

AVIAN DISEASE IN THE CENTRAL VALLEY OF CALIFORNIA

A Survey of Trends from 1980-2001



AVIAN DISEASE IN THE CENTRAL VALLEY OF CALIFORNIA:

A Survey of Trends from 1980-2001

Prepared for:

Central Valley Habitat Joint Venture
2800 Cottage Way, Suite W2606
Sacramento, CA 95825

Prepared by:

Robert Eddings
University of California Davis
Department of Wildlife, Fish and Conservation Biology
Davis, CA 95616

&

John Eadie (adviser)
University of California Davis
Department of Wildlife, Fish and Conservation Biology
Davis, CA 95616

March 31, 2003

**AVIAN DISEASE IN THE CENTRAL VALLEY OF CALIFORNIA:
A Survey of Trends from 1980-2001**

Robert Eddings

John M. Eadie, adviser

University of California Davis, Department of Wildlife Fish and Conservation Biology
Davis CA, 95616

INTRODUCTION:

Disease has long been known to be an important source of mortality for waterfowl. Avian botulism has been recorded in the western states back to the early 1900's. Avian cholera has been a more recent concern, first appearing in the United States in 1944 and spreading rapidly across major waterfowl migration routes in recent years (Baldassarre and Bolen 1994). Both diseases have caused outbreaks in which over 100,000 birds have died, and outbreaks of several thousand are fairly common.

Avian botulism is caused by the ingestion of a neurotoxin produced by the bacterium, *Clostridium botulinum* Type C. This bacterium is present in many wetland soils and can persist for many years in unfavorable conditions (Wobeser et al. 1987, Sandler et al. 1993, Marion et al. 1983). *C. botulinum* is generally found in a resting spore state that is harmless to waterfowl. Under certain conditions these spores germinate and become vegetative. It is during the vegetative state that these bacteria are capable of producing the neurotoxin. The toxin can only be produced under certain conditions that are usually associated with high ambient temperatures and low oxygen concentrations (Smith 1979, Barras and Kadlec 2000). Animal protein, often provided through decaying carcasses, is also a necessary ingredient for the production of the toxin (Bell et al. 1955). Animal carcasses result when high waters flood insect colonies and other animals and then subsequently strand fish when the water recedes (Mays 1940, Parish and Hunter 1969, Stribling and Harper 1969, Hines 1973, Beam 1978). The receding water leaves shallow areas that facilitate the warming of the water and reduces the oxygen concentration. For these reasons, botulism is usually seen in late spring through fall. The toxin is thought to be transmitted between birds through the ingestion of maggots and other invertebrates that have fed

on or near infected carcasses (Reed and Rocke 1992, Duncan and Jensen 1976). These birds then die and create more carcasses, leading to increased toxin production and an escalating cycle of mortality (Reed and Rocke 1992).

Avian cholera is also caused by a toxin produced by a bacterium, *Pasteurella multocida*, although the cycle and dynamics of cholera are less well understood than those for botulism. Because the bacteria has not been isolated from soils long after outbreaks (Price and Brand 1984, Bredy and Botzler 1989, Backstrand and Botzler 1986) it is thought that birds may be carriers of the disease and act as a reservoir from year to year (Samuel et al. 2002). When these carrier birds become stressed by environmental factors or physical factors, they may succumb to the disease and initiate an outbreak (personal communications with biologists). Overall, little seems to be known about the specific causes or triggers of cholera. Several studies have examined factors such as water quality, ion concentrations, lead intoxication, and temperature (Hunter 1978, Smith and Higgins 1990, Gordus 1993, Combs and Botzler 1991) but we have yet to obtain a good understanding about the dynamics of avian cholera outbreaks.

Disease is probably the single largest source of non-hunting mortality in waterfowl populations. Most current disease control is reactive once an outbreak occurs, and typically involves the collection and disposal of carcasses with the hope of reducing or eliminating sources of toxin. Recent studies in the Canadian prairies have questioned the effectiveness of such programs and, indeed, pick-up efforts are no longer being pursued in some areas (R. Clark, CWS, pers. comm). An alternative approach has been to focus on habitat availability. With the loss of wetland habitat over the last 150 years, birds have become increasingly concentrated into smaller areas, potentially increasing the chance of diseases transmission and leading to more frequent disease outbreaks that are more devastating (Friend 1981, Friend 1992, Smith and Higgins 1990). Accordingly, efforts to increase the amount of wetland habitat may lead to reduced concentrations of birds and, hence, a lower risk of disease outbreaks.

During a meeting in March 2002 between the United States Fish and Wildlife Service (USFWS) and the California Department of Fish and Game (CDFG) the need for a comprehensive survey of waterfowl disease in the Central Valley of California was expressed. The goal of this study was to survey the trends in avian botulism and avian cholera within the Central Valley, and specifically to evaluate the effect of recent habitat restoration efforts within the valley on waterfowl disease mortality.

METHODS:

We surveyed disease and wetland acreages in the Central Valley for the years 1980-2001 (22 years). We initiated our study by sending a survey form (Appendix A) to all National Wildlife Refuges and State Wildlife Areas within the Central Valley to acquire annual disease mortalities of waterfowl and annual wetland acreages. All refuges that were contacted are listed in Appendix B. Disease numbers are reported for the biological year, from June through May the following year. We collected total reported mortalities, not estimated mortality, except where unavoidable. Disease mortalities were collected for botulism and cholera separately. To supplement these data, we also obtained annual disease loss reports from both the USFWS and CDFG when available. We cross-referenced all reports to assure consistency, and filled in data gaps where possible. Wetland data were collected in total wetland acres available per year. We initially attempted to separate these acreages into seasonal and permanent wetlands, but that was not feasible for any scale larger than the individual refuges in this study. Further wetland estimates were obtained through lists of WRP easements provided by the Central Valley Habitat Joint Venture. We also obtained wetland trends and some acreage data from a survey of private wetlands in the Tulare Basin (Jones and Stokes 1988). Rice fields (other than those that were part of refuges) were not considered for this study due to the lack of disease monitoring on them. This should be addressed in future research.

