which are designed to pass only radially polarized light, appear dark. The contrast between this behavior and that of the comet is seen clearly in Fig. 1b, where the bright southwest streamer is almost completely cut off by the analyzer ring at 5 $R_\odot$, while the comet tail is nearly unaffected.

In some of the enhanced frames of Fig. 2, a very slight effect of the analyzer may be barely perceptible at the more diffuse (northerly) edge of the tail. This is expected because material separated from the cometary nucleus by solar heating undergoes selective acceleration (*17*), producing a distribution spectrum of particle sizes. As the released material is driven farther from the cometary orbit, the separation according to grain size becomes more pronounced. Large grains, 1 to 10 μm or more in diameter, remain for some time near the original orbit path. They produce, in the aggregate, an appearance similar to an aircraft's "contrail" in the upper atmosphere, although individual particles continue to orbit with velocities close to that of the comet's head. Smaller particles are driven away from the original parabola more rapidly than the large ones. In this cosmic winnowing process, when a substantial fraction of the grains in a certain area are much smaller than the wavelength of the incident photospheric light ($<< 0.5$ μm), the scattering process will best be described by Rayleigh theory (*20*) and polarization at right angles to the illuminating rays should be evident. No net polarization is expected for the larger particles.

Conclusion. We have reported the observation of a new comet, apparently the first to be discovered from a spacecraft and the first observed to collide with the sun. Preliminary analysis has provided a set of orbital elements of modest accuracy and suggests that the comet may be one of the Kreutz sungrazers. Disintegration products from the encounter caused a major change in the coronal brightness distribution, which persisted for more than one full day. Indications of the encounter on the solar surface have not yet been identified but are a distinct possibility. We invite other researchers to communicate to us any corroborative observations of either solar surface or coronal manifestations of this unusual observation.

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10. A complete description of the polarization analyzer system is given by Koomen *et al.* (*7*).
11. *American Ephemeris and Nautical Almanac—1979* (Government Printing Office, Washington, D.C., 1977), pp. 231 and 336.
12. The transformation is more precisely defined as follows. Suppose that the parabola lies in the xy plane and that the parabolic axis and the y axis coincide. Then consecutive rotations R_α (about y), R_β (about z'), and R_γ (about x'') lead to a rectangular coordinate system $x''y''z''$ in which the x'' axis is the line of sight and the z'' and y'' axes are the directions of solar north and west, respectively, projected onto the plane of the sky.
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17. Because the radiative flux density and the gravitational force both vary as the inverse square of heliocentric distance, the ratio of the gravitational attraction to the repulsive radiation force is a constant, depending on the particle's size, density, and optical properties. Values typical for the interplanetary dust, which we here assume are applicable to the cometary dust, are given by S. F. Singer, in *Solar Physics*, J. M. Xanthakis, Ed. (Interscience, New York, 1967), p. 485.
18. From projection geometry alone we cannot distinguish whether the widely dispersed cometary material is in or out of the orbital plane, or both. If in the plane, a 6-$R_\odot$ projection in the plane of the sky represents cometary material between 16 and 43 $R_\odot$ distant from the sun, where it would presumably have a very low brightness, as both particle density and radiative flux density are expected to fall off as r^{-2}. Photometric and other studies may help to define the distribution.
19. In addition to heating, the atmospheric drag reduces an incoming body's velocity, dropping it to lower orbits, so that the atmospheric entry process is a self-accelerating one. For a homely comparison, densities in the solar chromosphere are on the order of 10^{-11} g/cm^3, comparable to regions in the earth's ionosphere, at 75 to 100 km, at which altitudes meteoroids are routinely observed by their glowing trails against the nighttime sky.
20. For example, see M. Born and E. Wolf, *Principles of Optics* (Pergamon, New York, 1959), chap. 13.
21. We wish to acknowledge a debt of gratitude to B. Marsden, F. Whipple, and E. Roemer for their insights and helpful discussions. In the experimental area, many persons have contributed to the successful development and long orbital lifetime of the SOLWIND coronagraph. We gratefully acknowledge the dedication with which D. Roberts, F. Harlow, R. Chaimson, R. Seal, W. Funk, D. Conroy, R. Perlut and R. Davies assisted in design, preparation, and test of the instrument. We are indebted to the Space Test Program, administered by the U.S. Air Force for the Department of Defense, for P78-1 spacecraft integration, launch, and operational support; to the Air Force Satellite Control Facility MCC-F team for patience and perseverance in the on-orbit operations that produced these observations; and to the NASA Office of Space Science for contribution of extensive hardware inventories that made possible development of the necessary solar pointing control. Finally, we thank R. Gilbert of the Ball Aerospace Division and his engineering team for outstanding performance in design and implementation of the spacecraft systems and interfaces.

