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Smallpox and Climate in the American Southwest

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For many decades anthropologists have assumed that native populations in the American Southwest were relatively unaffected by Spanish contact and by later intrusions of Hispanic and Anglo populations. In spite of the recorded occurrence of disease in census documents, smallpox, measles, influenza, and other European-introduced crowd infections were believed to have reached the Southwest only during the last 200 years. Consequently, estimates of contact-period populations as well as reconstructions of various groups' social, political, and economic organization generated between 1900 and 1960 were thought to mirror the past rather well. A growing list of arguments suggests the above situation is factually incorrect.

In this paper I focus on two important and interrelated issues relevant to this topic: (1) modeling the incidence and spread of disease using data from the quantitative epidemiological literature concerning smallpox, and (2) examining the effects of climate on the incidence and severity of epidemic smallpox. I show that there is a high probability that this disease spread to the American Southwest during the period before systematic Spanish exploration of the region (A.D. 1519–81).

The Disease Model

Several anthropologists have considered the effects of epidemic diseases on precontact- and contact-period aboriginal populations. Only two, however, have attempted to bring quantitative data from the field of epidemiology to bear on the problem of depopulation due to the spread of disease (Milner 1980; Ramenofsky 1982). Milner presents what is known as the Reed-Frost model (Sartwell 1976); Ramenofsky presents a modified version of the Hamer-Soper model (Hamer 1906; Soper 1929). Both models can be used to quantify the spread of an epidemic disease through a susceptible host population, and both are deterministic models, meaning that the future state of the epidemic process can be determined once the initial number of susceptibles and infectious individuals, as well as the attack, recovery, birth, and death rates are known (see Bailey 1975 for a full discussion of deterministic models).

All deterministic models assume that infection spreads through an independent isolated group that is subject to homogeneous mixing. The models assume further that once the individuals are infected with the disease, they, in turn, are immediately infectious. In other words, the models do not account for a period of latent infection before the onset of observable illness (Bailey 1975:81). This critical shortcoming makes the use of these models with New World contact-period demographic data highly questionable.

The initial iteration of such models assumes that 100% of a given population is susceptible to a particular disease. The assumption of 100% susceptibility can be seen in the initial set of differential equations,

$$dx/dt = -Bxy \quad (1)$$
$$dy/dt = Bxy - y'y \quad (2)$$
$$dz/dt = y'y \quad (3)$$

where x is the number of susceptibles, y the number of infectives in circulation, z the number of removals, B the infection rate, y' the removal rate, and t a specified time interval (Bailey 1975:82). Thus $x + y + z$ = all the members of a given community, while the differential equations describe changes in the values of x, y, and z (see Milner 1980 and Ramenofsky 1982

for a graphic portrayal of a deterministic model for general epidemics).

One major problem with using the equations for a general deterministic epidemic is finding values for B. To make use of equations 1–3, one must make certain simplifying assumptions. First, in determining various values for B one can assume that the infection rate, that is, the number of new cases appearing in a population during a particular time interval, equals either the contact rate or some fraction of the contact rate. In other words, the contact rate parameter equals the number of susceptibles contacted by an infective per unit of population per unit of time. With models assuming a fixed number of infectives, the contact rate can be figured simply as a function of the frequency of contact per unit of time. Thus, any infective coming in contact with a group of susceptibles will transmit the disease to the susceptibles, or to some portion thereof, making them infectives. One can further assume that these new infectives, after time interval t, will then transmit the disease to a new group of susceptibles at the same rate. Thus, the infection rate will be an ascending exponential curve, with the slope of the curve varying with the magnitude of person-to-person contact (Figure 1).[1]

Examination of Figure 1 reveals how quickly infection can spread in a susceptible host population given even the smallest contact rate parameter. Of course, the curves represented in Figure 1 are slightly unrealistic, since in most communities individuals have a more or less confined circle of contacts and rarely in a group of a specified size would all people contact n number of new individuals daily. Nevertheless, under certain nucleated settlement conditions, all individuals within a community of n size could be contacted within a very short period of time.

