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Finding Evidence for Black Holes

I READ G. C. BOWER ET AL'S RESEARCH Article "Detection of the intrinsic size of Sagittarius A* through closure amplitude imaging" (30 Apr., p. 704; published online 1 Apr.; 10.1126/science.1094023) with alarm. Like many before them, the authors are presuming too much about black holes, considering the present state of knowledge. Black holes are popular topics of conversation, beloved of science fiction writers, but, as yet, not one has been identified beyond all reasonable doubt. Indeed, although there is strong evidence for the existence of black holes, it is not compelling because there is no proof yet that any of the candidates possesses that defining property of a black hole, an event horizon.

Various claims have been made for black hole candidates (1, 2), but none stand up to one simple test. From general relativity, it follows that for a black hole, the ratio of mass to radius of the event horizon must satisfy $M/R \ge 6.7 \times 10^{26}$ kg/m.

In neither of these cases is this inequality satisfied or the existence of an event horizon even considered. Again, in the case of Bower *et al.*, the data provided do not lead to this inequality being satisfied. Interestingly, this quoted relation is precisely the expression for the ratio of mass to radius that Michell derived in 1784 (3) for a body possessing an escape speed greater than, or equal to, that of light.

A possible alternative explanation for the above observations could be the presence of quark or even subquark stars (4, 5) clustered near the center of our galaxy. Such an explanation gains some credence from simple order of magnitude calculations. Alternatively, the central mass could be composed of a mixture of baryonic and dark matter that could involve a number of normal stars of roughly solar mass, contained within a distributed source of gravitation able to constrain the mixture within a stable limited volume forming the galactic center. It is too early to rule out completely other explanations for relatively recent observations. If black holes do exist and there is one at the center of our galaxy, care must be taken not to claim proof of its existence until its presence is established beyond all reasonable doubt. That point has not been reached yet.

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Response

DUNNING-DAVIES'S ALARM IS MISPLACED. WE have certainly not claimed proof of the existence of black holes on the basis of our research. In fact, our research is motivated in part by the desire to find the strongest possible evidence of the existence of black holes. We are well aware that current efforts fall short of excluding all possible alternatives to the black hole hypothesis for Sagittarius A*. Demonstration of the black hole mass-radius relation would be compelling evidence, for instance. Our recent limit on the size of the radio-emitting region of Sgr A* combined with astrometric measurements showing that Sgr A* is virtually motionless with respect to the Galaxy (1) provide the tightest constraint yet on the mass density of a black hole system. Yet this limit is about five orders of magnitude less than the canonical black hole mass density. Future imaging and astrometric experiments will narrow this gap substantially in the coming decades. Nevertheless, the current density limit is sufficient to eliminate all existing alternative models on the grounds that clusters of particles or compact objects such as strange stars would evaporate on time scales much, much less than the age of the Galaxy (2).

Evidence for or against black holes, of course, can be obtained on the basis of studying the numerous other properties determined by the space-time metric in their vicinity (3). Ultimately, we hope to achieve a resolution of only a few Schwarzschild radii through submillimeter very long baseline interferometry. With such an experiment, we expect to see the effects of the black hole's mass and spin on radiation emitted at small radii (4).

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Letters to the Editor

Letters (~300 words) discuss material published in *Science* in the previous 6 months or issues of general interest. They can be submitted through the Web (www.submit2science.org) or by regular mail (1200 New York Ave., NW, Washington, DC 20005, USA). Letters are not acknowledged upon receipt, nor are authors generally consulted before publication. Whether published in full or in part, letters are subject to editing for clarity and space.

Extending Life-Span in *C. elegans*

THE LIFE-SPAN OF THE NEMATODE *CAENORhabditis elegans* can be extended by at least six different mechanisms, including calorie restriction, reduced Ins/IGF-1 signaling, germline ablation, food sensing amphid ablation, mitochondrial deficiency, and decreased temperature. Reduced Ins/IGF-1 signaling and calorie restriction can also increase the life-span of flies and mice. The Brevia "Healthy animals with extreme longevity" by N. Arantes-Oliveira *et al.* (24 Oct. 2003, p. 611) showed that *daf-2* RNAi treatment and gonad ablation of worms carrying the *daf-2(e1368)* hypomor-

phic mutation in the gene encoding the *C.* elegans Ins/IGF-1 receptor increases their life-span 6.0-fold. We have

⁶ found that the average life-span of daf-2(e1370) mutants grown in axenic medium [a sterile liquid medium based on yeast extract, soy peptone, and hemoglobin; see (1)] was 90.9 days, representing a 6.3-fold life extension and a 7.5-fold adult life-span extension relative to wild-type controls grown on plate cultures seeded with live *E. coli* cells (1).

