Assisting, But Not Dictating

WHEN READING A JOURNAL SUCH AS SCIENCE, one is easily seduced into believing that empirical evidence can resolve moral disputes. In his Letter “Human being redux” (16 Apr., p. 388), M. S. Gazzaniga defends human embryonic stem cell research because of the vast discrepancy between a tiny ball of cells that can fit on the head of a pin and a live human being. J. T. Durkin (“The case against stem cell research,” Letters, 3 Sept., p. 1402) minimizes this disparity by emphasizing that “[t]he embryo and the adult are different stages in the development of the human being.” By referring to empirical information, they seem to think that the right (good) social policy for stem cell research can be justified. G. E. Moore’s philosophical position, known as the naturalistic fallacy, argues that “goodness” is indefinable, and therefore its meaning cannot be logically derived by empirical means (1). That is, our biological underpinnings cannot prescribe what is good and right. However, facts in combination with a democratic ethic can assist in determining a policy decision. Although individuals will differ in their opinions, a democracy can decide whether the benefits of embryonic stem cell research outweigh any disadvantages. Science can assist in making this decision, but cannot dictate it (2).

HOWARD H. KENDLER
Department of Psychology, University of California, Santa Barbara, Santa Barbara, CA 93106, USA.

References

Microbial Life in the Atacama Desert

IN THEIR REPORT “MARS-LIKE SOILS IN THE Atacama Desert, Chile, and the dry limit of microbial life,” R. Navarro-Gonzáles et al. found only very low levels of culturable bacteria in the Mars-like soils of the Atacama Desert, and they did not recover DNA (Reports, 7 Nov. 2003, p. 1018). In contrast, we have found easily cultured, low numbers of bacteria and recoverable bacterial DNA from soils in the extreme arid core of the Atacama Desert in northern Chile. Soil samples taken from a 4500-m elevational transect just south of the Tropic of Capricorn (−24°S) all yielded culturable bacteria on R2A agar (1, 2), including samples from elevations of absolute desert that have not harbored plant life for a million years or more. Four of our samples were taken in the vicinity of the dry Yungay region, in close proximity to those studied by Navarro-Gonzáles et al. (elevation ~1000 m: S 24°4.16’, W 69°51.98’ and S 24°4.18’, W 69°51.96’). Our three closest sites (987 m: S 24°4.16’, W 69°51.98’ and S 24°4.18’, W 69°51.98’), W 70°12.55’; 1315 m: S 24°21.787’, W 69°56.757’; and 1931 m: S 24°28.135’, W 69°24.472’) yielded counts of 1.3 × 10^5, 5.4 × 10^4, and 9.1 × 10^4 CFU/g of dry soil, respectively. A fourth site (703 m: S 23°57.417’, W 70°17.157’) yielded only 1 or 2 colonies per plate, which is a value too close to the detection limit of the spread plating method to quantify accurately but is still higher than that reported by Navarro-Gonzáles et al. (<10 colonies found on 100 plates).

Bacterial DNA was successfully extracted (3) from all of our samples (Navarro-Gonzáles et al. report no recovery of DNA from the Yungay samples), and 16S rRNA genes were amplified (4, 5) and profiled by denaturing gradient gel electrophoresis (DGGE). Statistical analysis of DGGE profiles demonstrates a similar bacterial community structure in samples taken from soil profiles in the absolute desert portions of our Atacama transect. This community structure is quite different from that found in profiles from vegetated zones supported by fog or precipitation below (<500 m) and above (>2500 m) the absolute desert, respectively. Our results demonstrate the existence of life in one of the driest regions on Earth. We may have been able to demonstrate life because we sampled at a depth of 20 to 30 cm, in comparison to Navarro-Gonzáles et al., who sampled the upper 10 cm of the soil. This only emphasizes the critical nature of the sampling protocol used in any extreme environment on Earth and particularly on Mars.

R. M. MAIER, J. K. DREES, J. W. NEILSON
1Department of Soil, Water and Environmental Science, 2Department of Animal Sciences, Division of Epidemiology/Biostatistics, 3Department of Geosciences, University of Arizona, Tucson, AZ 85721, USA.

D. A. HENDERSON, J. QUADE, L. BETANCOURT
1Department of Civil Engineering, University of Minnesota, Minneapolis, MN 55455, USA. 2U.S. Geological Survey, 1675 West Anklam Road, Tucson, AZ 85745, USA.