Although the primary use of most epidemiological models is to estimate the number of probable new cases of a disease before or during the actual outbreak of an epidemic, equations do exist for calculating mortality rates for particular diseases. This class of equations is derived from vaccination models, and although some assumptions must be made, they provide a useful way of estimating average total and average daily mortality. To determine average mortality for varying values of B, we can solve for $D(n)$, the average number of deaths due to a given disease (Bailey 1975:366) where:

$$D(n) = v\{1 + \frac{y' Bn}{w(y' - Bn)}\} \quad (4)$$

In this equation, v corresponds to the case mortality rate, y' to the mean infectious period before removal, B to the contact rate parameter, w to the mean duration of the infectious period, and n to the total population at risk. Using this equation, it is possible to calculate average mortality for different levels of disease recognition and periods of infectiousness, while controlling for community size and estimated levels of face-to-face interaction.

Smallpox: Ecology and Mortality

The pathology, virology, and clinical manifestations of smallpox are well known and have been discussed in detail by many medical researchers (Christie 1980; Fenner and White 1976; Horsfall and Tamm 1965; Rhodes and Van Rooyen 1962; Top and Wehrle 1972; Van Rooyen and Rhodes 1948). Table 1 presents the various terms used to describe the manifestations of Variola major and shows the associated mortality rates reported by different researchers for each strain of the virus. As can be seen, standard terminology has not been developed and there is no clear consensus regarding the severity (i.e., mortality rates) of the various forms of this virus.[2]

Variola Major: Infectivity and Mortality

The infectiousness and transmissibility of smallpox have been the subject of some recent debate (see Fenner and White 1976:325; Christie 1980:230). Most medical researchers agree that smallpox is spread as a droplet infection, with the site of entry being the upper respiratory tract. The infectivity of any one individual depends on the virus being released from the body, something that can only occur

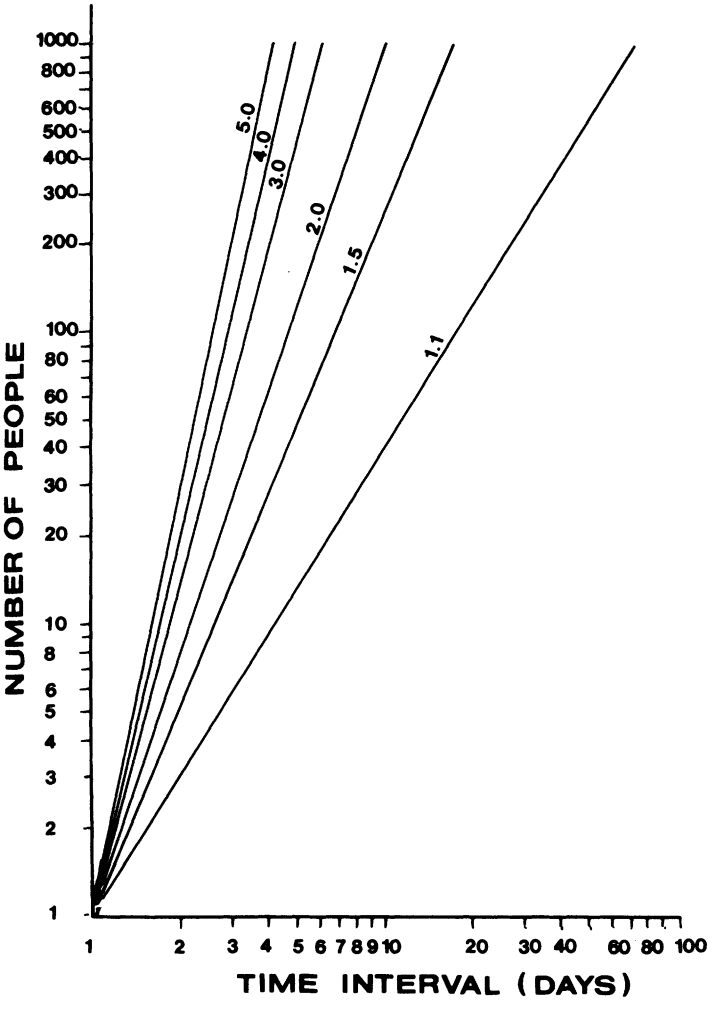

Figure 1
The spread of infection in a completely susceptible host population of 1,000 using different contact parameters.

when the virus breaks through either the skin or the mucous membrane of the upper respiratory tract. This generally occurs on or about the fourth day after the onset of illness. The incubation period of the virus before the onset of illness varies from 9 to 16 days. In other words, an individual becomes infectious between 13 and 20 days after the virus has been inhaled. At this point the virus is released into the air from the infected individual. The total infectious period can last a little more than three weeks (a mean of 26.75 days [Dixon 1962:297]) and terminates with either the patient's recovery or death. It has long been recognized, however, that the smallpox corpse is a potent and continuing source of infection.

