Table 1. Summary of correlations between V_I data separated into groups at least 2 days apart and corresponding cosmic radiation.

Data sources	Results		
V_I/cosmic rays	r	N	P
Bahamas/satellite	.663	11	<.05
Bahamas/Mount Washington	.486	15	<.05
Germany/satellite	.367	52	<.01
Germany/Mount Washington	.402	151	<.0001

monitor records were also analyzed; they provide a ground-level measure of secondary cosmic radiation going back to 1954.

Barouch mentions the problem of independence in analysis of solar wind data and states that daily averages provide about the right time scale. Analysis of the daily averages of the Bahamas V_I data versus the corresponding satellite cosmic-ray daily averages gave a correlation coefficient $r = .705$, $N = 20$, $P < .001$. To further ensure data independence without reducing the number of points too severely, all V_I data were separated into groups that were at least 2 days apart (generally longer) and averaged. Table 1 summarizes the results of correlating the grouped V_I averages versus the corresponding grouped cosmic radiation averages. In all cases the correlations are positive and significant at or beyond the 5 percent level.

To verify that V_I is predominantly controlled by changes in ionizing radiation compared to upper atmosphere generators, the Bahamas V_I data were correlated with both the B_z component of the interplanetary magnetic field and the AE geomagnetic index. The former is highly correlated with magnetospheric-ionospheric electric fields, while the latter is a good measure of the auroral electrojet, which is maintained by the magnetospheric-ionospheric generator. Both B_z and the AE index were uncorrelated with V_I.

The significance of these results is that we can gain insight regarding the mechanism: for the earth's electric field to increase with greater atmospheric ionization, the effect must be due to larger currents from existing thunderstorms plus possibly positive feedback in the form of increased thundercloud electrification. The effect cannot be due to enhanced ionization in the fair-weather portion of the global electrical circuit because this would partially short out V_I, causing it to become smaller. These findings are in agreement with a proposed mechanism by which solar variability can modulate atmospheric electrification (*2*).

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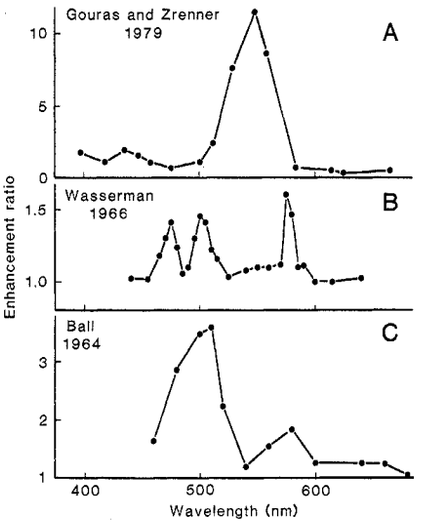

Fig. 1. (A) Gouras and Zrenner's single-cell data replotted. The enhancement ratio was calculated from the different sensitivities to low and high flicker rates [figure 1B in (*1*)]. (B) Wasserman's behavioral results [figure 3 in (*3*)]. (C) Ball's behavioral data [figure 5 in (*4*)]. The enhancement ratio was calculated relative to the Talbot level.

Wavelength-Dependent Enhancement in Brain and Behavior

Gouras and Zrenner have reported that wavelength-dependent latency differences cause color-opponent ganglion cells in the monkey retina to exhibit a wavelength-dependent response enhancement when the frequency of a flickering light is varied (*1*). Their single-cell results correspond to the wavelength-dependent enhancement found when human observers are asked to judge the brightness of intermittent lights (*2-4*). The psychobiological correspondence can best be examined if Gouras and Zrenner's data are replotted in the same form as the most complete behavioral results. Figure 1A shows the enhancement exhibited by Gouras and Zrenner's nine red-green cells. Two peaks occur—a large one near 550 nm and a small one near 435 nm. Figure 1B shows the results from Wasserman's single-flash behavioral study, which found three peaks (*3*). The middle peak in the behavioral data would be expected to correspond with a peak in Gouras and Zrenner's yellow-blue cells. Those data were not reported although their reference 6 leads one to expect that their yellow-blue cells did exhibit a third peak. Figure 1C shows the results of Ball's multiple-flash behavioral study (*4*). He also found wavelength-dependent enhancement peaks, although the two shorter wavelength peaks fused. Two peaks at shorter wavelengths were resolved by Ball when the observer was chromatically adapted to 540 nm.

