual variability of productivity. Unless there were distinct taxonomic patterns reflecting interannual variability evident in the McMinn *et al.* core data before the ozone depletion events initiated in the 1970s, there is no reason to suspect that core data would provide any insight into ultraviolet-B effects.

Last, the statement that levels of predation (grazing) are unlikely to have changed significantly over the past 20 years is not supported by any data (as acknowledged by McMinn *et al.*). Recent research in shallow, freshwater habitats[4] has indicated that grazing populations can be more susceptible to ultraviolet-B damage than primary producers and altered trophic-level interactions can produce greater effects on diatom communities than can direct effects of ultraviolet-B. It is now clear that changes in diatom numbers or species alone do not adequately address the issue of ecosystem change. The impact of ozone depletion on the Antarctic ecosystem is still very uncertain. Our concern is that the limited data provided by McMinn *et al.* do not substantiate the implication of the title of their paper.

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Diabetes genes — *mutatis mutandis*

SIR — In your recent editorial ("*Nature* Sister Publications", *Nature* **374**, 95; 1995), there were several errors in the description of our paper reporting a mutation predisposing to type 1, or insulin-dependent diabetes mellitus (IDDM).

Insulin-dependent diabetes mellitus is never abbreviated as "IDS", for example, nor is the HLA complex on chromosome 6 ever referred to as the "LA complex". The susceptibility locus on chromosome 11p15 is called *IDDM2*, and this locus, which we (*Nature Genetics* **9**, 284–292; 1995) identified as the variable number of tandem repeats locus within the transcriptional regulatory 5′ region of the insulin gene is never referred to as a "VENTURE". It is commonly referred to as a VNTR, and this correct abbreviation is used later in the editorial.

In our paper, we used families from the United Kingdom, United States and Denmark, but not from France. One of our interesting results was the finding that the 698-base-pair allele of the VNTR appears to encode different susceptibility from other VNTR alleles. This allele has 34 repeats, not 50 as claimed in the editorial. This is of key importance, as the number and DNA sequence of the repeats appears to be a critical factor for disease susceptibility and insulin gene expression (see also Kennedy *et al.*, *Nature Genetics* **9**, 293–298; 1995).

In the same issue of *Nature Genetics*, Hager *et al.* (**9**, 299–304; 1995) report that a mutation of the glucagon receptor gene is associated with type 2, or non-insulin-dependent, diabetes mellitus in