The above data on the incubation and infectious period of smallpox allow for the solution of equation (4) for virgin-soil populations. Normally, solution of an equation such as this would require extensive knowledge about the effects of a particular disease on a given population and about patterns of community interaction. Fortunately, Becker (1979) has compiled just such information for the incidence of smallpox in the United States. All parameters and assumptions of the

Table 1
Variable terminology used to describe the manifestations of Variola major.

Researcher	Terms	Mortality
Beneson 1972	Ordinary type	30%
	Hemorrhagic type	100%
	Flat type	75% (vaccinated)
		96% (unvaccinated)
	Modified type	<5% (occurs only among vaccinated individuals)
Downie 1965	Classical smallpox	
	var. purpura variolosa	high mortality
	var. Variola postulosa haemorrhagica	high mortality
Van Rooyen and Rhodes 1948	Discrete smallpox	6%
	Confluent smallpox	45%
	Hemorrhagic smallpox	78%
	Purpuric smallpox	100%
Rhodes and Van Rooyen 1962	Discrete smallpox	10%
	Confluent smallpox	50%
	Hemorrhagic smallpox	>80%
	Secondary hemorrhagic smallpox	100%
Fenner and White 1976	Classical smallpox	15%
Christie 1980	Typical smallpox	variable
	Hypertoxic smallpox	
	var. hemorrhagic type	near 100%
	var. purpura variolosa	100%
	Modified smallpox	low
Dixon 1962	Fulminating smallpox	100%
	Malignant confluent smallpox	70%
	Malignant semiconfluent smallpox	23%
	Benign confluent smallpox	20%
	Benign semiconfluent smallpox	10%

model have been extended to virgin-soil conditions and pertain to the first exposure to smallpox in a completely susceptible host population. The parameters and assumptions of the model are listed below. Becker's (1979) remarks are italicized.

B = *"This contact rate parameter is very community dependent. No estimate was available. . . ."* I have substituted a variety of contact rates ranging from 1.1 to 5.0. The contact rate parameter is an exponential measure.

$y' = 2$. *"Although the period of infectiousness may be about three weeks, normal public health practices of contact tracing, early diagnosis, etc. considerably reduces the effective value of y'. The figure of $y' = 2$ corresponds to a mean infectious period before removal of 0.5 days."* I have assumed that in the first round of the disease in a virgin soil population, the mean period of infectiousness would correspond to the total duration of the infec-

tious period, or 26.75 days. To simplify the equation I have assumed that y' attains a maximum value of 100 for each 25-day cycle.

$v = 10^{-1}$. *"The case mortality rate [in the United States] is roughly 1 in 10."* In attempting to derive values for v, I have used the relatively conservative estimate of Crosby (1972), who suggests that mortality from smallpox in an unvaccinated population is approximately 30%.

$w = 0.2$. *"Based on the mean duration of the infectious period prior to discovery being five days."* In this equation, w is the decimal equivalent of y'. In other words, w attains a maximum value of 1.00 for each 25-day cycle.

$n = 2 \times 10^8$. *"Approximate population size of the U.S."* I have used a constant population size of 1,000 individuals for all solutions of the equation.

Using the values noted above for virgin-soil populations, nine solutions to the equation were generated using different contact rate parameters and varying values of y' and w. The solutions appear in Table 2. What is immediately apparent from the table is that slight variations in the contact rate result in dramatically different projected daily case mortality rates. Although the projected total number of deaths will always approximate 30% of the total population (since v = .30), the daily mortality from smallpox varies from 0.4% to 7.0% of the total population. The impact of variation in projected daily case mortality has enormous sociopolitical and economic implications for aboriginal populations, both in terms of immediate effects and long-term, transgenerational effects (see Lycett 1984).

Variola Major: Non-Host Transmissibility and Aerial Convection

It is generally believed that the normal mode of smallpox transmission is direct person-to-person contact. A good deal of data, however, suggest that aerial convection can and does occur under certain environmental conditions (Brachman 1970; Christie 1980:229–230; Morris et al. 1970; Thomas 1974; Wehrle et al. 1970). The notion of aerial convection is relevant to the present discussion, since the transmission of the smallpox virus *without* direct person-to-person contact has substantial implications for the spread of this disease in virgin-soil populations. Two variables are relevant for aerial convection: (1) the absolute quantity of smallpox virus present in the atmosphere and (2) the ambient conditions that favor the survival of the smallpox virus outside of the host.