References
3. Fast DNA Spin Kit for Soil, Qbiogene, Carlsbad, CA.

Response
IN OUR PAPER, WE REPORTED EXTREMELY LOW levels of culturable organisms and no recoverable DNA in the surface soils of the extreme arid core of the Atacama Desert near the abandoned town of Yungay. We could not claim that there was no life in these soils on the basis of our results, and therefore we presented our data as indicating an upper limit of 100 culturable heterotrophic bacteria per gram of soil (see fig. 2E of our Report) for surface materials. This upper limit is orders of magnitude less than the concentrations of bacteria found in soils south of this Mars-like region of the Atacama. In more recent published work (1), we have reported that below the surface, there are discrete layers with higher numbers of culturable bacteria. For example, at a Yungay site, we have found negligible levels of bacteria at the surface (<100 CFU/g) but recovered less than 1 × 10^6 to 2.96 × 10^7 CFU/g of soil in subsurface layers (1). In addition, we have conducted an extensive survey of surface and subsurface soils in the arid core of the Atacama (1–4). The data presented by Maier et al. for subsurface samples are consistent with our published work [our Report; (1–4)] and do not necessi-
tate any reassessment or reevaluation of the conclusions of our Report. We agree with their conclusion regarding the critical nature of the sampling protocol used in any extreme environment on Earth and Mars.

RAFAEL NAVARRO-GONZÁLEZ, FRED A. RAINEY, CHRISTOPHER P. MCKAY
1Laboratorio de Química de Plásmas y Estudios...
Letters

Varshavsky's Contributions

We are writing to express our enthusiasm that the discovery of the ubiquitin conjugation system has been acknowledged with the award of the Nobel Prize in Chemistry to three outstanding biochemists: Avram Hershko and Aaron Ciechanover of the Technion Institute in Israel, and Irwin Rose of the University of California at Irvine (“Gold medal from cellular trash,” G. Vogel, News Focus, 15 Oct., p. 400). Unraveling the chemistry that underlies the attachment of ubiquitin to proteins that are destined to be degraded was a magnificent achievement and is fully deserving of this recognition.

The mechanism of ubiquitin conjugation and its role in proteolysis was selected for recognition by the Nobel Committee in large part because of the vital role that the ubiquitin-proteasome system (UPS) plays in the physiology of cells and organisms. Investigations on the physiological functions of the UPS, which dominate current research in this field, were pioneered largely by Alexander Varshavsky of the California Institute of Technology. Several core principles that guide our current understanding of the ubiquitin system had their origins in Varshavsky’s work, including the following: (i) the UPS is the predominant mechanism for selective protein turnover in the cytoplasm and is essential for cellular function; (ii) in addition to its role in turning over damaged proteins, the UPS controls diverse physiological processes such as the cell cycle, DNA repair, and stress responses; (iii) ubiquitin ligases (E3s) are highly specific receptors that underlie the remarkable specificity of ubiquitination by binding to defined sequences within proteins (degrons); and (iv) a ubiquitin chain linked via the lysine-48 residue of ubiquitin governs targeting of substrates to the proteasome for degradation. In addition to these fundamental contributions, Varshavsky ushered the ubiquitin field into the age of molecular genetics by identifying mutants and characterizing

References

“...We suggest that the impact of Varshavsky’s work on the physiology of the ubiquitin system and its relationship to fundamental processes such as mitosis and chromosome segregation justifies serious consideration for a future Nobel Prize in Physiology or Medicine.”

—Baumeister et al.

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the genes that define ubiquitin and the E1, E2, and E3 components of the conjugation cascade. In our opinion, the appreciation of the significance of the UPS owes more to Varshavsky’s work than to that of any other individual.

We suggest that the impact of Varshavsky’s work on the physiology of the ubiquitin system and its relationship to fundamental processes such as mitosis and chromosome segregation justifies serious consideration for a future Nobel Prize in Physiology or Medicine. As we extend our heartfelt congratulations to the winners of this year’s Nobel Prize in Chemistry, we wish to make it clear that Varshavsky’s contributions are also deeply respected by his colleagues.