In addition to wetland acreages, we also collected a series of weather variables from a University of California weather station for each region from an online database. Our main goal was to control for temperatures and water supply differences from year to year. We were unsure as to which variables would be more appropriate so we collected a series of variables. List of variables and descriptions collected for each disease are listed in Appendix C. We also obtained the midwinter counts from CDFG as an index of waterfowl populations. We did not calculate bird density because that would indicate equal bird use of wetlands, and would suggest habitat equality between all wetlands. Instead we used total population estimates to infer waterfowl supply to give an estimate of total possible mortalities. This does not assume any type of use pattern or quality of habitat; rather, it simply allowed us to control for variation in waterfowl population sizes among years and regions while evaluating the influence of total habitat availability.

All data were consolidated into four regions of the Central Valley that have different wetland characteristics and dynamics: Sacramento Valley, Suisun Marsh, Grasslands (the Upper San Joaquin Valley) and Tulare Basin (the Lower San Joaquin Valley). We performed analyses at the valley-wide scale and at the regional scale.

We used an information theoretic approach, based on Akaike's Information Criterion, to evaluate different a priori models of the factors influencing the occurrence of each disease (Anderson et al. 2000). All models considered four main variables: wetland habitat acreage, bird populations, a water variable and a temperature variable, alone and in combination. Several measures were available to index water availability and temperature (see Appendix C). To reduce redundancy and to choose the best measure in each case, we first ran a subset of models for all water variables and all temperature variables, respectively, for each disease. We then used the variable (or combination) with the lowest AIC in all subsequent analyses. Maximum likelihood estimates were determined from the residual sum of squares derived from regression analysis (Burnham and Anderson 1998:17) and second order AIC values (AICc) for small samples, delta AICc, Akaike weights and the cumulative weights of evidence for each variable were calculated according to Burnham and Anderson (1998). We used the MINITAB 11.21 statistical package for all regression analyses.

We conducted three sets of analyses for each disease. First, we analyzed the data on the valley-wide scale to detect any large-scale trends among years ($N = 22$) for the entire valley. For this analysis, we totaled all wetland acreages, disease mortalities by each disease, population estimates, water supply variables, and averaged temperature variables across all regions. We calculated AIC statistics for all models for cholera and botulism using annual total wetland acreage, total annual disease mortality, total population estimates, and the single best temperature and water supply model for each, for a total of 15 models for each disease. We followed a similar procedure for our second analysis, although here, we separated each region and treated each as an independent observation ($N = 4$ regions \times 22 years each = 88). This approach allowed us to consider not only valley-wide trends, but also any interregional trends. All AIC statistics were again calculated for all possible models including wetland acreage, population estimates, temperature, and water supply variables for each disease across all regions for a total of 15 models for each disease.

Finally, we conducted a last set of analyses for each individual region that had significant mortalities (2 regions for each disease). These last analyses allowed us to focus on the patterns within a specific region to determine whether wetland acreage or any other variable, influenced the occurrence of each disease within that region alone. By using a hierarchical series of analyses (valley-wide, inter-regional and intra-regional), we provide an assessment of the degree to which patterns of botulism or cholera are consistent at different spatial scales.

RESULTS:

Trends in Botulism and Cholera

The summary of botulism and cholera outbreaks is shown in Figure 1. Large botulism outbreaks were seen in 1980, 1982, 1983, 1987, 1997, and 1998. Large cholera outbreaks were seen in 1983, 1987, 1994, and 1996-1999. There are several of these years that overlap; 1983, 1987, 1997, and 1998.

We found a large variation in disease mortalities between the four regions. The distribution of botulism and cholera mortalities are shown in Figures 3 and 4. The Sacramento Valley (61,735) and Tulare Basin (88,575) had almost 100% of the total botulism mortalities (Fig. 3). The Grasslands had very few mortalities (166), and the Suisun Marsh had none. Cholera was also distributed unevenly; Sacramento Valley (96,566) and the Grasslands (51,765) contained 99% of the total mortalities during our study (Fig. 4). Tulare Basin had virtually no reported cholera (96), and the Suisun Marsh had only 1,246 mortalities reported.

Patterns of Disease and Wetland Acreage

The relationships between total wetland acreage and both diseases are shown in Figure 2. Although very weak, both diseases show an increasing trend with increased acreage. We also wanted to compare differences in trends between regions. We graphed annual botulism mortalities against Total Wetland Acreage for each region in Figure 5 to compare between regions. The trend line shows a negative relationship with acreage between regions. Cholera, when graphed in the same manner (Fig. 6) also shows a slight negative trend.

We also looked at each individual region to examine intraregional trends. We only looked at the regions that had large mortalities for each disease (Figures 3 and 4). In the Sacramento Valley acreage shows a slight negative impact on botulism as shown in Figure 7. In the Tulare basin, however, acreage shows a stronger positive effect on botulism mortalities.

When cholera was plotted against acreage in the Sacramento Valley, it shows a slight positive effect (Fig. 9). This is also the case in the Grasslands, as shown in Figure 10.

Model Selection for Botulism and Cholera

(a) Valley-wide scale

The results of the analysis of the occurrence of botulism across the entire valley using AIC are presented in Tables 1 and 2. The best model (lowest AICc) included only water availability (spring/summer precipitation), while the second best model included water and temperature (Table 1). The Akaike weights (w_i) for both models are low (0.498 and 0.145) suggesting a low level of confidence in the model. The best model accounted for 29% of the variation in botulism, while the second best accounted for only slightly more (30.8%). Calculation of the cumulative weights of evidence support (Table 2), a measure of the degree of confidence in the effect of each variable on the occurrence of botulism, indicate that only water availability has a strong effect on botulism outbreaks (Table 2). The regression equations for the best models are:

- i. $Botulism = -1965 + 4591 (Spring/Summer\ Precipitation)$
- ii. $Botulism = 25470 + 4660 (Spring/Summer\ Precipitation) - 582 (Summer\ Min)$

The results of the analysis for cholera across the entire valley are presented in Tables 3 and 4. The best model included wetland acreage while a second model that included only winter maximum temperature differed by only 1.69 in the AICc value. The model with only acreage had somewhat higher Akaike weight (.337) relative to that with only temperature (.145), but again, the low weights suggest that a low level of confidence in the models. This is reflected in the fact that the best model accounted for only 13.3% of the variation in cholera across the valley, while the second best accounts for 6.4% (Table 3). The cumulative weights of evidence support (Table 4) show that acreage and water have the greatest effect on cholera mortalities. The regression equations for the best models are:

- i. $Cholera = -1 + 123 (Fall/Winter\ Precip)$
- ii. $Cholera = -13674 + 0.121 (Total\ Acreage)$

(b) Inter-regional scale

To examine trends between regions, we next divided the valley into four regions and treated the data for each region as independent data points. Our analysis results for botulism at this scale are reported in tables 5 and 6. Our best model, that includes acreage and water, only

has an Akaike weight of .332, and an R-squared of .351. Our best two model regressions are as follows:

- i. $Botulism = 3327 - 0.0830 (Total\ Acreage) + 983 (Spring/Summer\ Precip)$
- ii. $Botulism = 2800 - 0.0826 (Total\ Acreage) + 970 (Spring/Summer\ Precip) + 0.000551 (Midwinter\ Pop)$

The cumulative weights of evidence support (Table 6) show that Spring/Summer Precipitation and Total Acreage have the largest effect on mortality with weights of 0.916 and 0.809 respectively.

