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# Climate controls on valley fever incidence in Kern County, California

Charles S. Zender

Department of Earth System Science, University of California at Irvine

Jorge Talamantes

Department of Physics and Geology, California State University at Bakersfield

**Abstract.** Coccidioidomycosis (valley fever) is a systemic infection caused by inhalation of airborne spores from *Coccidioides immitis*, a soil-dwelling fungus found in the southwestern United States, parts of Mexico, and Central and South America. Dust storms help disperse *C. immitis* so risk factors for valley fever include conditions favorable for fungal growth (moist, warm soil) and for aeolian soil erosion (dry soil and strong winds). We analyze and intercompare the seasonal and interannual behavior of valley fever incidence and climate risk factors for the period 1980–2002 in Kern County, California, the US county with highest reported incidence. We find weak but statistically significant links between disease incidence and antecedent climate conditions. Precipitation anomalies eight and twenty months antecedent explain only up to 4% of monthly variability in subsequent valley fever incidence during the 23 year period tested. This is consistent with previous studies suggesting that *C. immitis* tolerates hot, dry periods better than competing soil organisms, and, as a result, thrives during wet periods following droughts. Furthermore, the relatively small correlation with climate suggests that the causes of valley fever could be largely anthropogenic in Kern County.
Seasonal climate predictors of valley fever in Kern County are similar to, but much weaker than those in Arizona, where previous studies find precipitation explains up to 75% of incidence. Causes for the discrepancy between climate associations with valley fever in California and Arizona are not yet understood. Higher resolution temporal and spatial monitoring of soil conditions could improve our understanding of climatic antecedents of severe epidemics.

## 1. Introduction

Coccidioidomycosis is a systemic infection caused by inhalation of airborne spores of *Coccidioides immitis*, a soil-dwelling fungus found in the southwestern United States, parts of Mexico [Maddy and Coccozza, 1964], and Central and South America [Centers for Disease Control and Prevention, 1994]. *C. immitis* thrives in moist soils, is spread by wind events, and therefore has many environmental risk factors. Epidemiologic studies in the 1930's [Deresinski, 1980; Larwood, 2000] linked coccidioidomycosis to the regional disease known as San Joaquin Fever, also known as valley fever. Risk management and cost-effectiveness studies show that a vaccine for valley fever is plausible and should be ad-

ministered to newborns in highly endemic counties including Kern in California and Pima in Arizona [Galgiani, 1999; Barnato et al., 2001]. These and earlier studies [Centers for Disease Control and Prevention, 1994, 1996] recommend intensifying efforts to better characterize climate risk factors for acquiring infection. This study explores climate-related risk factors for valley fever in Kern County and quantifies their level of significance.

Early studies of environmental causes of valley fever [Smith et al., 1946; Maddy, 1957; Hugenholtz, 1957] elucidated a lifecycle that accounts for many observed features of *C. immitis* blooms and subsequent coccidioidomycosis incidence. Pappagianis [1988] synthesized the climatological aspects of this lifecycle gathered from those and following studies. *C. immitis* thrives in the soil ("blooms") during wet periods lasting several weeks. Infections tend to occur in the dry season when soils are most mobile. Incidence often increases after a heavy wet season following a prolonged dry spell.

In the most quantitative analysis of climate controls on valley fever incidence to date, Kolivras and Comrie [2003] found that antecedent precipitation and temperature are moderate climate risk factors for valley fever in Pima County (which includes Tucson), Arizona, USA. They developed a multivariate model to predict valley fever incidence in Arizona in a given month based on climate conditions and anomalies in the antecedent 3.5 yr. Moreover, Kolivras and Comrie's statistical model uses and predicts a metric called the transformed incidence anomaly. This is the monthly incidence anomaly relative to the annual (rather than climatological, or climatological monthly) mean. The maximum transformed incidence anomalies they reported in Pima County are about 10%, and their statistical model predicts up to half of some anomalies.

The transformed incidence is insensitive to uniform increases in monthly incidence which result in an absolute annual increase (e.g., an epidemic) but which do not change the relative contribution of each month to the annual incidence. By contrast, the 1991–1995 epidemic in Kern County increased interannual and intra-annual variations in incidence by about 1000% (ten-fold). This appears to be the largest well-documented valley fever epidemic on record.

Previous studies identify no clear cause for the 1991–1995 epidemic [Centers for Disease Control and Prevention, 1994; Jinadu, 1995; Kirkland and Fierer, 1996]. The most likely climate factor contributing to the epidemic was the increased rainfall that ended a five year drought in California in March, 1991 [Jinadu, 1995; Kirkland and Fierer, 1996]. The following two winters were twice as wet as normal [Jinadu, 1995]. Possible exacerbating demographic factors were an increased immuno-suppressed population, and less prior exposure (which develops immunity) in the general population [Kirkland and Fierer, 1996; Centers for Disease Control and Prevention, 1996].