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Development of a Monoclonal Antibody Against a Tumor-Associated Antigen

Abstract. *A monoclonal antibody-producing hybrid cell line was obtained by fusing mouse myeloma cells with spleen cells from a mouse immunized with C6 glioma cells. This antibody binds to a specific cell-surface antigen that is present on C6 rat glioma cells, transformed astrocytes and oligodendrocytes, and a human glioma cell line but is absent on a normal glial cell line, fibroblasts, and primary cultures of astrocytes and oligodendrocytes. The antigen also appears on tumor tissue of transformed oligodendrocytes but not on normal brain tissue.*

The nature of neoplastic transformation has been the object of numerous investigations in a variety of tissues (*1*), but the literature on the study of brain neoplasms is scant. The potential targets of neoplastic transformation in the brain are its three main cell types, the neurons and the principal glial cells, namely, the oligodendrocytes and the astrocytes. A major obstacle to the detailed study of tumor induction in brain is the overwhelming complexity of the intricate interactions among these various cell types that greatly hinders experimental design and data interpretation. This problem was overcome by the development in our laboratory of a novel technique [McCarthy and de Vellis (*2*)] to grow separate cultures of oligodendrocytes and astrocytes. Use of the homogeneous cell populations has greatly facilitated investigations into the changes that occur specifically during glial transformation. Recently, Bressler *et al.* (*3*) demonstrated that both astrocytes and oligodendrocytes can undergo spontaneous or ethylnitrosourea (ENU)-induced transformation. We now report the development of a monoclonal antibody that binds specifically to the surface of transformed glial

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Table 1. Specificity of antibody 217c against cells in culture. All target cells were cultured in wells of Linbro multiwell plates in a medium (0.1 ml) containing 0.2 μCi of [^{125}I]iododeoxyuridine. After 24 to 48 hours, plates were washed with phosphate-buffered saline (PBS). Antibody 217c was added to the cells at different concentrations. Myeloma S194–conditioned medium containing 1.32 μg of mouse IgG per milliliter was used as a control. After a 1-hour incubation period at 37°C, plates were washed with PBS buffer and ^{131}I-labeled staphylococcal protein A (approximately 200,000 count/min) was added to each well. Plates were washed again at the end of a 1-hour incubation at 37°C and then dried. Each plate was band-sawed into individual wells, and each well was counted for both [^{125}I]iododeoxyuridine and the ^{131}I-labeled protein A. The amount of ^{131}I-labeled protein A bound was expressed as counts per minute per 2×10^4 cells. The data are reported as total binding (counts per minute) per 2×10^4 cells (± standard error of the mean) and as binding ratios to the S194 myeloma–conditioned medium containing 1.32 μg of mouse IgG per milliliter.

Cells in culture	S194-conditioned medium (plus 1.32 μg of mouse IgG per milliliter)		Antibody 217c (1.32 μg per milliliter of culture medium)		Antibody 217c (1:100 dilution)	
	Counts per minute (± S.E.M.)	Binding ratio	Counts per minute (± S.E.M.)	Binding ratio	Counts per minute (± S.E.M.)	Binding ratio
Rat glioma C6	248 ± 17.6	1.00	629 ± 48.5	2.54	478 ± 24.8	1.93
ENU-induced transformed rat oligodendrocytes (TOP-ET-1)	382 ± 21.4	1.00	1947 ± 192.0	5.09	2524 ± 145.5	6.61
Spontaneously transformed rat astrocytes (BD-ST-1)	346 ± 51.7	1.00	1290 ± 70.5	3.72	1002 ± 53.0	2.89
Human glial tumor cells	1262 ± 101.9	1.00	6606 ± 133.2	5.23	6215 ± 664.9	4.92
Primary culture of astrocytes from newborn rat	472 ± 57.8	1.00	642 ± 67.1	1.36*	622 ± 11.4	1.31*
Primary culture of oligodendrocytes from newborn rat	270 ± 11.5	1.00	320 ± 17.3	1.18*	240 ± 10.0	0.89*
Normal rat glial cell line (BD-N-2)	315 ± 24.5	1.00	439 ± 16.9	1.39*	339 ± 4.67	1.07*
Normal rat skin fibroblast cell line	269 ± 12.1	1.00	294 ± 36.1	1.09*	306 ± 43.0	1.14*
Primary rat hepatocytes culture	1213 ± 26.0	1.00	2039 ± 77.4	1.68	1127 ± 77.7	0.93*
Rat Reuber hepatoma (H-4-II-E-C3)	250 ± 34.6	1.00	652 ± 25.3	2.61	385 ± 54.5	1.54*
Mouse hepatoma (HEPA 1)	843 ± 37.3	1.00	1737 ± 48.3	2.06	959 ± 70.9	1.13*