Our analysis results for cholera at this scale are presented in tables 7 and 8. Our best two models both include both Total Acreage and Midwinter Population. Our best model, Total Acreage and Midwinter Population, has an Akaike weight of .463 and an R-squared of 0.205. The cumulative weights of evidence support (Table 8) show that Midwinter Population, Run-off & Precipitation and Total Acreage all had high support with 0.994, 0.859 and 0.732 respectively. Our best two predictor-models are:

- i. $Cholera = -1621 + 0.0493 (Total\ Acreage) + 0.0127 (Midwinter\ Pop)$
- ii. $Cholera = 2789 + 0.0465 (Total\ Acreage) + 0.00122 (Midwinter\ Pop) - 58.4 (Winter\ Max)$

(b) Intra-regional scale

We finally analyzed each of the regions mentioned earlier that had large numbers of mortalities for each disease. The results for botulism in the Sacramento Valley are reported in tables 9 and 10. Our top two models included Summer Minimum, and Summer Minimum and Spring Summer Precipitation respectively. These both had low Akaike weights (0.203 and 0.16) and R-squares (0.119 and 0.215). The cumulative weights of evidence support (Table 10) shows that Spring/Summer Precipitation has the largest effect with an Akaike weight of 0.823. Our best two predictor-model regressions are:

- i. $Botulism = 21857 - 401 (Summer\ Min)$
- ii. $Botulism = 20971 + 1241 (Spring/Summer\ Precip) - 363 (Summer\ Min)$

Our results for botulism in the Tulare Basin are shown in tables 11 and 12. Our best model includes Total Acreage and Spring/Summer Precipitation and has an R-squared of 0.718 and an Akaike weight of 0.620. These two variables also had very large cumulative weights of evidence support (Table 12). Our best two predictor model regressions are:

- i. $Botulism = -10371 + 0.494 (Total\ Acreage) + 2108 (Spring/Summer\ Precip)$

ii. $Botulism = -9536 + 0.421 (Total\ Acreage) + 2010 (Spring/Summer\ Precip) - 0.00204 (Midwinter\ Pop)$

Our results for cholera in the Sacramento Valley are reported in tables 13 and 14. The best model includes Winter Maximum with an Akaike weight of 0.232 and our second best includes Midwinter Population with weight of 0.199. Both models have low R-squares (0.048 and 0.034). The cumulative weights of evidence support (Table 14) show that no variable has a strong effect on cholera mortalities. Our best two predictor-models are:

i. $Cholera = 21937 - 245 (Winter\ Max)$

ii. $Cholera = 7176 - 0.00107 (Midwinter\ Pop)$

Our best model that includes acreage is the third best and is:

iii. $Cholera = 1899 + 0.0576 (Total\ Acreage)$

Our results for cholera in the Grasslands are presented in tables 15 and 16. Here, our best model includes only Total Acreage with an Akaike weight of 0.317 and an R-squared of 0.106. Our best two predictor-models are:

i. $Cholera = -7649 + 0.195 (Total\ Acreage)$

ii. $Cholera = 875 + 0.00233 (Midwinter\ Pop)$

The cumulative weights of evidence support (Table 16) show that although no variable has a very big effect on mortality, Total Acreage has the largest with 0.546.

DISCUSSION:

Disease and Acreage

If assumptions that increased habitat availability would decrease bird density and therefore decrease the chance of birds becoming infected with disease, we should see a negative correlation between total wetland acreage and disease mortality. The graph of botulism and cholera mortalities against acreage in the Central Valley (Fig. 2) shows the opposite trend for both diseases. However, trying to reliably determine the effect of habitat availability on disease mortality at such a large scale is probably outside the ability of our data, and is probably not appropriate.

There are large variations between regions in mortality distribution as shown in figures 3 and 4. For this reason we compared disease mortality and acreage between regions. At this scale both diseases show slight negative correlations with habitat availability as would be

expected if assumptions were correct expected (fig. 5 and 6). However, looking at the graphs, it becomes evident that each region occupies a different area on the acreage scale. This gives each regions unequal weight across the acreage scale, and could skew our results. For this reason we analyzed each region individually that had large mortalities. We thought this analysis would be more appropriate for attempting to infer management implications for habitat effects on disease from our results. At this scale only botulism in the Sacramento Valley shows a negative trend. Botulism mortality in Tulare Basin shows a strong positive relationship with wetland acreage. This is a factor of the dynamics of the wetlands in the region. Being a drained lakebed, the basin is extremely prone to flooding. These floodwaters are included in the wetland acreages. This creates some confounding problems with acreage and water supply because they are so closely linked. If possible, an study should be done to access the effect of increased managed wetlands on botulism mortality in Tulare to remove this problem. The Tulare Basin is a very interesting place as far as avian botulism is concerned, and should be a place of future research to test possible botulism management strategies. Cholera mortality in both the Sacramento Valley and Grasslands also shows a slight positive relationship with acreage.

Except for botulism in the Tulare Basin, all analyses of wetland acreage and disease at all scales show weak trends in both directions. The fact that neither disease has the same trend between the different analytical scales suggests that there is not a strong relationship between habitat availability and disease mortality in either direction.

Other Observation

The similarities between botulism and cholera outbreaks across the Central Valley suggests that there are some common variables that promote or allow disease to effect waterfowl at the valley-wide scale. This is reflected by the fact that the water variable chosen for each disease at that scale has the largest single effect on mortality. This indicates that within the Central Valley, years with higher water supply should have greater disease mortality. Water was also found to have a strong effect in all analyses except within each region for cholera. This indicates that water supply is an important component of botulism outbreaks at all levels, and for cholera on a large-scale view.