We analyze the links between climate and *C. immitis* epidemiology using the Jan. 1980 to Dec. 2002 record (23 years) of monthly statistics from Kern County, California. Our objectives are twofold: First, we explore climate-related risk factors for valley fever in Kern County and quantify their level of significance. In the second portion of our study we contrast our results from Kern County to results from a similar study in Pima County, Arizona [Kolivras and Comrie, 2003] which experiences a significantly different climate. This comparison shows us the extent to which valley fever predictability depends on local climate, and how that may differ with climate regime.

The paper is organized as follows. Section 2 describes the climate and epidemiological data sources and the procedures used to correlate them. Section 3 shows the results of our tests for statistically significant correlations between incidence and climate. Section 4 discusses the climate-related predictability of valley fever in Kern County, and its relationship to other regions. Section 5 summarizes our findings.

## 2. Materials and Methods

Climate variables in Kern County are from the Solar and Meteorological Surface Observational Network Dataset (SAMSON, available from National Climatic Data Center, Asheville, North Carolina) for 1961–1990. We use NOAA Hourly United States Weather Observations (HUSWO) for 1990–1995, NOAA Integrated Surface Hourly Observations (ISHO) for 1995–2000, and NWS Hanford Forecast station website for daily data in 2001–2002. All four datasets come from measurements taken at Bakersfield airport. Hourly and daily weather data are averaged to obtain monthly means.

Valley fever incidence statistics for Kern County were obtained from the California Department of Health Services (CDHS). Monthly incidence reports are available from 1980–2002, while annual incidence data pre-date 1980. Kern County is the national center for serologic testing for valley fever. The high awareness in Kern County leads to better reporting.

### 2.1. Climatological and Monthly Anomalies

We base our analysis on the climatological monthly anomalies of incidence and climate data. From a physical standpoint, one expects climatological anomalies to drive incidence anomalies. For example, wind speed threshold velocities for dust production depend directly on soil conditions, and not on season per se. Similarly, one expects *C. immitis* to respond to deviations in the climatological mean amount of

soil moisture, rather than the climatological monthly mean soil moisture. However, we learned more about the link between climatological and biological processes by examining the climatological monthly anomaly (i.e., deviation from the mean annual cycle) than by examining the absolute climatological anomaly.

The very strong annual cycles of the (raw) time series we considered dominate the physical picture—correlation analysis of these time series only shows this effect, and nothing else. We therefore removed the annual cycle from all data. The resulting time series are strongly autocorrelated (e.g., a particularly warm July likely follows an unusually hot June). Analysis based on *these* time series results in artificially strong correlations between incidence and meteorological parameters, while teaching us nothing about incidence anomalies in general, and epidemics in particular. We therefore removed those autocorrelations by applying an autoregression procedure of sufficiently high order [Chatfield, 2004]. Finally, we performed lag correlation analyses only after correcting for annual cycles and time-series autocorrelations.

### 2.2. Lifecycle

Unique among pathogenic fungi, *C. immitis* forms infectious spores known as arthroconidia at the distal ends of filimentary hypha [Kirkland and Fierer, 1996]. During wind events, the hypha terminating in arthroconidia may rupture, releasing them to the atmosphere. Infection occurs almost exclusively through the respiratory route. After inhalation, the arthroconidia develop into spherules each containing hundreds to thousands of offspring known as endospores. These spherules rupture in 2–3 days, and each endospore may develop into a mature spherule, repeating the process.

The *C. immitis* lifecycle is dimorphic, with saprophytic and parasitic phases [e.g., Pappagianis, 1988; Kolivras et al., 2001]. In saprophytic phase, *C. immitis* lives in the upper layers of soil where it obtains nourishment from dead or decaying organic matter. Appropriate temperature, soil moisture, and soil texture conditions allow the fungus to develop hyphae, slender filaments of cells which grow upward in the soil. As conditions dry and the fungus becomes drought stressed, alternate cells in the hyphae undergo suicide. The remaining, viable cells may become arthroconidia which develop into parasitic endospores. Forces which disturb the soil, rupture the hypha, and dislodge spores include natural events such as wind gusts and anthropogenic disturbances.

Precipitation influences valley fever directly in at least two ways. First, *C. immitis* thrives in moist soils, which are thought to be required for blooms to occur [Pappagianis, 1988]. Moreover, there is some evidence that *C. immitis* tolerates drought longer than competing species [Kolivras et al., 2001]. This would allow it to recover quickly post-drought and to become more pervasive.

### 2.3. Dispersal via Saltation-Sandblasting

Previous discussions of *C. immitis* dispersal [e.g., Pappagianis and Einstein, 1978; Kolivras et al., 2001; Kolivras and Comrie, 2003] are incomplete in that they mention only wind and dust storms as the primary natural dispersal mechanism. *C. immitis* arthrospores range from $1.5$–$4.5\,\mu\mathrm{m}$ in width and $5.0$–$30\,\mu\mathrm{m}$ in length [Pappagianis, 1988; Kolivras et al., 2001]. Clay and small silt-sized particles like arthrospores are tightly bound to the soil surface, or to larger particle aggregates, by inter-particle cohesion [Iversen and White, 1982], electrostatic, and capillary forces [Hillel, 1982; McKenna-Neuman and Nickling, 1989]. Wind tunnel experiments [Iversen and White, 1982; Shao and Lu, 2000, e.g.,] show that wind speeds required to directly entrain particles as small as *C. immitis* hypha and arthrospores exceed about $20\,\mathrm{m\,s^{-1}}$. Maximum monthly wind speeds recorded in Kern County since 1961 are usually less than $15\,\mathrm{m\,s^{-1}}$ and exceed $20\,\mathrm{m\,s^{-1}}$ only five times, so direct entrainment by wind is probably responsible for only a small minority of *C. immitis* dispersal events.