Some researchers have attempted to determine the optimal climatic conditions that favor the persistence of Variola virus outside of a host (Downie and Dumbell 1947; Huq 1976; MacCallum and McDonald 1957; Mitra et al. 1974; Rogers 1926). Variola virus is notoriously stable and durable. Although the virus completely loses infectivity at a temperature of 39°C (102.2°F [Downie 1965:936]) and can be killed in approximately ten minutes by moist heat at 60°C (140°F [Christie 1980:228]), particular climatic conditions aid in the virus' survival. MacCallum and McDonald (1957) conducted experiments to determine how long Variola virus could survive in an infective state under different conditions of temperature and humidity. They found that at temperatures of between 20° and 24°C (53.8° and 75.2°F) and at a relative humidity of approximately 55% Variola virus survived in a highly infectious state for at least 18 months. The virus continued to be viable at the end of this period, but the experiment was terminated. Downie and Dumbell (1947) obtained similar results, finding that Variola virus isolated from scabs survived for 417 days at 20° to 22°C (53.8° to 71.6°F) in an environment with a relative humidity of 35% to 65% (see also MacCallum and McDonald [1957:248] for a description of this experiment).

These results have been augmented by the research of Huq (1976), who suggests that Variola virus remains infective for several weeks in ambient temperatures as high as 30°C (86°F) with a low relative humidity (ca. 25%–30%), although 30°C would seem to be the upper limit of the virus' infectivity. He also indicates that

Table 2
Iterations of the vaccination model per 1,000 population with an estimated 30% case mortality and variable contact parameters.

No. infected people	Contact parameter[a]	Standardized contact rate[b]	y'	No. days to contact 1,000	w	$D(n)$	Projected avg. no. deaths	Projected daily case mortality
1,000	1.1	.015177	290	72.476574	2.90	.309591	310	4
1,000	1.5	.088046	68	17.036621	.68	.309052	309	18
1,000	2.0	.200687	40	9.965784	.40	.306107	306	31
1,000	2.5	.331617	30	7.538825	.30	.303668	303	40
1,000	3.0	.477121	25	6.287710	.25	.302541	303	48
1,000	3.5	.634746	22	5.514016	.22	.301906	302	55
1,000	4.0	.802747	20	4.982892	.20	.301500	302	61
1,000	4.5	.979819	18	4.592686	.18	.301232	301	66
1,000	5.0	1.164950	17	4.292030	.17	.301036	301	70

[a]The contact parameter is based on an exponential contact rate so that x infected individuals each contact the same number of individuals in the population who, in turn, spread the infection at the same rate. Thus, at a contact parameter of 5.0, a population of 1,000 would be contacted in 4.292030 days ($5^{4.292030}$).

[b]The standardized contact rate is derived by dividing the contact parameter by the time interval necessary to contact the total population at risk. In this case the standardized contact rate = the contact parameter/days. The population at risk = 1,000.

under optimal climatic conditions Variola virus can remain infective for several years! Interestingly, Huq found that variation in temperature and humidity contributed to the loss of infectivity in the virus. In other words, in environments with temperatures between 22° and 30°C and with relative humidities between 25% and 55%, Variola virus remains stable and infective for a number of years. As temperature and humidity rise above 30°C and 55% respectively, Variola virus rapidly loses infectivity.

Unfortunately, laboratory research on the survivability of the smallpox virus has not been directed at determining the minimum temperature in which the virus can survive. Although there is no firm evidence, it would appear that 0°C (32°F) represents the lower limit of survivability.

Other modes of nonhost smallpox transmissibility have been identified by several researchers. These include the spread of infection from handling the clothing and bedding of infectives, ingestion of the virus contained in contaminated food, and contact with animals that have been in an infected environment. Smallpox can also be transmitted by flies that have come in contact with the secretions from skin eruptions. These data reinforce the findings relating to the survivability of smallpox outside the host and have substantial implications for the spread of infection in totally susceptible host populations under aboriginal conditions.