Botulism and cholera are both generally associated with extreme temperatures; botulism usually occurs during hot weather, while cholera often occurs during times of cold temperatures. We found it interesting that except for botulism at the valley-wide scale, the best predicting

temperature variable was the opposite of that which the disease is generally associated. This suggests that these diseases may be more controlled by inhibition rather than promotion.

When population estimates were included in our predictor-models, it consistently had a very small and positive coefficient. This suggests that increased bird numbers does increase mortality, as expected, but by a very small amount.

SUGGESTIONS:

To further our understanding avian botulism and avian cholera outbreaks, several areas of future research would be profitable. Conversations with refuge biologists yielded valuable insights into the factors that might be operating on their areas. For example: during a cholera outbreak in the grasslands, one biologist plotted the rate of deaths against weather conditions (fog, rain, overcast, sunny) and found that during fog, and overcast rainy days mortality was high, and the day the sun came out, the outbreak stopped. He hypothesized that the birds were under thermal stress and were succumbing to the toxin at greater rates. Further research on this possibility would be revealing.

A second suggestion to facilitate future studies would be to establish a standardized method of record-keeping for disease mortality in all regions of the valley (and perhaps elsewhere). It proved difficult to obtain disease records on a consistent basis. There is no central database or location where reports are archived and disease labs for both federal and state refuges often had limited data. Collection of information for this study required sorting through volumes of hand written notes and standardized forms that changed frequently. Development and implementation of a standardized method of recording disease loss would facilitate future analysis of trends in disease outbreaks. Such analyses, conducted with an expanded standardized data set, could help considerably in understanding the factors that influence these diseases, and undertaking management actions minimize their impact on waterfowl populations.

Another method that may improve the predictor-models for disease outbreaks would be to analyze disease mortalities on an outbreak/non-outbreak year categorical basis. This way we would instead be attempting to predict years that would have large outbreaks, rather than the actual number of mortalities, which has little significance.

ACKNOWLEDGMENTS

Thanks to all of the following for their help in data collection and assistance during this survey: Chris Courtright, Richard Moss, Greg Gerstenburg, Dean Kwasny, Jay Dee Gar, Cathy Osugi, John Beam, Shawn Milar, Mark Petrie, Timothy Keldson, Kevin Foerster, Dennis Becker, Bill Cook, Steve Cordes, Earl Cummings, Dave Feliz, Les Howard, Robert Huddleston, Jamie Jackson, Bruce Wicklund, Mike Womack, and Michael Samuel. And special thanks to Ruth Ostroff, Mike Wolder, David Hardt and Greg Mensik and Central Valley Habitat Joint Venture.

Literature Cited

- Anderson, David R., K. P. Burnham, and W. L. Thompson. 2000. Null Hypothesis Testing: Problems, Prevalence, and an Alternative. *Journal of Wildlife Management* 64(4):2000
- Anderson, David R., W. A. Link, D. H. Johnson, and K. P. Burnham. 2001. Suggestions for Presenting the Results of Data Analyses. *Journal of Wildlife Management* 65(3):2001
- Backstrand, J. M. and R. G. Botzler. 1986. Survival of *Pasteurella multocida* in Soil and Water in an Area Where Avian Cholera is Endemic. *Journal of Wildlife Diseases* 22(2):257-259
- Barras, Scott C. and J. A. Kadlec. 2000. Abiotic Predictors of Avian Botulism Outbreaks in Utah. *Wildlife Society Bulletin* 28(3):724-729
- Beam, John. 1978. Avian Botulism in the Southern San Joaquin Valley, 1978.
- Bell, J. F., G. W. Sciple, and A. A. Hubert. 1955. A Microenvironment Concept of the Epizootology of Avian Botulism. *Journal of Wildlife Management* 19:352-357
- Bredy, James P. and R. G. Botzler. 1989. The Effects of Six Environmental Variables on *Pasteurella multocida* Populations in Water. *Journal of Wildlife Diseases* 25(2):232-239
- Duncan, Ruth and W. I. Jensen. 1976. A Relationship Between Avian Carcasses and Living Invertebrates in the Epizootology of Avian Botulism. *Journal of Wildlife Diseases* 12(1):116-126
- Friend, Milton. 1981. Waterfowl Management and Waterfowl Disease: Independent or Cause and Effect Relationship?. *Transactions of the North American Wildlife and Natural Resource Conference* 46: 94-103
- Friend, Milton. 1992. Environmental Influences on Major Waterfowl Diseases. *Transactions of the North American Wildlife and Natural Resource Conference* 57:517-525
- Gordus, Andrew G.. 1993. Lead Concentrations in Liver and Kidneys of Snow Geese During an Avian Cholera Epizootic in California. *Journal of Wildlife Diseases* 29(4):582-586
- Hines, Roy A.. 1973. Avian Botulism in the Southern San Joaquin Valley, 1973. _____
- Hunter, Brian. 1978. Interactions Between Lead, Lead-iron and Iron Shot and Avian Cholera in Waterfowl. Prepared for: Wildlife Disease Association Meeting Fort Collins, Colorado
- Marion, Wayne R., T. E. O'Meara, G. D. Riddle, and H. A. Berkhoff. 1983. Prevalence of *Clostridium botulinum* Type C in Substrates of Phosphate-mine Settling Ponds and Implications for Epizootics of Avian Botulism. *Journal of Wildlife Diseases* 19(4):302-307
- Mays, Alfred S.. 1940. Observations on Duck Disease at Tulare Lake Basin, 1940. *California Fish and Game* 27(3):153-164
- McLean, Donald D.. 1945. Duck Disease at Tulare Lake. *California Fish and Game* 32(2):71-80
- Parrish, John M. and B. F. Hunter. 1969. Waterfowl Botulism in the Southern San Joaquin Valley, 1967-68. *California Fish and Game*
- Price, Jessie I. and C. J. Brand. 1984. Persistence of *Pasteurella multocida* in Nebraska Wetlands Under Epizootic Conditions. *Journal of Wildlife Diseases* 20(2):90-94