We speculate that saltation is usually the proximate cause of hypha rupture, and that sandblasting is the normal dispersal mechanism for the airborne arthrospores. Saltation-sandblasting theory is well-known in agricultural and dust emissions studies [e.g., Iversen and White, 1982; Alfaro et al., 1997; Grini et al., 2002]. Saltation and sandblasting can disperse arthrospores at much lower wind speeds ($6$–$10\,\mathrm{m\,s^{-1}}$) than direct wind entrainment, and is therefore the only plausible mechanism for many dispersal events.

Saltation, the direct entrainment and movement of particles by wind, initiates when the surface wind friction speed $u_*$ (a measure of surface wind stress) exceeds a soil-dependent wind friction threshold $u_{*\mathrm{t}}$. Soil texture, moisture, and crustal strength determine $u_{*\mathrm{t}}$. For typical dry soils, $u_{*\mathrm{t}}$ ranges between $0.2$–$0.5\,\mathrm{m\,s^{-1}}$, corresponding to threshold wind speeds of $6$–$15\,\mathrm{m\,s^{-1}}$ at $10\,\mathrm{m}$ height. Once $u_* > u_{*\mathrm{t}}$, the wind directly entrains loose sand-sized particles into a shallow layer (the saltation layer) between the surface and about $1\,\mathrm{m}$.

However, most directly entrained (i.e., sand-sized) particles are too large to rise above the saltation layer. As the turbulence carries them down wind, they repeatedly impact the surface. Each surface impact may dislodge or disaggregate both smaller (silt and dust) and larger (coarse sand) particles which also may become entrained into the saltation layer [Grini et al., 2002]. This process is known as sandblasting. A well developed saltation layer may cause mil-

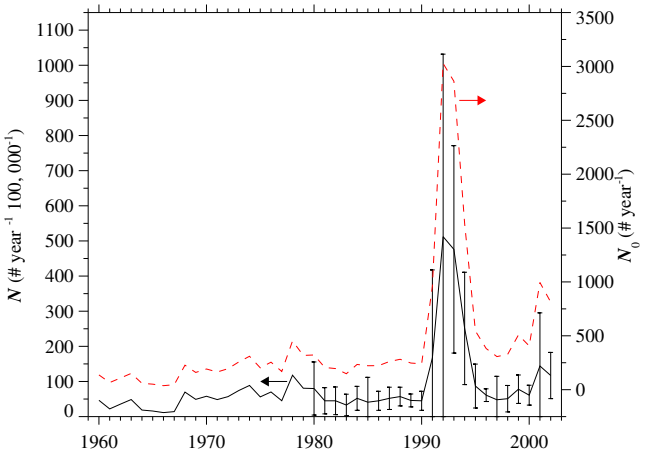


**Figure 1.** Annual incidence $N$ [# yr$^{-1}$ (100,000)$^{-1}$] (solid line) and total number of reported cases $N_0$ [# yr$^{-1}$] (dashed line) of valley fever in Kern County from 1960–2002. Bars show two standard deviations of each year's monthly incidence statistics projected to annual rates.

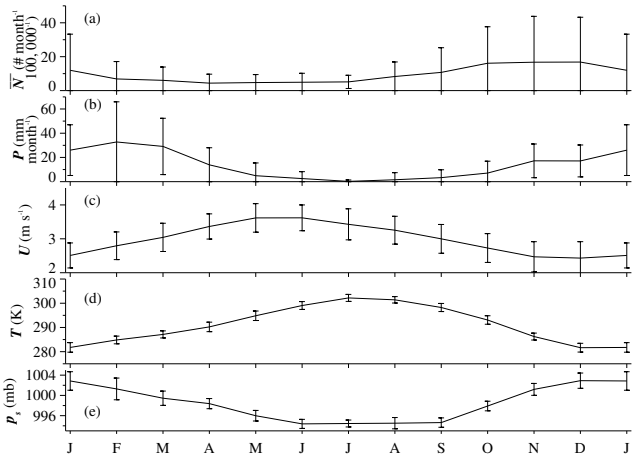


**Figure 2.** Annual cycle of coccidioidomycosis incidence and potential climate risk factors from 1980–2002. Shown are monthly mean (a) incidence $\bar{N}$ [# mo$^{-1}$ (100,000)$^{-1}$] (b) precipitation $\bar{P}$ [mm mo$^{-1}$], (c) wind speed $\bar{U}$ [m s$^{-1}$], (d) surface temperature $\bar{T}_s$ [K], (e) surface pressure $\bar{p}_s$ [mb]. Bars span two standard deviations of the inter-annual variability computed separately for each month. Standard deviations computed using 1980–2002 data for incidence, 1961–2002 for climate variables.