Smallpox and the Environment

In 1926, Sir Leonard Rogers completed a study correlating the effects of climate on the incidence of smallpox in India. Rogers's specific intent was to investigate how particular climatic conditions affected variation in the rate of death due to smallpox. At the time of Rogers's study, smallpox was endemic to most areas of the Indian subcontinent. Records charting the occurrence of smallpox indicated that epidemic spread of the disease followed a clear cyclical pattern, but one that was not apparently related to the introduction of new infectives into areas that were comparatively free of the disease. Instead, smallpox was occurring in endemic areas and appeared to be corre-

lated with climatic conditions. Rogers's analysis revealed strong relationships between the increased incidence of smallpox, deficient rainfall, and specific conditions of absolute humidity (Rogers 1926:21–22). Rogers portrayed these relationships graphically and also attempted to quantify the data using correlation coefficients. Although Rogers's analysis is complete and detailed, the periodicity of the data prevented him from specifying the precise relationships between epidemic smallpox and climatic conditions.

In an effort to resolve this problem and obtain data useful for predicting the ideal climatic conditions favoring the spread of smallpox, two regression analyses were performed. The first, a standard least squares multiple linear regression, revealed several interesting relationships (see Table 3). Most important, the analysis reveals time to be the most important of the variables. This result is not surprising, since the climatic data as well as death rate are all time-dependent variables. More important, however, the initial regression analysis shows that absolute humidity, Rogers's explanatory variable, is poorly correlated with the dependent variable. Mean temperature and relative humidity are much more highly correlated with death rate. Although absolute humidity is a composite variable that measures both temperature and relative humidity, it does not appear on the basis of these results that Rogers's conclusion is completely warranted.

Because the obvious cyclical nature of Rogers's data and the problem of autocorrelation in the linear regression analysis (Durbin-Watson = 0.618117), another regression analysis was undertaken in which the data from the seven Indian provinces were averaged, and periodic terms for the variable of time were added to the list of independent variables (see Mendenhall and Reinmuth [1978:542] for a discussion of sinusoidal models). Results of the regression analysis are presented in Table 4.

The sinusoidal least squares model provides an excellent approximation of the relationships between the climatic

Table 3
Multiple linear regression on Rogers's raw data.

Dependent variable: Death rate

Coefficient of determination: 0.372218
Multiple correlation coefficient: 0.610097
Estimated constant term: −4.71234
Standard error of estimate: 1.94949

Analysis of Variance for the Regression

Source of variance	DF	Sum of squares	Mean of squares
Regression	5	175.762	35.1523
Residuals	78	296.439	3.8005
Total	83	472.201	

F Test: 9.24941, $p = .001$

Variable	Regression coefficient	Standardized coefficient	Correlation with dependent
Month	−0.373610	−0.543967	−0.552864
Mean temp.	0.143567	0.548198	0.215949
Rainfall	−0.003336	−0.007937	−0.056590
Relative humid.	0.049340	0.331762	−0.149164
Absolute humid.	−4.980530	−0.455765	−0.004682

Durbin-Watson = 0.618117

Table 4
Sinusoidal least squares regression with periodic terms and averaged data.

Dependent variable: Death rate

Coefficient of determination: 0.997626
Multiple correlation coefficient: 0.998812
Estimated constant term: −4.195690
Standard error of estimate: 0.146549

Analysis of Variance for the Regression

Source of variance	DF	Sum of squares	Mean of squares
Regression	7	36.09070	5.15582
Residuals	4	0.08590	0.02147
Total	11	36.17660	

F Test: 240.065, $p = .001$

Variable	Regression coefficient	Standardized coefficient	Correlation with dependent
Month	−0.019800	−0.039370	−0.718801
Mean temp.	0.102155	0.457918	0.316324
Rainfall	−0.368187	−0.890930	−0.300189
Relative humid.	0.175183	0.898977	−0.792864
Absolute humid.	−14.69940	−1.430240	−0.214501
Cosine	−4.222280	−1.719120	−0.529440
Sine	0.154838	0.063070	0.837536

Durbin-Watson = 2.81847

measures and death rate. The periodic model again indicates that the climatic variables of mean temperature and relative humidity are the strongest determinants of changes in the rate of death due to smallpox. It is also significant that mean temperature is positively correlated with changes in the death rate and relative humidity is negatively correlated. Thus, an increase in the death rate would be correlated with increasing temperature and decreasing humidity.

