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22 May 1979

## Oxidant Effects on Californian Coastal Sage Scrub

Abstract. *Causes for the reduced cover of native species of coastal sage scrub in certain southern Californian sites were sought among 43 habitat variables. The mean annual concentration of oxidants (which averaged 18 parts per 100 million on the 11 most polluted sites) is statistically indicated as the most likely causal factor. Sites of high oxidant levels in the region are also characterized by declining species richness and equitability.*

Southern California experiences the highest mean annual concentrations of photochemical oxidants in the United States. In 1976, for example, the federal standard for oxidants [8 parts per hundred million (pphm)] was exceeded on 206 days in the South Coast air basin (*1*). While the effect of these oxidants on montane conifer vegetation (*2*) and crop species (*3*) grown in the region has been investigated in some detail, information is lacking on the effect of oxidants and other air pollutants on the predominant native shrubland vegetation of the region: chaparral and coastal sage scrub. I report here suggestive evidence that regional oxidant levels are causing a deterioration of the natural structure of sage scrub communities in the most polluted portions of their range.

Coastal sage scrub dominants (such as species of *Salvia*, *Eriogonum*, and *Encelia*) are drought-deciduous, mesophyllic, shallow-rooted, and typically 0.5 to 2.0 m tall. By contrast, the dominant shrubs of the chaparral are evergreen, sclerophyllous, deep-rooted, and typically 1 to 3 m tall. Coastal sage scrub occupies lower elevations (generally below 500 to 900 m) on the coastal and interior sides of the coast ranges, from San Francisco to El Rosario (Baja California). The chaparral occupies upper elevations of the coastal mountains, extending into the North Coast ranges, east to central Arizona, and south into Baja California (*4*).

In 1977–1978 I sampled 67 sites of coastal sage scrub throughout its range. I recorded foliar cover of all plant species along four 25-m line transects within a sample plot 25 m on a side, and recorded the presence of any additional species within the plot which did not intercept the transect. This sample intensity was shown to be adequate for 99 percent replicability in chaparral vegetation (*5*). In addition, for each site I obtained information on 43 habitat variables concerning community structure, topographic position, substrate, climate, fire and grazing history, and mean annual air pollution concentrations (*6*). No sampled site had burned less than 7 years previously, and past or present grazing intensity at the sites was such that the present shrub cover was not noticeably affected. Sites were embedded within areas supporting comparable vegetation, extending at least an additional 25 m on all sides.

Data for eight air pollutants monitored in a network in southern California were obtained from the California Air Resources Board and the South Coast Air Quality Management District. Annual mean concentrations of each pollutant were calculated for as many years as data were available [mean ± standard deviation (S.D.) = 7 ± 4 years; range 1963 to 1977] at each monitoring station. The mean concentration of the air pollutant at the sampling site was estimated by averaging values from nearby monitoring stations and adjusting the values by distance from the site by using the formula of Oshima (*7*). No station was used if it was (i) more than 30 km from the site, except for six coastal sites where prevailing wind conditions made the use of closer inland stations inappropriate; (ii) separated from the site by a topographic barrier greater than 1000 m in elevation and 5 km in length;