lions of impacts per square meter per second. The saltation layer develops, thickens, and roughens as directly entrained and sandblasted particles accumulate with the fetch of the wind [Gillette et al., 1997]. Sandblasting, not direct wind entrainment, injects the vast majority of surface particles into the saltation layer and thence the free atmosphere. However, unusually strong gusts exceeding about 20 m s$^{-1}$ can entrain arthrospores directly (without saltation sandblasting).

### 2.4. Epidemiology

We are most interested in identifying climate and soil-related anomalies in *C. immitis* in order to assess the susceptibility of endemic regions to increased incidence of valley fever given accurate predictions of seasonal-to-interannual climate anomalies. Seasonal-to-interannual climate predictability has improved in recent years as teleconnections between climate modes (e.g., ENSO) and regional climate become better understood and represented in models [e.g., Glantz et al., 1991]. Unfortunately, data abundance of *C. immitis* in soil are unavailable. At this time the best proxy available is case incidence, even though many steps separate growth of *C. immitis* in soil from case incidence [Kolivras and Comrie, 2003].

The absolute incidence $N_0$ [# yr$^{-1}$] of valley fever in Kern County from 1960–2002 and the incidence per unit population $N$ [# yr$^{-1}$ (100,000)$^{-1}$] are nearly identical in shape (Figure 1). We always use $N$ rather than $N_0$ for statistical comparisons. During this period the Latino population fraction increased about 7% per decade since 1970 to about 35% now. This demographic trend is not detectable in the incidence statistics, suggesting that Latinos are as susceptible as the original demographic.

The inter-annual variability (one standard deviation) in annual valley fever incidence from 1960–2002 is 102 yr$^{-1}$ (100,000)$^{-1}$, 120% of the mean incidence of 85 yr$^{-1}$ (100,000)$^{-1}$. The interannual variability from 1991–2002 is 164 yr$^{-1}$ (100,000)$^{-1}$, significantly greater than 23 yr$^{-1}$ (100,000)$^{-1}$ for the period 1960–1990. Incidence $N$ in 2001 and 2002 was higher than any previously recorded level except the epidemic of 1991–1995 [Jinadu, 1995].

The intra-annual variability is shown for 1980–2002, when monthly incidence data were available. The fractional intra-annual variability $\bar{\sigma}$ is the standard deviation of annual incidence rates computed from monthly rates multiplied by twelve. Despite large interannual changes in $N$, the mean $\bar{\sigma}$ is close to 122 yr$^{-1}$ (100,000)$^{-1}$. Thus the monthly incidence is within 244 yr$^{-1}$ (100,000)$^{-1}$ of the annual mean incidence in 10–11 months in most years.

### 2.5. Climatology

Coccidioidomycosis incidence $\bar{N}$ [# mo$^{-1}$ (100,000)$^{-1}$] in Kern County and the climate risk factors that may be associated with it show pronounced annual cycles (Figure 2). Monthly valley fever incidence since 1980 shows

a strong annual cycle superimposed on a relatively uniform background rate of order $5\,\mathrm{mo}^{-1}\,(100{,}000)^{-1}$. Exposures due to non-environmental causes, e.g., construction, excavations, are expected to contribute to the background incidence [Maddy, 1957; Kolivras et al., 2001]. Incidence increases from $4.7\,\mathrm{mo}^{-1}\,(100{,}000)^{-1}$ during spring months (Apr–Jun) to $17\,\mathrm{mo}^{-1}\,(100{,}000)^{-1}$ during fall (Oct.–Dec.), when 60% of all cases are reported. One should keep in mind that the minimum time from exposure to incidence is about two weeks, and that many cases progress unreported for months, until victims' conditions are serious enough to require medical care [Pappagianis and Einstein, 1978]. On average, it takes about five weeks from infection to reporting (T. R. Larwood, personal communication, 2003).

The most variable climate characteristic in Kern County is rainfall. The climatological mean precipitation $\bar{P}$ from 1961–2002 is $15.8 \pm 23.1\,\mathrm{cm\,yr}^{-1}$. *C. immitis* prevalence decreases in climates with precipitation rates $\bar{P} < 10\,\mathrm{cm\,yr}^{-1}$ and $\bar{P} > 50\,\mathrm{cm\,yr}^{-1}$ [Kolivras et al., 2001]. Thus Kern County receives enough precipitation for growth of *C. immitis* in average and moist years. Incidence in California peaks from Oct.–Jan., the end of the dry season, as noted in previous studies [e.g., Smith et al., 1946; Pappagianis, 1988]. Precipitation from the cold northwesterly frontal systems peaks in late winter, and seems to reduce further incidence, perhaps by dampening soil and suppressing Aeolian erosion.