Using the regression coefficients from the sinusoidal model, a series of estimates have been generated that portray the relationship between smallpox death rate and mean temperature and relative humidity (see Table 5). Only three variables from the regression analysis were utilized in computing these estimates: month, mean temperature, and relative humidity. The estimates confirm the inverse relationship between temperature and humidity, with maximum death rate occurring with maximum mean temperature and minimum relative humidity.[3]

In the seven Indian provinces, the mean annual death rate is 4.02 ± 2.38 and is correlated to a mean annual temperature of 77.5°F and a mean relative humidity of 67.3%. In other words, any values for death rate that exceed this mean that are within the climatic parameters that have been specified would suggest climatic situations that favor the spread of smallpox. To provide a conservative estimator, we can add the standard deviation to the mean death rate for the Indian provinces. Thus, death rates higher than 6.40 indicate the most favorable climatic conditions. Interpreted in this manner it can be seen that relatively specific climatic conditions favoring the survivability of the smallpox virus can be specified.

Smallpox and the American Southwest

It is important to note at this point that direct application of the disease model presented earlier in this paper is not pres-

Table 5
Estimates of death rate generated from the sinusoidal regression analysis.

Mean annual temp (°F)	Mean annual relative humidity (%)									
	10	20	30	40	50	60	70	80	90	100
10	Loss of infectivity at 32° F									
20										
30	**7.84**	**6.75**	5.67	4.58	3.50	2.41	1.33	.24	—	—
40	**8.54**	**7.46**	6.37	5.29	4.20	3.12	2.03	.94	—	—
50	**9.25**	**8.17**	**7.08**	5.99	4.91	3.82	2.74	1.65	.57	—
60	**9.96**	**8.87**	**7.79**	**6.70**	5.62	4.53	3.45	2.36	1.28	.19
70	**10.66**	**9.58**	**8.49**	**7.41**	6.32	5.24	4.15	3.07	1.98	.89
80	**11.37**	**10.28**	**9.19**	**8.11**	**7.03**	5.94	4.86	3.77	2.69	1.60
90	Loss of infectivity at 86° F									
100										

Note: Figures in boldface indicate optimal climatic conditions favoring the survivability of the smallpox virus.

ently possible. Accurate demographic data and information on patterns of community interaction for the Southwest are simply not available for the time periods in question. Nevertheless, there are data on the contact history of the Southwest that indicate smallpox could have been transmitted to native populations before systematic investigation by the Spanish. Additional data suggest that climatic conditions were ideal for the survival of Variola virus outside of the host. If the following scenario is correct, then the kinds of mortality rates provided by the disease model can be used to account for population loss among native southwestern groups during the contact period.

Like most other kinds of historical information, the evidence for epidemic smallpox in the American Southwest before A.D. 1800 is sketchy. The most complete treatment to date regarding the spread of smallpox in the New World has been offered by Dobyns (1983). Although his work is concerned with the effects of a variety of diseases on virgin-soil populations, smallpox is given the most detailed treatment. One of Dobyns's most controversial assertions, and one that certainly seems plausible given the data presented in this paper, is that a hemispheric pandemic swept New World populations in the years between A.D. 1520 and 1524. The foundation of this pandemic according to Dobyns was smallpox, although other European-introduced diseases were also certainly prominent.

Apart from Dobyns's assertion regarding the hemispheric pandemic, the evidence for the presence of smallpox in the

greater Southwest is remarkably poor before 1780. Although Dobyns argues that epidemics of smallpox occurred in Sinaloa in 1592–93 and again in 1602, it is unclear whether the disease in question was actually smallpox (see Dobyns 1983:28–29; Gibson 1964:449; Sauer 1935). Thus, the first unequivocal occurrence of epidemic smallpox in the Southwest was in 1780–81 (Bancroft 1889:266; Stearn and Stearn 1945:48), 264 years after the disease had been introduced to the New World and 242 years after the Spanish first entered the Southwest. Following 1780–81, epidemics of smallpox occurred in the Southwest about every 18 years (± 4.96) up to the beginning of the 20th century (Dobyns 1983:28–29), or about once every generation.

There is a remarkable discrepancy in the disease history of the American Southwest. In virtually every other region of the New World where populations were contacted by the Spanish, epidemics of smallpox followed contact almost immediately. If we are to place our faith in the records of Spanish contact, however, populations in the Southwest somehow escaped the effects of diseases like smallpox until well into the 18th century. This seems highly unlikely. The first European contact with southwestern groups occurred in 1539 (Marcos de Niza). The Spanish did not visit the Southwest between 1540 (Coronado) and 1581 (Rodriguez-Chumascado). Although there is no record of epidemics during this time, it is reasonable to assume that southwestern populations were no different from other New World groups in their lack of resistance to diseases brought by the Spanish.