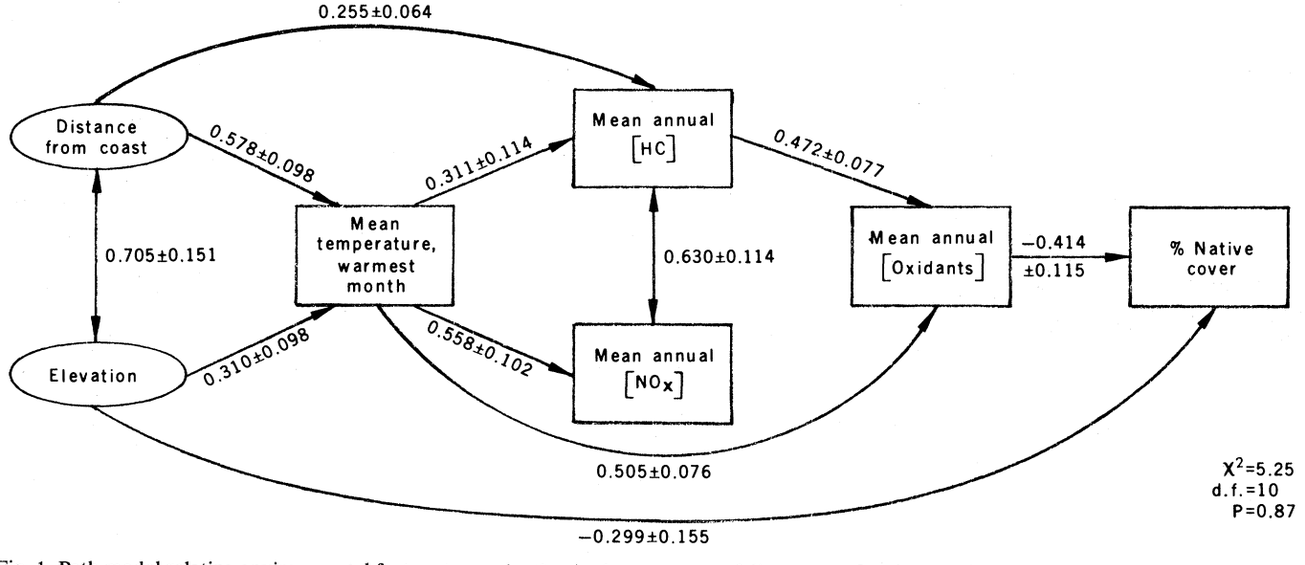


Fig. 1. Path model relating environmental factors to a reduction in the percentage foliar cover of native species of coastal sage scrub in 67 sites in California and Baja California. Single-headed arrows are causal paths (numbers are path coefficients ± standard errors); double-headed arrows indicate correlations; *HC* indicates total hydrocarbons; $NO_x$ indicates oxides of nitrogen.


0036-8075/79/0907-1001$00.50/0
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or (iii) located downwind of the prevailing winds. Of the annual records of oxidants, 61 percent are for ozone, peroxyacyl nitrates, and $NO_2$ (with interference by $SO_2$). The remainder of the oxidant records report concentrations of ozone only, as a result of changes in sampling methods. Sandberg *et al.* (*8*), using the two methods in parallel, found an average difference of 2 percent in readings.

The 43 habitat variables at the 67 sites were tested for their Pearson correlation with the percentage of total cover at each site composed of native species. The variable which showed the most significant correlation with the percentage of native cover was the mean annual oxidant concentration ($r = -.58$; $P < .0001$). Two other variables that showed strongly significant correlations were elevation and mean maximum temperature of the warmest month ($r = -.52$; $P < .0001$). These results suggested the possibility that native cover might be declining at the warmer, higher, inland sites (for example, Riverside basin) due to factors associated with climate, and that air pollution was an insignificant covariable. The partial correlation coefficients of oxidants with the percentage of native cover, however, remained high when the covariations with elevation, mean maximum temperature of the warmest month, and distance from the coast were extracted ($r = -.41$, $-.35$, and $-.42$ respectively). In order to explore further the interrelations of these variables, a series of models were tested, by means of path analysis (*9*), to determine the most likely route of causation of the decline in native cover.

A path model that adequately fits the data examined (chi-square test, $P = .87$) is shown in Fig. 1. While each of the variables could act independently to reduce native cover, a plausible model of causation also exists. Since prevailing winds in southern California are westerly during the summer when oxidant formation is greatest, oxidants that form from the photocatalytic reaction of reactive hydrocarbons and nitrogen oxides are blown inland to warmer, somewhat higher elevation sites in Riverside and San Bernardino counties before they are reduced. Increasing distance from the coast does not significantly influence native cover in a direct fashion, but does so through resulting higher mean summer temperatures (continental climate) and higher concentrations of hydrocarbons (from prevailing westerlies) (Fig. 1). Higher mean temperatures are known to increase the rate of formation of oxidants, and this, too, is reflected in the model. The model also suggests that hydrocarbons, rather than nitrogen oxides, are rate-limiting to oxidant formation in the region of study, a conclusion for which there is independent confirmation (*10*). Higher elevation appears to decrease native cover, both through a direct influence and through its influence on oxidant formation. Apart from the gradual increase in elevation inland, it is generally the case that oxidant levels are higher just below the ceiling of prevailing inversion layers, which often occur at 300 to 600 m in this region. The model indicates that the single strongest direct cause of the decline in native cover is the increasing mean annual concentrations of oxidants.