The climatological mean wind speed $\bar{U}$ in Kern County from 1961–2002 is $3.0 \pm 0.6\,\mathrm{m\,s}^{-1}$. Instantaneous wind speed $U$ (not shown) from Kern County exceeds the 6–$15\,\mathrm{m\,s}^{-1}$ at 10 m height required for soil deflation many times each month. Seasonal winds peak in May–July, the beginning of the dry season. The seasonal coincidence of peak winds with drying soil in Kern County seems to favor Aeolian distribution of arthoroconidia, and thus infection, in summer.

## 3. Results

### 3.1. Univariate Correlations

Similar to previous studies in other regions [Hugenholtz, 1957; Kolivras and Comrie, 2003], we examined correlations between annual cycles of valley fever incidence, precipitation, winds, and temperature (cf. Figure 2). Figure 3 shows the unranked (Pearson) linear correlation coefficient $r$ between the autoregression-corrected climatological monthly valley fever anomaly $N^*$ and the (autoregression-corrected) climatological monthly anomalies of four potential climate risk factors: precipitation $P^*$, wind speed $U^*$, surface temperature $T_\mathrm{s}^*$, and surface pressure $p_\mathrm{s}^*$. The results are qualitatively similar if ranked (Spearman) correlations $r_\mathrm{s}$ are used instead.

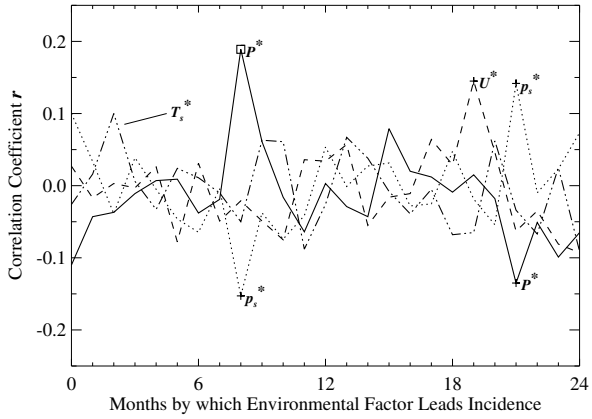


**Figure 3.** Lag correlation coefficient $r$ between climatological monthly valley fever anomaly $N^*$ and climatological monthly anomalies of four potential climate risk factors: precipitation $P^*$, wind speed $U^*$, surface temperature $T_\mathrm{s}^*$, and surface pressure $p_\mathrm{s}^*$. Plusses (+) and squares (□) indicate confidence statistics $p$ better than 5% and 1%, respectively.

The confidence statistic $p$ is only better than 1% once, for correlation between precipitation anomaly eight months antecedent. The confidence statistic $p$ is better than 5% once for wind speed anomaly (19 months antecedent), once more for precipitation anomaly (21 months antecedent), twice for surface pressure anomaly (discussed below), and never for surface temperature anomaly. Table 1 summarizes these results. It shows, in decreasing order of significance, all statistically significant confidence statistics $p < 0.05$ between valley fever anomalies and climate anomalies in Figure 3. All associated lag-correlation coefficients $r$ and $r_\mathrm{s}$ are $< 0.20$. Hence our central result is that climate anomalies do not provide a robust method for predicting incidence in Kern County, based on 23 years of monthly data.

Three climate risk factors ($P^*$, $U^*$, and $T_\mathrm{s}^*$) directly influence the lifecycle or dispersal of *C. immitis* as described in Section 2.2. Of these three, precipitation anomalies are the only highly statistically significant ($p < 0.01$) climate indicators of monthly valley fever incidence in Kern County in the year preceding incidence (Figure 3). The statistically significant ($p < 0.05$) links between incidence and surface pressure anomalies appear to be false positives since there is no physically plausible direct relationship between $p_\mathrm{s}^*$ and *C. immitis* lifecycle. The remaining statistically significant ($p < 0.05$) correlations between incidence and precipitation and wind speed may also be false positives. The long lags

**Table 1. Significant Correlations with Climate Anomalies**[b]

| Anomaly | # mo.[c] | $r$[d] | $r_s$[e] | $p$[f] |
|---|---|---|---|---|
| Precipitation $P^*$ | 8 | 0.19 | 0.11 | 0.0020 |
| Precipitation $P^*$ | 21 | −0.14 | −0.13 | 0.028 |
| Wind Speed $U^*$ | 19 | 0.15 | 0.074 | 0.018 |
| Sfc. Pressure $p_s^*$ | 8 | −0.15 | −0.08 | 0.018 |
| Sfc. Pressure $p_s^*$ | 21 | 0.14 | 0.16 | 0.028 |

[a]Sample size $M = 264$ for $P^*$, $U^*$, and $T_s^*$; $M = 240$ for $p_s^*$.

[b]Sample size $M = 264$ for $P^*$, $U^*$, and $T_s^*$; $M = 240$ for $p_s^*$.