It should be remembered that during the interval between 1520 and 1581, the Spanish were only one of several potential disease vectors that could have transmitted smallpox to populations in the Southwest. Riley (1980, 1982) has described in some detail the kinds of linkages that existed between native populations throughout the greater Southwest between 1520 and 1650. Many of these linkages were economic and involved the exchange of goods and people. What is more important, however, is that potential disease vectors were introduced to the Southwest in 1540. Mexican Indians, presumably Tlaxcalans, who accompanied the Coronado party in 1540 remained in the Southwest and were identified by the Spanish some 40 years later (Riley 1974:30–32, 1982). These Indians had been exposed to the Old World diseases, particularly smallpox, for nearly 20 years before arriving in the Southwest. Thus, although the evidence is purely inferential, the 40-year hiatus in Spanish documents would have provided ample time for a smallpox epidemic to have occurred.

Southwestern Climate and Smallpox

As noted earlier in this paper, the incidence and severity of smallpox is highly correlated with particular climatic conditions; conditions favoring the survivability of the smallpox virus outside of the host can be specified (see Table 5). Table 6 presents generalized mean annual temperature and relative humidity data for various elevations in the greater Southwest. The data were derived from a regression equation in which elevation was predicted on the basis of mean temperature and relative humidity.[4]

It is immediately apparent that the mean annual conditions of temperature and relative humidity fall within the optimal range of climatic conditions that favor the survivability of the smallpox virus outside of the host. In fact, for elevations beween sea level and 6,000 ft, mean temperature and relative humidity in the Southwest might be described as ideal. What these data do not show is the variation in mean temperature and relative humidity, something that is critical to the virus' survival. An examination of monthly mean temperature data for the region indicates that for elevations below 1,500 ft there are likely to be three months, on the average, with temperatures that exceed 86°F. Similarly, elevations above 7,000 ft are likely to have an average of three months with temperatures below 32°F. In other words, areas in the Southwest between about 2,000 and 6,000 ft provide the most stable climatic

Table 6
Generalized climatic data by elevation for the Southwest.

Elevation (ft.)	Mean annual temperature (°F)	Mean annual humidity (%)
Sea level	74.0	30.7
200	73.3	30.9
400	72.5	31.2
600	71.7	31.5
800	70.9	31.8
1000	70.2	32.1
1500	68.4	32.7
2000	66.5	33.4
2500	64.6	34.1
3000	62.7	34.9
3500	60.8	35.6
4000	58.9	36.3
4500	57.0	37.0
5000	55.1	37.7
5500	53.2	38.4
6000	51.4	39.1
6500	49.5	39.8
7000	47.6	40.5
7500	45.7	41.2
8000	43.8	41.9
8500	41.9	42.6
	error = 2.4°	error = 1.6%

conditions for smallpox, although daily fluctuations in summer and winter temperatures, particularly at the lowest and highest elevations in this range, may also hinder the virus' survival.

These data suggest that once smallpox entered the Southwest, climatic conditions would favor its continued survival. They also indicate that modes of disease transmission other than direct person-to-person contact probably occurred. If climatic conditions in the Southwest are so favorable for smallpox, why didn't the disease become endemic to the area? The answer to this question must lie in the size of the reserve population following the introduction and spread of smallpox. Small numbers of susceptibles following an epidemic may have effectively limited the extent to which the disease could become entrenched in the population.

A Probable Scenario

My data strongly indicate that all of the conditions that existed in the Southwest during the early contact period—a completely susceptible host population, the introduction of potential disease vectors, and the conditions of climate—favored the spread of epidemic smallpox. Because of both this situation and the lack of textual information pertaining to the occurrence of smallpox in the Southwest before 1780, I offer a likely scenario for the spread of this disease to southwestern groups.

The hemispheric pandemic described by Dobyns probably affected populations living in the Southwest. The repeated textual references to epidemic smallpox in the Southwest after 1781 indicate that it is likely this disease reoccurred after the initial pandemic. A likely date for another reoccurrence of smallpox in the Southwest is in the interval following the departure of Coronado in 1541 and before the 1581 visit by the Rodriguez-Chumascado expedition. It is probable, given the frequency of later smallpox attacks in the Southwest, that at least one other epidemic occurred between the 1580s and the 1780s.