*Not significant; t-test, $P > .01$.

cells and to tumor tissue of transformed oligodendrocytes but not to the surface of normal glial cells.

To generate the monoclonal antibody against glioma cells, BALB/c female mice were injected intraperitoneally with 1×10^7 C6 glioma cells, followed by another injection 14 days later. Three days after the second immunization spleen cells were fused with S194/5.XXO.BU.1 mouse myeloma cells at a ratio of 4 to 1 in the presence of 40 percent polyethylene glycol. Hybrid cells were selected with HAT (hypoxanthine, aminopterin, and thymidine) medium and cloned by the limited dilution method (*4*). The presence of immunoglobulin against glioma C6 cells was detected by the direct binding method with the use of ^{125}I-labeled protein A from *Staphylococcus aureus* (*5*).

The hybrid cell line 217c, which was selected, has remained stable for 1 year. Fluorescence microscopy, utilizing fluorescein isothiocyanate–conjugated rabbit antiserum against mouse immunoglobulin G (IgG), indicated that this monoclonal antibody binds to live C6 glioma cells and that the antibody is in the IgG class. The concentration of 217c antibody secreted into the culture medium was determined by solid phase radioimmunoassay and was found to be 1.32 μg per milliliter (*6*).

The specificity of the 217c antibody was studied by two assay systems: the ^{131}I-labeled staphylococcal protein A binding assay (*5*, *7*) and the direct tissue binding assay (*8*). The ^{131}I-labeled protein A binding assay allows us to control for variations in cell number by using [^{125}I]iododeoxyuridine. Results (Table 1) indicated that undiluted 217c antibody, or antibody at a 1:100 dilution, recognizes C6 rat glioma cells, ENU-induced transformed oligodendrocytes (TOP-ET-1), and spontaneously transformed astrocytes (BD-ST-1). It also binds to a human glioma cell line. On the other hand, it does not bind to normal rat cell lines of skin fibroblasts and glial cells (BD-N-2) or to primary cultures of rat oligodendrocytes or astrocytes. Weak binding by the undiluted antibody occurred on two rodent hepatoma cell lines, a rat Reuber

Table 2. Direct binding of antibody 217c to rat tissues. A sample of 200 mg of liver, brain, thymus, spleen, or tumor tissue was collected from 30- to 50-day-old Wistar rats, washed three times, and homogenized in PBS buffer containing 0.42 percent trisodium citrate. These homogenates were incubated with undiluted 217c antibody for 60 minutes at room temperature, using S194-conditioned medium containing 1.32 μg of mouse IgG per milliliter as control. Unbound IgG was removed by washing three times with PBS buffer. Protein A labeled with ^{125}I (200,000 count/min) was added to detect the bound IgG. At the end of a 1-hour incubation, the homogenates were washed free from unbound ^{125}I-labeled protein A and counted in a gamma counter. Values are given ± standard error of the mean.

Tissues	S194-conditioned medium (plus 1.32 μg mouse IgG per milliliter) (counts per minute per microgram of protein)	Antibody 217c (1.32 μg/ml) (counts per minute per microgram of protein)	Binding ratio
Brain	177 ± 9.5	182 ± 10.3	1.02
Liver	355 ± 42.8	1006 ± 103.8	2.83
Thymus	128 ± 9.5	138 ± 22.5	1.08
Spleen	345 ± 10.0	446 ± 36.0	1.29
TOP-ET-1 tumor (transformed rat oligodendrocytes)	248 ± 10.1	1158 ± 88.5	4.67
Rat C6 glioma (cell line pellet)	181 ± 29.5	871 ± 116.5	4.81

hepatoma (H-4-II-E-C3) and a mouse hepatoma (HEPA 1), as well as on primary rat hepatocyte cultures. At a 1:100 dilution of the 217c antibody, this binding was insignificant. Direct tissue binding experiments (Table 2) demonstrated that the 217c antibody binds to a C6 glioma cell pellet, as well as to transformed oligodendrocyte (TOP-ET-1) tumor homogenates, but does not bind to normal rat brain, thymus, or spleen. Some binding to liver tissue was observed, but the nature of the binding sites needs further study.