Even though the size and locations of the peaks vary, the psychobiological correspondence is clearly very good. But certain problems still remain. (i) Gouras and Zrenner's data seem to indicate that the enhancement increases as the flicker frequency is raised from about 1 Hz to about 15 Hz and then declines only slightly at 33 Hz. But the behavioral results, as well as earlier single-cell research conducted with white lights (*5*), would have led one to expect a much greater decline at high frequencies. (ii) Although many behavioral investigations (*2-4*) found wavelength-dependent enhancement effects, other studies (*6*) found that enhancement was independent of wavelength. Therefore, the major current problem in this area is to account for the two different outcomes. One possible hypothesis would be that a wavelength-independent enhancement effect occurs in the achromatic cells in addition to the wavelength-dependent effect in the chromatic cells. Since the relative contribution of the two cell types varies as a function of stimulus size and intensity as well as light adaptation (*7*), one would then expect that studies done under conditions favoring the achromatic cells would tend to show wavelength independence while studies done under conditions favoring the chromatic cells would tend to show wavelength dependence. Gouras and Zrenner may have data that would contribute to the resolution of these questions. If so, the presentation of these data would be very valuable.

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Wasserman (*1*) draws an interesting comparison between a subjective wavelength-dependent brightness enhancement to relatively brief pulses of light and the enhancement of luminance flicker that we found in color-opponent primate retinal ganglion cells (*2*). We did not mention this in our report because we wanted to stress our main theme, that color opponent cells play a role in both color and luminance contrast detection.

The effects Wasserman refers to and our electrophysiological ones are probably related. This is still difficult to establish, however, because there are differences in the results obtained not only between the psychophysical and electrophysiological experiments but also among different psychophysical experiments. The physiology could provide some clues to why such differences occur. In the primate retino-geniculate system, there is a great range in the strength of color opponent interactions a cell may show, especially among the largest fraction of cells, those which only show such interactions between the two mid-spectral or so-called red and green cone mechanisms. Some cells have a spectral cross-over point, that is, a spectral region where their response to light changes from excitation to inhibition, in the middle of the visible spectrum; others have their spectral cross-over point at one or the other end of the spectrum, and some have a cross-over point only when they are chromatically adapted (*3*). The cross-over point of any one cell also varies with both the spatial (*4*) and temporal (*2*) pattern of stimulation, and the entire ensemble of cross-over points of different cells subserving the same area of visual space also changes in a systematic way with distance from the fovea (*3*). The range of cross-over points among retino-geniculate cells may be responsible for the differences found in different experiments, since the wavelength-dependent enhancement depends upon these cross-over points.

In addition, as Wasserman mentions, there is a parallel retino-geniculate system of phasic cells, which also respond to luminance contrast and which do not show overt color opponent interactions (*5*). This phasic system has a retinal distribution different from the tonic one, described above, and consequently its contribution to luminance detection will change in different ways with changes in stimulus size or retinal position.

It is valuable to establish links between single unit neurophysiology and human perception, as Wasserman has attempted to do. Our results agree on an interesting point, namely that channels, in which color-opponent interactions occur, contribute to the perception of luminance contrast. In the visual cortex, there are two distinctly different classes of cells, those responding only to luminance and those responding only to color contrast (*6*, *7*). It is important to understand how these separate contrast detectors are synaptically constructed from the retino-geniculate input to visual cortex. Extant models of color vision imply that this segregation is already established in the retina, under the assumption that color-opponent cells handle color information and non-color-opponent cells handle luminance information (*8*). Recent evidence, however, suggests that color-opponent retino-geniculate cells affect both luminance and color contrast detectors in the visual cortex (*7*) and imply that previous models of color vision are incorrect. The use of color-opponent interactions in luminance contrast detection could have the advantage of ensuring a strong response to luminance across a wider range of spectrally different borders.

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Calcitonin: Aversive Effects in Rats?

Freed *et al.* (*1*) report that injections of the peptide hormone calcitonin reduce food intake in rats after a latency of some hours after injection. They conclude on the basis of a conditioned aversion test that their result "appears not to be the result of illness or aversive effects." Unfortunately the test they chose can reveal only extremes of illness even when such illness is induced within minutes of the ingestion of the preferred novel solution. Freed *et al.*'s (*1*) rats were given access to water only 15 minutes per day and were tested 24 hours after their last drink and were given only a highly preferred taste to drink during the test. Any conditioned aversion would have to overcome both the extreme motivation to drink where the rat has no other choice and the strong preference for a sweet taste. It has been shown (*2*) that such a paradigm (called single bottle because the rat is offered only one bottle during the test) is much less sensitive than a two-bottle test. In a two-bottle test two novel flavors are used. One flavor is followed by an injection of vehicle on one day and the second flavor is followed on a subsequent day by an injection of the drug. On the test day, the rat is given a choice between the two flavors. In this way the rat is not forced to drink a flavor to which it has a conditioned aversion, because another source of water is available. That this is not merely a theoretical objection here may be shown by the case of cholecystokinin (CCK). Reduction in feeding after an injection of CCK was attributed to an induction of satiety (*3*) because CCK did not produce a conditioned taste aversion in the same test as used by Freed *et al.* (*1*). However, when we used a two-bottle test (*4*) a large conditioned taste aversion to CCK appeared. Further work (*5*) has shown that the sickness or malaise produced by the dose of CCK used to show food intake reduction was due to the nonphysiological amounts injected. There is a further problem with calcitonin. The food intake

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