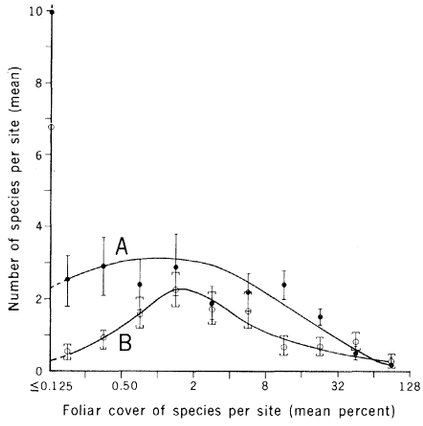


Fig. 2. Lognormal distributions of coastal sage scrub species in 11 clean air sites (A, 4 pphm oxidants) and 11 floristically comparable polluted sites (B, 18 pphm oxidants).

Studies of the effect of pollution on natural ecosystems have indicated that, in general, the effect of long-term, continual damage is to decrease not only the total foliar cover of the vegetation, but also to decrease species richness and to increase the concentration of dominance, hence, decrease the equitability, by favoring a few, tolerant species (*11*). This generalization is the basis for a second test of the effect of oxidants on coastal sage scrub. If the decline in native cover observed in the inland, desert-margin sites of high oxidant level were due to natural factors associated, for example, with a decline in precipitation, one would expect species richness and equitability to be maintained or even increased as desert elements enrich the stands. The direct correlation of native cover with mean annual precipitation is considerably weaker ($r = .23$, $P < .06$) than that with oxidants ($r = -.58$). Further, nine of the ten most dominant species in the sage flora show a stronger correlation of percentage cover with oxidants than with precipitation.

The effects of oxidants on diversity relations were examined in two ways. The first approach was by use of Pearson correlation coefficients on the 67 sites. As mean annual oxidant concentrations increased, species richness declined ($r = -.23$; $P < .05$), equitability as measured by Whittaker's log-cycle equitability index (*12*) declined ($r = -.24$; $P < 0.5$), and concentration of dominance as measured by Simpson's index (*13*) increased ($r = .24$; $P < .05$). These results are consistent with the hypothesis that community structure is being directly affected by long-term continual pollution, not with the hypothesis that the decline in native cover is due merely to an increasing desert influence. The conclusion would be strengthened if a direct comparison of diversity in sites of high oxidant level were made with floristically comparable sites of low oxidant level. This comparison was approached in the following way.

A primary axis of floristic variation was extracted from the 67 sites by reciprocal averaging ordination (*14*). The 11 sites with highest mean annual oxidant concentrations ($\bar{x} \pm$ S.D. = $18 \pm 3$ pphm) were found to be among the 22 sites at one extreme of the axis. These sites were from the eastern Los Angeles and Riverside basins (desert-margin sites). The other 11 sites included within the range of high-oxidant sites along the ordination axis, and thus of highest floristic similarity to them, were from San Diego County and northern Baja California (including several strongly desert-influenced sites). The mean annual oxidant concentration of the latter 11 sites was $4 \pm 3$ pphm. The relative importance of species within sites was compared for the two groups by the semilog plot method of Preston (*15*). Percentage foliar cover was used as the measure of importance. Sampling methods were not sufficiently sensitive to obtain accurate cover estimates below 0.125 percent cover; the mean number of species present at this value or less is indicated on the ordinate.

The results in Fig. 2 indicate that the more polluted sites have a lower number of species per octave, and a lower total richness ($\bar{x} = 18$ as compared to 29 at the less polluted sites). An increase in concentration of dominance relative to the less polluted sites is indicated by the narrower gap between curves among the higher abundance octaves. These effects of pollution on the lognormal plot were also found by Patrick *et al.* (*16*) in studying abundance of diatoms in polluted and unpolluted streams.