- Reed, Thomas M. and T. E. Roche. 1992. The Role of Avian Carcasses in Botulism Epizootics. *The Wildlife Society Bulletin* 20(2):175-182
- Samuel, Michael D., W. P. Johnson, D. J. Shaddock, D. R. Goldberg, and C. C. Terry. 2002. The Role of Lesser Snow Geese as Carriers of Avian Cholera in the Playa Lakes Region. _____
- Sandler, Renee J., T. E. Rock, M. D. Samuel, and T. M. Yuill. 1993. Seasonal Prevalence of *Clostridium botulinum* Type C in Sediments of a Northern California Wetland. *Journal of Wildlife Diseases* 29(4):533-539
- Smith, Brian J., K. F. Higgins, and W. Lee Tucker. 1990. Precipitation, Waterfowl Densities and Mycotoxins: Their Potential Effect on Avian Cholera Epizootics in the Nebraska Rainwater Basin Area. *Transactions of the North American Wildlife and Natural Resource Conference* 55: 269-282
- Smith, Brian J. and K. F. Higgins. 1990. Avian Cholera and Temporal Changes in Wetland Numbers and Densities in Nebraska's Rainwater Basin Area. *Wetlands* 10(1):1-5
- Smith, L. P.. 1979. The Effect of Weather on the Incidence of Botulism in Waterfowl. *Agricultural Meteorology* 20:483-488
- Stribling, Charles R. and T. J. Harper. 1969. Waterfowl Botulism in the Tulare Lake Basin. _____
- Wobeser, G. 1992. Avian Cholera and Waterfowl Biology. *Journal of Wildlife Diseases* 28(4):674-682
- Wobeser, G., D. B. Hunter, B. Wright, and R. Isbister. 1979. Avian Cholera in Waterfowl in Saskatchewan, Spring 1977. *Journal of Wildlife Diseases* 15(1):19-24
- Wobeser, G., S. Marsden, and R. J. MacFarlane. 1987. Occurrence of Toxigenic *Clostridium botulinum* Type C in the Soil of Wetlands in Saskatchewan. *Journal of Wildlife Diseases* 23(1):67-76

TABLES AND FIGURES

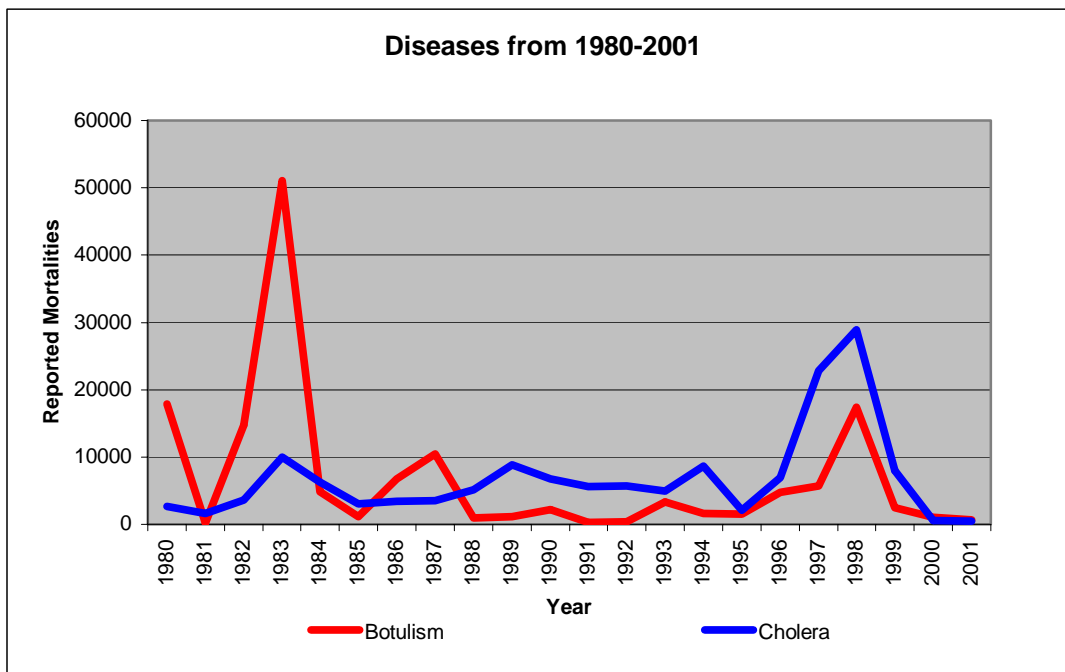


Fig 1. Total annual botulism and cholera mortalities within the Central Valley from 1980-2001.

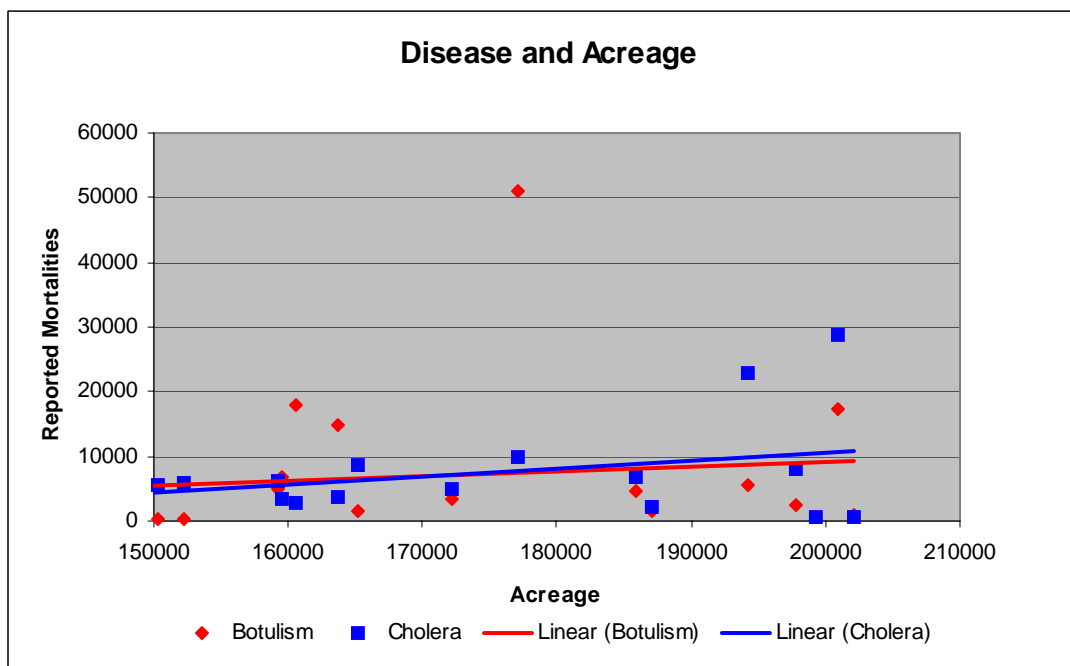


Fig. 2 Total annual botulism and cholera mortalities within the Central Valley plotted against total wetland acreage in the Central Valley.