Stefan Bengtson, Graham Budd

The report by Chen et al. of coelomate bilaterian fossils from ancient phosphorites (Research Articles, 9 July 2004, p. 218) is not well founded. The morphological features reported can be simply accounted for by familiar taphonomic and diagenetic processes. The structures may well be eukaryotic microfossils, but their present appearance has little resemblance to the once-living organisms.

Full text at www.sciencemag.org/cgi/content/full/306/5700/1291a

Jun-Yuan Chen, Paola Oliveri, Eric Davidson, David J. Bottjer

The premise presented by Bengtson and Budd is incorrect, and their example is irrelevant. We provide two new images of the holotype specimen that demonstrate that the definitive characters of the specimen discussed by us in the original report are even more extensively evident than was initially apparent.

Full text at www.sciencemag.org/cgi/content/full/306/5700/1291b
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Konstanz, 78457 Konstanz, Germany. Max Delbrueck Centre for Biomedical Research, Berlin 13122, Germany. Cold Spring Harbor Laboratory, Cold Spring Harbor, NY 11724, USA. Samuel Lunenfeld Research Institute, Toronto, ON M5G 1X5, Canada. University of Wisconsin, Madison, WI 53706, USA. National Cancer Institute, National Institutes of Health, Frederick, MD 21702, USA. Emory University, Atlanta, GA 30322, USA. University of Stuttgart, D-70569 Stuttgart, Germany.

CORRECTIONS AND CLARIFICATIONS

Reports: “Mitotic Golgi partitioning is driven by the membrane-fissioning protein CtBP3/BARS” by C. Hidalgo Carcedo et al. (2 July, p. 93). This paper reported that BARS is crucially involved in mitotic Golgi partitioning and entry into mitosis. CtBP3/BARS (BARS) is a protein involved in Golgi membrane fission [S. Spanò et al., J. Biol. Chem. 274, 17705 (1999); R. Weigel et al., Nature 402, 429 (1999)] and a member of the CtBP family that comprises CtBP1 and CtBP2, both of which are transcriptional co-repressors [G. Chinnadurai, Mol. Cell. 9, 213 (2002); G. Chinnadurai, Bioessays 25, 9 (2003)]. BARS is almost certainly a Ctbp1 gene product and therefore a splice variant of Ctbp1. A KO mouse has been generated in which both ctbp1 and ctbp2 have been deleted (and therefore also lacks BARS). This KO is embryonically lethal, but cells derived from these embryos proliferate normally, indicating that the partitioning of their Golgi complex should occur during mitosis (although it is not clear that the Golgi partitions normally) [J. D. Hildebrand, P. Soriano, Mol. Cell. Biol. 22, 5296 (2002)]. This is apparently discrepant with the report by Hidalgo Carcedo et al. that BARS is crucially involved in mitotic Golgi partitioning and entry into mitosis. Similar discrepancies between KO and classical cell biological studies in cultured cells are frequent and provide useful insights into the process under study [M. Pagano, P. K. Jackson, Cell 118, 535 (2004)]. The following is a brief discussion of a few hypotheses that can cast light on this specific case. First, more than one fission mechanism might be involved in mitotic Golgi partitioning. For instance, mitotic Golgi fragmentation involves two stages, one consisting of the consumption of Golgi membranes by the irreversible budding of COPI vesicles, and the second, of the tubulation of Golgi cisternae followed by their cleavage into smaller pieces [J. Shorter, G. Warren, Annu. Rev. Cell. Dev. Biol. 18, 379 (2002)]. The latter component is likely to be the one that is dependent on BARS. It is possible that in embryonic cells lacking BARS, the COPI-dependent mechanism might carry the Golgi partitioning process far enough to allow mitosis to proceed. Another possibility is that in these cells, once the Golgi cisternae have been transformed into tubules during mitosis (presumably via phosphorylation of the relevant golgins) [Shorter and Warren], a dynamin-like protein is able to cleave these tubes into small pieces. Second, BARS might not be a core fission protein, but rather a regulator, which could be replaced in BARS-null cells by a related gene with a similar function. Finally, it is possible that the Golgi structure in embryonic cells is organized differently and does not require the BARS-controlled machinery to enter mitosis. This last possibility is supported by morphological studies that are presently in progress. The above mechanisms (and possibly others) might allow the cells to undergo mitosis and execute Golgi partitioning even in the absence of BARS. This could result in mitotic Golgi phenotypes that might or might not be different from those in control cells.