In sum, observations by correlation

and path analysis suggest an adverse effect of annual oxidant concentration on the proportion of total cover represented by native species of coastal sage scrub. Further tests suggest that oxidants are affecting community structure not only by reducing the total cover of native species, but by reducing species richness and equitability. These tests are relatively standard methods, and their results are consistent with other studies on the effects of pollution on community structure (*11, 16, 17*). It must be recognized, however, that such synecological methods, like epidemiological methods examining the effects of pollution on public health, can only suggest likely causal routes of damage. Actual demonstration of an effect of oxidants on particular coastal sage species must await laboratory experiments. In view of the importance of the natural functioning of coastal sage scrub to the environment and economy of southern California (*18*), these results suggest the value of initiating such laboratory experiments promptly.

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21 February 1979; revised 14 May 1979

## Insulin Receptor Binding in Obesity: A Reassessment

Abstract. *A defect in the binding of insulin to circulating monocytes occurs when obese patients are hospitalized and fed a liberal carbohydrate diet. Under ordinary circumstances, most obese patients have normal insulin binding despite very high concentrations of serum insulin. These results show that insulin does not necessarily regulate its own receptor in vivo—as it does in vitro.*

A decrease has been observed in the number of insulin receptors in lymphocytes and hepatocytes that have been grown in the presence of insulin (*1*). Thus, it has been suggested that "down regulation" of receptor binding by insulin itself explains the apparent insulin resistance noted in certain clinical states characterized by hyperinsulinemia (*2*). Obesity is commonly associated with an increase in the concentration of serum insulin and an apparent resistance to the action of exogenous insulin (*3*). Several studies (*4*) indicate that obese patients have decreased insulin binding to circulating monocytes and isolated fat cells, which suggests that a defect in the interaction between insulin and its receptor may account for the apparent insulin resistance of obese persons (*2*). In one study (*5*), however, no defect was found in the binding of insulin to fat cells from obese patients; their insulin resistance was attributed to a defect in intracellular glucose metabolism.

We investigated insulin binding to circulating monocytes in 27 massively obese patients prior to giving them intestinal bypass surgery (*6*). These patients had been observed in our clinic for 2 to 3 months before our investigation. They were instructed to maintain their usual diet and level of activity. Their average weight gain during this period of time was 0.7 ± 0.3 kg per month (mean ± standard error). Blood was obtained on the morning after the patients were admitted to the hospital and had fasted

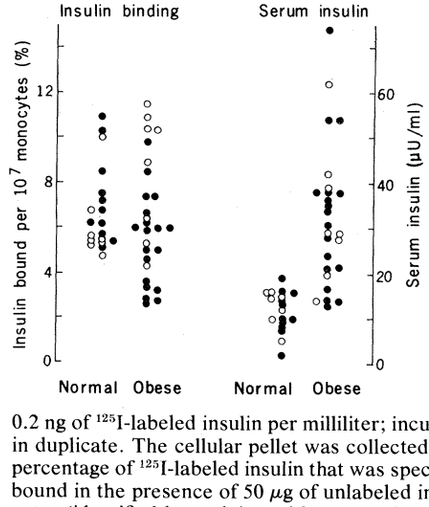


Fig. 1. Serum insulin concentrations during fasting and insulin binding in obese patients and in controls of normal weight. Males are denoted by ○ and females, by ●. Mean serum insulin was 33 ± 3 μU/ml (mean ± standard error) in obese patients and 12 ± 1 μU/ml in controls. Insulin binding was 6.2 ± 0.5 percent in obese patients and 6.8 ± 0.4 percent in controls. [Insulin was measured by immunoassay (*11*).] The method for determining the binding of insulin to circulating monocytes has been described by de Meyts (*12*). Mononuclear leukocytes were separated from whole blood by centrifugation on a Ficoll-Hypaque gradient. Approximately 25 million cells were incubated for 90 minutes at 25°C in Hepes buffer [4-(2-hydroxyethyl)-1-piperazineethanesulfonic acid; 0.5 ml] containing 0.2 ng of $^{125}$I-labeled insulin per milliliter; incubations were performed in triplicate and sampled in duplicate. The cellular pellet was collected and washed in a Beckman microcentrifuge. The percentage of $^{125}$I-labeled insulin that was specifically bound (percentage total minus percentage bound in the presence of 50 μg of unlabeled insulin per milliliter) was expressed per $10^7$ monocytes (identified by staining with nonspecific esterase). Samples from obese patients were assayed at the same time as samples from control subjects. In nine controls, the data plotted are means of multiple determinations.


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