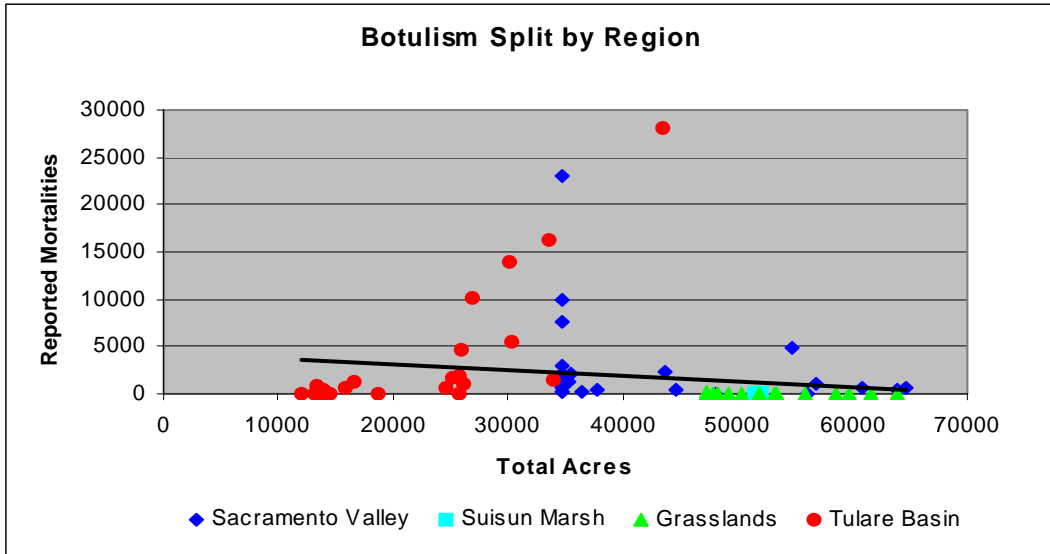


Fig. 5 Annual botulism mortality in the Central Valley separated into four regions with linear trend line for whole valley.

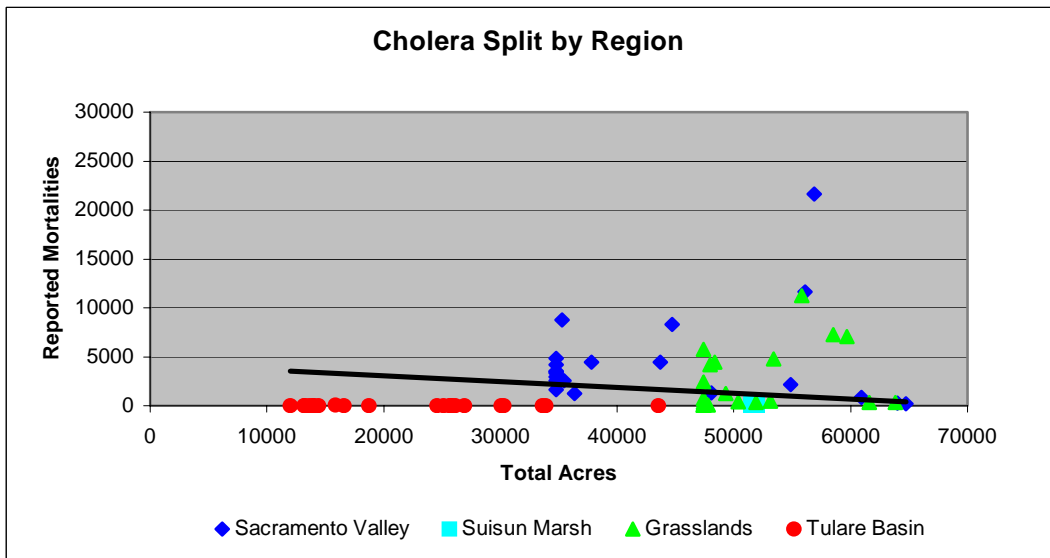


Fig. 6 Annual cholera mortality in the Central Valley separated into four regions with linear trend line for whole valley.

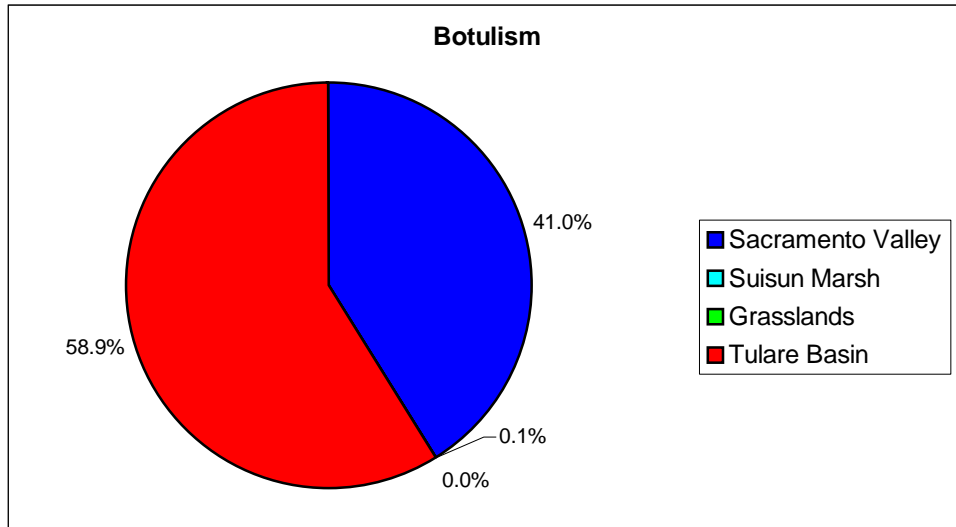


Fig 3. Total botulism mortalities within the Central Valley from 1980-2001 separated by region