[c]Number of months by which monthly climate anomaly $x^*$ leads monthly valley fever anomaly $N^*$ for this correlation

[d]Pearson correlation coefficient

[e]Ranked (Spearman) correlation coefficient

[f]Confidence statistic (probability that incidence and climate factor are uncorrelated)

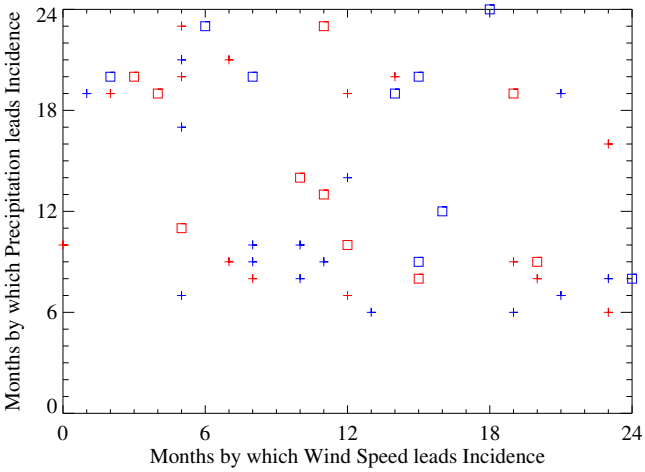


**Figure 4.** Lag correlation coefficient $r$ between valley fever incidence anomaly $N^*$ and product of precipitation and wind speed anomalies ($P^*U^*$) from 1980–2002. Plusses (+) and squares (□) indicate confidence statistics $p$ better than 5% and 1%, respectively.

(19 and 21 months, respectively) between incidence and $U^*$ and $P^*$ anomalies raise questions of causality that Kolivras and Comrie [2003] discuss in more detail.

### 3.2. Bivariate Correlations

We next examine whether combinations of climate anomalies explain more of the incidence anomaly than individual climate anomalies. Such bivariate associations are suggested by the *C. immitis* lifecycle discussed in Section 2.2. First we ask if valley fever incidence is more sensitive to a wind speed anomaly that occurs a certain number of months after a dry spell (or wet spell) than to a wind speed anomaly that occurs the same number of months after a normal precipitation period. Figure 4 shows the linear correlation coefficient $r$ between the valley fever incidence anomaly $N^*$ and the product of $P^*$ and $U^*$ (i.e., $P^*U^*$) from 1980–2002. The maximum bivariate $r$ value is about 0.25. This offers no significant improvement in incidence predictability relative to univariate regressions.

Figure 4 has two bands of bivariate significance, centered on precipitation anomalies 6–10 and 18–22 mos prior to incidence anomalies. The wind speed anomaly seems to have no preferred phasing with respect to the precipitation anomaly. Twenty seven and nineteen regression coefficients are significant ($0.01 < p < 0.05$) and highly significant ($p < 0.01$), respectively. With $25 \times 25 = 625$ regression coefficients, we expect about six false positives at the highly significant ($p < 0.01$) level. Hence precipitation anomalies about eight and twenty months prior to incidence both appear to play a statistically significant, detectable role in determining incidence. The twelve month offset between bands of statistical significance is consistent with the lifecycle discussed in Section 2.2. Due to its relative drought-tolerance, *C. immitis* populations are thought to rebound after rainy seasons following a prolonged drought.

Are temperature anomalies better incidence predictors in Kern County when combined with precipitation? We computed the linear correlation coefficient $r$ between the valley fever incidence anomaly $N^*$ and the product of precipitation and temperature anomalies from 1980–2002. The result (not shown) is very similar to Figure 4 and leads to the conclusion that coupling of $T_s^*$ to $P^*$ explains less of $N^*$ than does coupling of $U^*$ and $P^*$.

### 3.3. Epidemic Years

During the 1991–1995 epidemic, annual incidence $N$ increased five-to-tenfold, from 50 to 500 $\mathrm{yr}^{-1}$ $(100{,}000)^{-1}$ (cf. Figure 1). In non-epidemic years, 65% of cases are reported between November and January. The epidemic amplified this strong late-fall early-winter seasonality. During the 1991–1995 epidemic, 59% of the anomalous (actual minus expected) cases were reported in Nov.–Jan. This dramatic seasonal increase appears in the annualized monthly variability in $N$ (Figure 1). Incidence reports for 2001–2002 also show a significant increase above the long term background level. Thus understanding factors contributing to epidemic outbreaks is of current concern in California.