These three postulated occurrences of smallpox accord well with what little is known about the size of southwestern populations during this period. Elsewhere (Upham 1984) I have provided a revised population estimate for the Western Pueblo that is tied to a baseline date of 1520. The figures for the three groups of pueblos that I considered total 66,967 at this time (29,305 for the Hopi villages, 24,662 for the Zuni villages, and 13,000 for Acoma). Given a 30% mortality rate for each occurrence of smallpox, the spread of the initial pandemic in 1520 and another smallpox epidemic occurring in the 1541–81 interval would leave a total remaining Western Pueblo population of approximately 32,813 (14,359 for Hopi, 12,084 for Zuni, and 6,370 for Acoma). These figures are very close to what one would predict for these groups based on Dobyns's 20:1 depopulation ratio (see Dobyns 1966 and Upham 1982), an admittedly conservative estimator.

One of the first reliable population estimates for a Western Pueblo group was made in 1745 by two friars (cited in Donaldson 1893), who recorded 10,848 inhabitants in the Hopi villages. The postulated third unrecorded occurrence of smallpox after 1583, probably sometime in the 1600s and possibly related to one of the epidemics in Sinaloa cited by Dobyns, would result in a total remaining population at Hopi of 10,051 individuals, a figure almost identical to the estimate made by the two friars.

The above scenario is speculative and based only on the etiology of smallpox, inferential data regarding the spread of the disease, and likely routes of contact to the Southwest. In my view, the data are compelling and at the very least require renewed attention into the important issue of population reduction following Spanish contact.

Conclusion

The consideration of smallpox has provided a largely inferential way to assess how this disease may have affected southwestern populations during the contact period. There are several reasons why I have prepared this argument, with the emphasis on disease and consequent population decimation. First and most important is the widely held notion among southwestern anthropologists that southwestern populations were extremely small during the protohistoric and early contact period. Elsewhere I have addressed why I believe this idea has persisted and why it is wrong (Upham 1982, 1984). Second, the size of native populations in the Southwest is directly related to the kinds of interpretations of sociopolitical and economic systems that are generated. Ethnographic interpretations of the Pueblo, for example, have all been based on the study of small, relatively autonomous groups that survived the contact period and periods of Hispanic and Anglo domination. Many of these interpretations have been extended to the prehistoric period without consideration of the possible effects of Spanish contact and population reduction. Arguments presented here would indicate that the size and organizational complexity of populations recorded ethnographically are very poor analogs for periods earlier than about 1850.

Given the infectivity of smallpox and the climatic conditions in the Southwest, the introduction of infection to one group could have resulted in widespread transmission of the pathogen. If this occurred before 1540 or between 1541 and 1581, it is likely that no written record of the epidemics exists. Consequently, model building such as that offered here coupled with archeological research on sites dated to these intervals will provide the only sets of information that might resolve the important issue of disease and the decimation of native southwestern populations.

Notes

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[1]I have used a constant population size of

1,000 for all calculations presented in this paper. The relatively small population size is intended to approximate conditions of mortality as they might have existed in the protohistoric Southwest. In most applications of quantitative epidemiological models a base population size of at least 100,000 is customary.

[2]It should also be remembered that all of the data pertaining to mortality from smallpox listed in Table 1 are from areas where the occurrence of smallpox had a long history. Consequently, mortality figures should be viewed as indicative of how serious the disease was under certain circumstances in vaccinated and partially immune populations, not as ironclad assessments of how a particular strain of Variola virus affected all populations.

[3]Although the figures for estimated death rate are based on projections that are outside the parameter values of the regression equation, it is felt that the low standard error of the estimate (0.146549) allows for this kind of extrapolation. It should be remembered that these data were compiled in an area where smallpox was endemic and where contact tracing and vaccination programs were well organized. Because of this fact, the values should not be interpreted as direct measures; they should instead be considered as indices reflecting the kinds of climatic conditions that favor the spread of smallpox.

[4]Data from 22 weather stations at a variety of elevations in Arizona and New Mexico were used in the regression analysis. Because no data were available for climatic conditions below 32° latitude, predictions are less certain for areas south of the international boundary. Nevertheless, the data do provide generalized characterizations of climatic conditions at varying elevations for much of the greater Southwest.

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Language, Memory, and Focality: A Reexamination

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The relationship between language and cognition has often been explored using color stimuli to test what is commonly