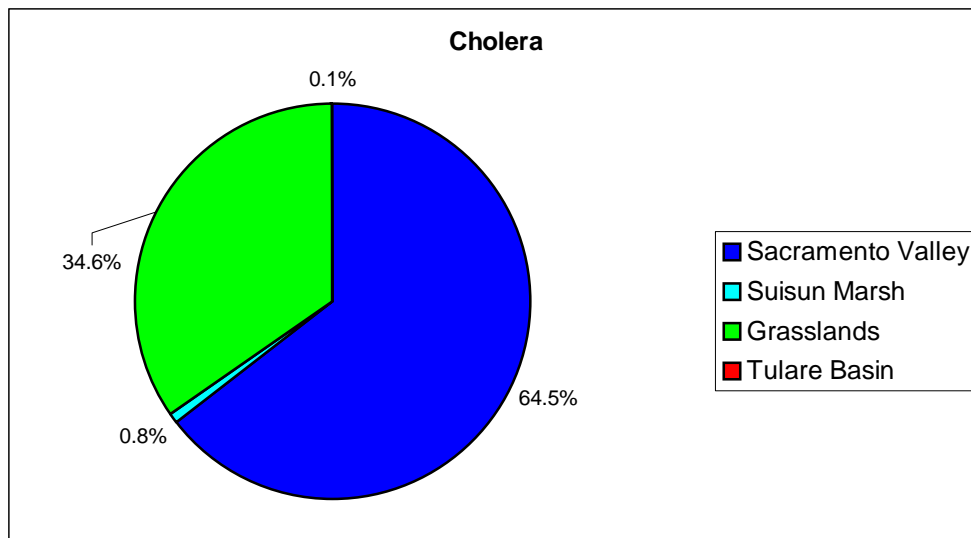


Fig 4. Total cholera mortalities within the Central Valley from 1980-2001 separated by region

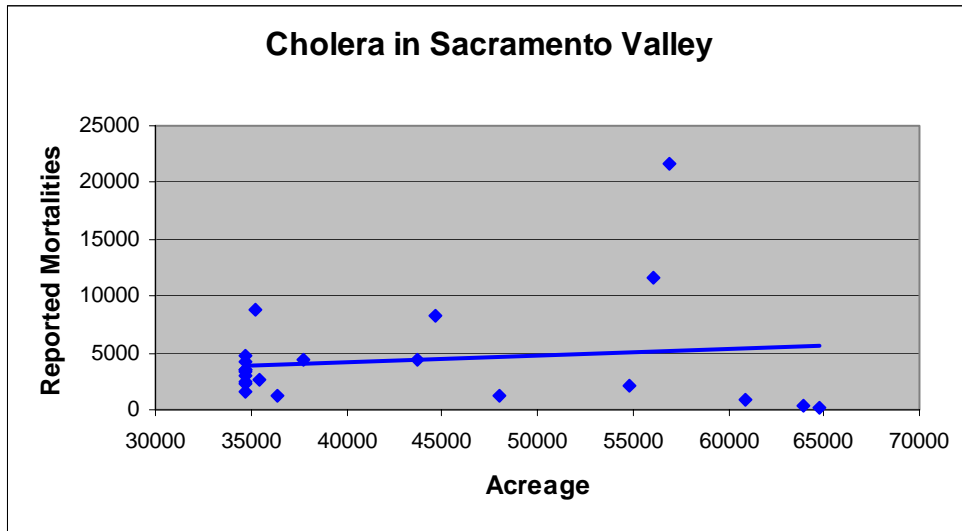


Fig. 9 Annual cholera mortality in the Sacramento Valley plotted against Total Acreage with linear trend line.

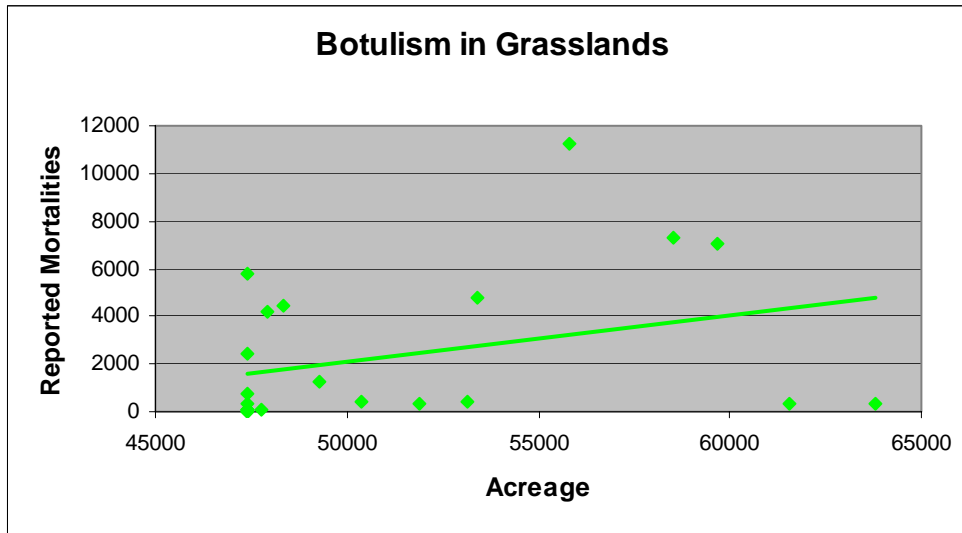


Fig. 10 Annual cholera mortality in the Grasslands plotted against Total Acreage with linear trend line.

Table 1. AIC results for all models for botulism across the Central Valley with (n) number of observations, (k) total number of estimatable parameters, (logL) log-linear likelihood, (AICc) AIC value for small samples, (DAIC) difference in AICc from top model, (wi) Akaike weights and R-squared for the regression.

Botulism: Valley-wide							
MODEL	n	K	logL	AICc	DAICc	wi	R-sq
water	22	3	-198.932	405.197	0.000	0.423	0.413
water +temp	22	4	-198.916	408.185	2.990	0.095	0.414
acreage + water	22	4	-198.098	406.549	1.350	0.215	0.456
water + birds	22	4	-198.527	407.408	2.210	0.140	0.434
water + birds + temp	22	5	-198.476	410.702	5.510	0.027	0.437
acreage + water +temp	22	5	-197.970	409.689	4.490	0.045	0.462
acreage + water + birds	22	5	-198.002	409.754	4.560	0.043	0.461
acreage	22	3	-204.575	416.482	11.290	0.001	0.019
temp	22	3	-204.634	416.601	11.400	0.001	0.014
birds	22	3	-204.785	416.904	11.710	0.001	0.001
acreage + temp	22	4	-204.381	419.114	13.920	0.000	0.037
acreage + birds + temp + water	22	6	-197.858	413.315	8.120	0.007	0.468
acreage + birds	22	4	-204.478	419.309	14.110	0.000	0.028
temp + birds	22	4	-204.633	419.618	14.420	0.000	0.014
acreage + temp + birds	22	5	-204.301	422.351	17.150	0.000	0.044

Table 2. Cumulative Akaike weights for each variable.