We selected a monoclonal antibody that demonstrates the existence of a cell surface antigen on transformed glial cells and glial tumor homogenates and its absence on nontransformed cells or tissue homogenates from most rat organs. Various investigators have previously referred to brain tumor–specific antigens (*9*), but these studies employed heterospecific antiserums that contain contaminating antibodies. The development of monoclonal antibody methodology (*10*) has permitted investigators to ascertain with greater specificity the existence of tumor-specific antigens on various neoplastic tissues (*11, 12*).

A monoclonal antibody against human glioma cell lines has been described recently (*13*), but these investigators did not demonstrate the presence of the antigen on tumor tissue. We have shown that this antigen is absent on the normal glial cell line but is expressed after these cells undergo neoplastic transformation. Therefore, the importance of a specific cell surface antigen during transformation can now be explained. This antibody also may have immunotherapeutic applications.

For example, Herlyn *et al.* (*12*) demonstrated the inhibition of colorectal carcinoma growth in nude mice by a monoclonal antibody against colorectal carcinoma cells. In addition, a new technique of conjugating diphtheria toxin A chain or ricin A chain to a specific antibody has been reported. The antibody will direct the action of A chain to its specific target cell and thus the conjugate becomes a very potent cell type–specific toxin (*14*). If conjugated ricin A–217c antibody can be developed, it might ultimately be used as a brain tumor–specific therapeutic agent.

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Delayed Light Imaging for the Early Detection of Plant Stress

Abstract. *Image-intensified photographs of delayed light emission (DLE) from soybean leaves exposed to sulfur dioxide showed evidence of the stress that developed during the exposure period. A comparison of DLE images taken during the fumigation with a conventional photograph taken 5 days later showed a clear correspondence between leaf areas that had the most diminished DLE intensity and those that showed the greatest visible injury. These results suggest that DLE imagery will be a useful tool in the investigation of the spatial distribution and temporal development of plant stress.*

Exposing plant tissue to an acute dose of SO_2 results in irreversible foliar injury most often characterized by interveinal chlorosis in broadleaf species (*1*). This example of spatially heterogeneous response to plant stress is apparently the result of irreversible perturbations in local physiological and metabolic phenomena. Heretofore, little use has been made of the spatial and temporal development of stress characteristics to elucidate plant response to stress. This deficiency is due largely to a lack of experimental techniques that can detail changes in physiological variables over small ($\sim$ 1 mm^2) areas of the whole leaf. Two recently developed methods should help eliminate these shortcomings.

One method, described by Omasa *et al.* (*2*), is a thermal scanning technique that permits spatial mapping of leaf surface temperatures. Since areas of higher or lower temperature correspond to areas of lower or higher transpiration (*2*), respectively, a spatial mapping of surface temperatures can be used to locate differences in transpirational activity and hence differences in gas exchange.

The second development is a method (*3, 4*) by which one can detect stress-induced biochemical or physiological changes in a leaf well before stress symptoms become apparent by measuring delayed light emission (DLE). The DLE phenomenon is universal in green plants and results when light-generated photosynthetic intermediates recombine in the dark to produce an electronically excited state of chlorophyll that gives rise to fluorescence (*5*). The emission originates from chlorophyll molecules associated with the photosystem II (oxygen-evolving) portion of the photosynthetic electron transport chain and is a very sensitive indicator of the status of photosynthetic electron transport (*5*).

Delayed light emission may only be observed in the dark after a period of illumination because its spectrum is identical to and its intensity is much less than that of light-generated chlorophyll fluorescence (*5*). Furthermore, the low intensity of DLE does not permit photography by conventional means (*3*). However, the relatively recent development of image intensifiers has provided a method for recording the spatial distribution of DLE in plants. Björn and Forsberg (*4*) used such a device to record the distribution of DLE from leaves that had been subjected to a number of physiological and pathological stresses. We report here the results of experiments in which this method was used to explore the

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