In order to help distinguish climatic from demographic causes of the 1991–1995 epidemic, we divide the data into

**Table 2. Time Series Analyzed Separately**

| | Description | Start | End | # mo.[a] |
|---|---|---|---|---|
| A | Entire 23 year record | 1980 | 2002 | 276 |
| B | Until epidemic start | 1980 | 1990 | 132 |
| C | Until epidemic end | 1980 | 1995 | 192 |
| D | Epidemic omitted | 1980 | 2002 | 216 |
| E | Epidemic end to record end | 1996 | 2002 | 84 |
| F | Epidemic only | 1991 | 1995 | 60 |
| G | Epidemic start to record end | 1991 | 2002 | 144 |

[a]Number of months in period

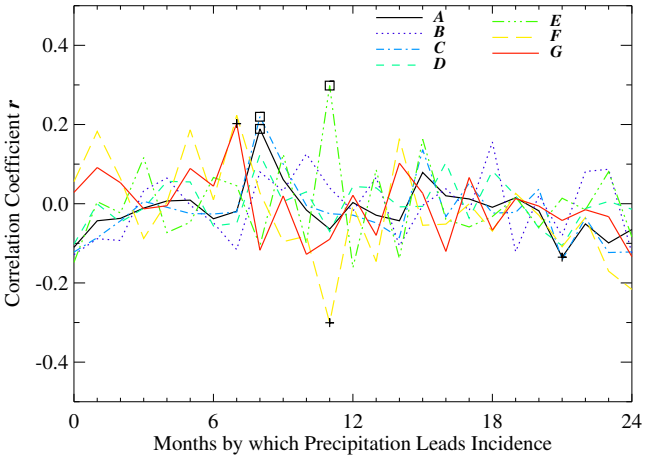


**Figure 5.** Lag correlation coefficient $r$ between valley fever incidence anomaly and precipitation anomaly $P^*$ for periods in Table 2. Plusses (+) and squares (□) indicate confidence statistics $p$ better than 5% and 1%, respectively.

pre-epidemic, epidemic, and post-epidemic time-series and analyze them separately. Table 2 describes the six subset time periods that we extract and analyze separately from the full time-series. We examine these subset time-series for significant changes in the correlations between precipitation and wind speed anomalies with valley fever incidence.

Figure 5 shows the linear correlation coefficient $r$ between the monthly valley fever incidence anomaly $N^*$ and the precipitation anomaly $P^*$ for the seven different periods enumerated in Table 2. (Series A, containing all available monthly data, also appears in Figure 3). The trends and phasing generally agree among the subset time-series. The highly significant correlation ($p < 0.01$) of wet anomalies with incidence anomalies eight months later appears in the entire 23 yr record (Series A) and from 1980–1995 (Series C). Examination of monthly time-series (not shown)

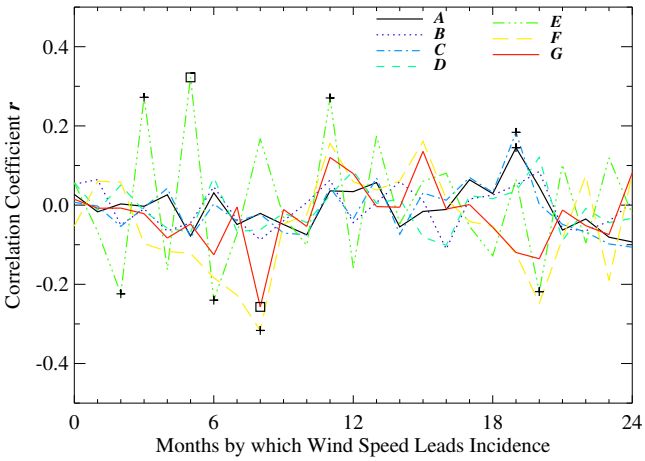


**Figure 6.** As in Figure 5, but for lag correlation coefficient $r$ between valley fever incidence anomaly and wind speed anomaly $U^*$.

reveals that winter (February–March) rains influence incidence the following winter (December–January), a pattern noted previously [Smith et al., 1946; Hugenholtz, 1957; Pappagianis, 1988; Jinadu, 1995; Kolivras and Comrie, 2003].

Since the epidemic, from 1996–2002 (Series E), precipitation anomalies occur 11 mos before incidence anomalies with $r = 0.30$ ($p = 0.0059$). Incidence during the 1991–1995 epidemic (Series F) shows no highly significant features. We note that the correlation required for a given confidence level is much greater for Series F due to its short length (60 months). The significant negative correlation with precipitation 11 mos prior may be a false positive since this would be inconsistent with Series E and with what is known presently of the lifecycle of coccidioidomycosis. On the other hand, Zender and Kwon [2005] show that dry anomalies in the previous rainy season are highly significantly associated with increased soil dispersion nine months later in many of Earth's dustiest regions. Hence increased incidence two months following increased dispersal is a plausible alternative explanation of the Series F behavior. Considered altogether, the indications of a significant connection between rainfall and incidence changes in epidemic years are unclear and somewhat contradictory.

Figure 6 shows the linear correlation coefficient $r$ between the valley fever incidence anomaly $N^*$ and the wind speed anomaly $U^*$ for periods in Table 2. Most of the statistically significant relationships of wind speed to incidence anomalies occur since the epidemic, from 1996–2002 (Series E). During this period, wind speed anomalies occur 5 mos before incidence anomalies with a correlation $r = 0.32$ ($p < 0.01$). However, the significant correlations

rapidly alternate from positive to negative during the first six months before incidence. Thus interpreting the significant wind speed anomalies as causally related to valley fever is problematic. Since 1991 (Series G) incidence anomalies occur eight months after opposite wind speed anomalies with $r = 0.42$ $(p < 0.01)$. This feature may be a cross-correlation artifact. As discussed above, winter rainfall anomalies dominate incidence anomalies with an eight month lag from 1980–2002 (Figure 5). Winds are slowest in winter (Figure 2c) and so may (coincidentally) anti-correlate with 1991–2002 monthly incidence anomalies, but not with the entire incidence dataset (Series A).