Arantes-Oliveira et al. also note the health of their long-lived worms. We observed that worms grown in axenic medium appear more vigorous than their monoxenically grown counterparts and that these worms exhibit an increase in metabolic rate (2), counter to the idea that a reduction of the metabolic rate is associated with a longer life-span. Moreover, both caloric restriction and reduced Ins/IGF-1 signaling increase the resistance to heat and oxidative stressors (1), and calorically restricted mice are less prone to age-related diseases. Thus, the life of worms can be extended without diminishing health. These results might be important for human aging as well, because both caloric restriction and cell signaling have been shown to regulate the aging rate in organisms ranging from yeast to mammals.

CREDIT: PRESTON HUEY/S

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Disagreements Over Cloud Absorption

IN THEIR LETTER "HAVE CLOUDS DARKENED since 1995?" (14 Nov. 2003, p. 1151), Z. Li and colleagues discuss points raised in an earlier news article on climate models and clouds ("Making clouds darker sharpens cloudy climate models," R. A. Kerr, News of the Week, 20 June 2003, p. 1859). Li *et al.* state that "Agreements [between model calculations and observations] within the range of uncertainties were found by all teams... except for one...," where a paper of ours is cited (1) as the exception and no references are given for "all teams." This statement is misleading and inaccurate. Cloud absorptances were calculated in (1)with a suite of five different radiative transfer models, and, contrary to Li et al.'s Letter, agreement within the uncertainties was indeed found for most models. Figures 11 and 14 and Table 3 in (1) show observed and modeled absorptances and the overlap of error bars. For example, in the 29 March case [the most favorable case for measurements and analysis (2)], the differences are 20 to 23 W m⁻² for three models and 61 W m^{-2} for the two other models (1). Other ARESE II studies find measurementcalculation differences of 18 to 35 W m⁻² (3) and 15 to 28 W m⁻² (4). Hence, the results in (1), (3), and (4) are in general agreement (given model and measurement errors and variations in model implementation between the various studies) for the higher performance models. Very importantly, however, all the studies find systematic model-observation discrepancies.

In our view, the true disagreement in the few cases studied is on the interpretation of

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the model-measurement differences. Li *et al.* and Ackerman *et al.* (4) appear to conclude that cloud absorptance can be calculated adequately, whereas Valero *et al.* (1) and O'Hirok and Gautier (3) conclude that model-measurement differences, even if within error bars, are important because of their systematic character; models consistently underpredict and never overpredict the value. The source (experimental or modeling) of such a bias is of major concern because these results are fundamental for both climate and remote sensing applications.

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Response

THE PURPOSE OF OUR LETTER WAS TO REFUTE a misimpression left by a news article by Richard A. Kerr: that atmospheric radiation models have become a lot more absorbing as a result of the claim of enhanced cloud absorption in 1995. In fact, the best models are not much more absorbing now than in 1995, and their extra absorption is due to gases and aerosols and a better treatment of surface albedo, not clouds. What is true about models is that many climate-model radiation packages were too transparent (1); this was brought to the community's attention by a few studies comparing modeled and observed solar energy disposition (2-4) that were published in 1995, independently of the enhanced cloud absorption controversy.

Our discussion of Valero's work was a side issue not directly related to this main point about whether models have really changed radically or not and the main factors driving the changes. The conclusion of his study seems to be rather mixed. If we misinterpreted his results, we apologize. We are not denying that there may still be a bias between models and measurements, nor are we denying the reality of disagreements that existed in 1990 as summarized by (5). We are merely saying that the general increases in atmospheric absorption in Global Climate Models since 1995 have been attributed much more to the treatment of clear-sky solar radiative transfer processes than to the cloud absorption. In spite of the substantial progress in observational technology since 1995, spurred by the controversy, we are still not at the point where the bias can be unambiguously separated from possible measurement error. More field campaigns with even better technology are necessary to nail down the remaining much smaller bias.

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CORRECTIONS AND CLARIFICATIONS

News of the Week: "Lab fails to reproduce protein's appetite-suppressing effects" by T. Gura (9 July, p. 158). The article stated that "leptin failed as a drug." Amgen of Thousand Oaks, California, which has an exclusive license from Rockefeller University to develop leptin, reports that it has discontinued commercial studies of leptin for obesity, but is supporting research on its possible use in therapy for general lipodystrophy.

News of the Week: "Report accuses Bush Administration, again, of 'politicizing' science" by A. Lawler and J. Kaiser (16 July, p. 323). The article incorrectly characterized a statement by Janet Rowley regarding her White House interview before being appointed to the President's Council on Bioethics. Rowley did not contact the council chair, Leon Kass, after being questioned about her support for President Bush and his policies.

Reports: "Role of NMDA receptor subtypes in governing the direction of hippocampal synaptic plasticity" by L. Liu *et al.* (14 May, p. 1021). The legend for Fig. 1C, which reads "HFS failed to produce LTP in the presence of NR2B antagonists," is incorrect. It should read "NR2B antagonists failed to block HFS-induced LTP."