## 4. Discussion

Univariate analyses showed the maximum correlation between incidence and climate anomalies ($r = 0.19$ for $N^*$ following $P^*$ by 8 mos) explains only $r^2 = 4\%$ of monthly incidence anomalies. This is too low to be useful in practical associations between climate predictions and valley fever incidence. However, incidence anomalies $N^*$ are quite complex, and can reach 1000% in epidemic years—so predicting a small fraction of this large variability is promising.

Seasonal climate predictors of valley fever in Kern County are similar to, but much weaker than those in Arizona. Komatsu et al. [2003] examined climate and valley fever incidence for the four year span from 1998–2001 in Maricopa County, Arizona. They report highly significant ($p < 0.01$) correlations between incidence with seven-month cumulative precipitation ($r^2 = 0.75$), two-month cumulative precipitation, three-month mean temperature, and coarse aerosol (PM10) load.

The discrepancy between climate associations with valley fever in California and recent years in Arizona are not understood. The differences in climate, methodology, and sampling between our study and Komatsu et al. [2003] are significant, and possibly large enough to explain these discrepancies. In particular, late summer southwest monsoon rains occur in Arizona but not in California. This pattern is consistent with the stronger annual cycle of incidence in California.

We speculated that saltation-sandblasting, rather than direct wind entrainment, is the normal mechanism by which wind mobilizes and disperses soil pathogens such as *C. immitis*. Saltation-sandblasting theory improves predictive capability of long range mineral aerosol transport models [Grini and Zender, 2004] and so could improve dispersal modeling of airborne soil pathogens including *C. immitis*.

Changes in the Mojave habitat for *C. immitis* may be significant in the coming century. Significant increases in temperature and precipitation are expected in western North America as a whole over the next century [Giorgi et al., 2001]. Unfortunately regional scale projections are much more robust for temperature than for precipitation. In some scenarios [Electric Power Research Institute, 2003; Hayhoe et al., 2004], increased precipitation causes desert areas of Kern County and uplands of the Mojave Desert to transition to grassland by 2100. The Mojave is currently too dry to sustain *C. immitis* [Kolivras et al., 2001] because it receives $P < 10\,\mathrm{cm\,yr^{-1}}$, about half what Kern County receives. We speculate that the Mojave might become a suitable environment for *C. immitis* were it to moisten significantly for a prolonged period. A significant portion of the Los Angeles metropolitan region is downwind of the Mojave during Sana Ana wind events. Santa Anas occur most frequently (3–$4\,\mathrm{day\,mo^{-1}}$) in Nov.–Jan. [Raphael, 2003], coincident with peak valley fever incidence in Kern County. Given the public health-related implications, the Mojave Desert is a natural focus region for future valley fever habitat and dispersion modeling using down-scaled climate predictions.

## 5. Conclusions

We tested monthly precipitation, wind speed, and temperature anomalies as potential predictors for valley fever incidence anomalies in Kern County from 1980–2002. The only climate indicator with highly significant correlations with incidence during this period is the precipitation anomaly. Precipitation anomalies eight months antecedent to reporting explain only up to 4% of monthly variability in subsequent valley fever incidence. Wind speed anomalies five months antecedent are highly significantly associated with incidence since the epidemic, from 1996–2002. The relatively small correlation of incidence with climate suggests that anthropogenic factors (e.g., construction) may play a large role in valley fever outbreaks in Kern County.

None of the potential climate indicators of incidence that we tested are highly significantly correlated with the 1991–1995 epidemic. Other potential univariate climate indicators of incidence (e.g., accumulated seven-month precipitation, wind gustiness) and multi-variate climate indicators (e.g., drought index) may show more predictive skill than those we tested [e.g., Komatsu et al., 2003]. Seasonal climate predictors of valley fever in Kern County are similar to, but much weaker than those in Arizona, where previous studies find precipitation explains up to 75% of incidence. Causes for the discrepancy between climate associations with valley fever in California and Arizona require further study.

Incidence reports for 2001–2003 in Kern County show a significant increase above the long term background level unprecedented except for the 1991–1995 epidemic. Reliable predictors of incidence will be extremely valuable whether

or not current incidence rates continue to rise. Higher resolution temporal and spatial monitoring of soil conditions in Kern County may improve our understanding of climatic antecedents of valley fever epidemics.

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C. S. Zender, Department of Earth System Science, University of California, Irvine, CA 92697-3100. (zender@uci.edu)

J. Talamantes, Department of Physics and Geology, California State University, Bakersfield, CA 93311. (jtalamantes@csub.edu)

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