Cumulative Weights of Evidence Support	
Total Acreage	0.313
Run-off	0.995
Summer Max	0.176
Midwinter Pop	0.219

Table 3. AIC results for all models for cholera across the Central Valley with (n) number of observations, (k) total number of estimatable parameters, (logL) log-linear likelihood, (AICc) AIC value for small samples, (DAIC) difference in AICc from top model, (wi) Akaike weights and R-squared for the regression.

<i>Cholera: Valley-wide</i>							
MODEL	n	K	logL	AICc	DAICc	wi	R-sq
water	22	3	-191.645	390.623	0.000	0.271	0.160
acreage	22	3	-191.988	391.310	0.690	0.192	0.133
water + acreage	22	4	-191.162	392.676	2.050	0.097	0.196
temp	22	3	-192.834	393.001	2.380	0.082	0.064
water + temp	22	4	-191.552	393.457	2.830	0.066	0.167
birds + acreage	22	4	-191.608	393.569	2.950	0.062	0.163
water + birds	22	4	-191.640	393.633	3.010	0.060	0.160
temp + acreage	22	4	-191.906	394.165	3.540	0.046	0.140
birds	22	3	-193.561	394.456	3.830	0.040	0.000
water + birds + acreage	22	5	-191.038	395.827	5.200	0.020	0.205
birds + temp	22	4	-192.777	395.908	5.290	0.019	0.069
water + temp + acreage	22	5	-191.158	396.066	5.440	0.018	0.196
birds + temp + acreage	22	5	-191.493	396.736	6.110	0.013	0.171
water + birds + temp	22	5	-191.551	396.852	6.230	0.012	0.167
birds + temp + water + acreage	22	6	-191.023	399.646	9.020	0.003	0.206

Table 4. Cumulative Akaike weights for each variable.

Cumulative Weights of Evidence Support	
Total Acreage	0.450
Fall/Winter Precip	0.546
Winter Max	0.259
Midwinter Pop	0.229

Table 5. AIC results for all models for botulism across the Central Valley separated by region with (n) number of observations, (k) total number of estimatable parameters, (logL) log-linear likelihood, (AICc) AIC value for small samples, (DAIC) difference in AICc from top model, (wi) Akaike weights and R-squared for the regression.

Botulism: All Regions							
MODEL	n	K	logL	AICc	DAICc	wi	R-sq
acreage + water	88	4	-737.0939	1482.670	0.000	0.332	0.351
acreage + water + birds	88	5	-736.2838	1483.299	0.630	0.242	0.139
acreage + water + temp	88	5	-737.012	1484.756	2.090	0.117	0.125
acreage + water + temp + birds	88	6	-736.1741	1485.385	2.720	0.085	0.142
water	88	3	-739.9504	1486.186	3.520	0.057	0.065
water + birds	88	4	-739.1691	1486.820	4.150	0.042	0.081
temp + birds	88	4	-739.1691	1486.820	4.150	0.042	0.024
temp + water	88	4	-739.7638	1488.009	5.340	0.023	0.069
water + birds + temp	88	5	-738.9417	1488.615	5.950	0.017	0.086
acreage	88	3	-741.4481	1489.182	6.510	0.013	0.032
acreage + birds	88	4	-740.5955	1489.673	7.000	0.010	0.225
birds	88	3	-742.0645	1490.415	7.740	0.007	0.019
acreage + temp	88	4	-741.3199	1491.122	8.450	0.005	0.035
acreage + temp + birds	88	5	-740.4324	1491.597	8.930	0.004	0.054
temp	88	3	-742.6838	1491.653	8.980	0.004	0.005

Table 6. Cumulative Akaike weights for each variable.

Cumulative Weights of Evidence Support	
Total Acreage	0.809
Spring/Summer Precip	0.916
Summer Min	0.297
Midwinter Pop	0.449

Table 7. AIC results for all models for cholera across the Central Valley separated by region with(n) number of observations, (k) total number of estimatable parameters, (logL) log-linear likelihood, (AICc) AIC value for small samples, (DAIC) difference in AICc from top model, (wi) Akaike weights and R-squared for the regression.

Cholera: All Regions							
MODEL	n	K	logL	AICc	DAICc	wi	R-sq
acreage + birds	88	4	-703.903	1416.288	0.000	0.463	0.205
acreage + temp + birds	88	5	-703.691	1418.113	1.825	0.186	0.209
birds	88	3	-706.178	1418.641	2.353	0.143	0.163
temp + birds	88	4	-705.662	1419.807	3.519	0.080	0.173
acreage + water + birds	88	6	-703.699	1420.435	4.148	0.058	0.209
water + birds	88	5	-705.466	1421.664	5.376	0.032	0.176
acreage + birds + temp + water	88	7	-703.577	1422.555	6.267	0.020	0.211
water + birds + temp	88	6	-705.283	1423.604	7.316	0.012	0.180
acreage + water	88	5	-708.135	1427.001	10.713	0.002	0.125
water	88	4	-709.747	1427.977	11.689	0.001	0.092
acreage + water +temp	88	6	-707.872	1428.780	12.492	0.001	0.130
water +temp	88	5	-709.405	1429.541	13.253	0.001	0.099
acreage + temp	88	4	-711.011	1430.505	14.217	0.000	0.066
acreage	88	3	-712.114	1430.513	14.225	0.000	0.042
temp	88	3	-712.418	1431.122	14.834	0.000	0.035

Table 8. Cumulative Akaike weights for each variable.

Cumulative Weights of Evidence Support	
Total Acreage	0.732
Run-off + Fall/Winter Precip	0.859
Winter Min	0.300
Midwinter Pop.	0.994

Table 9. AIC results for all models for botulism in the Sacramento Valley with (n) number of observations, (k) total number of estimatable parameters, (logL) log-linear likelihood, (AICc) AIC value for small samples, (DAIC) difference in AICc from top model, (wi) Akaike weights and R-squared for the regression.

Botulism: Sacramento Valley							
MODEL	n	K	logL	AICc	DAICc	wi	R-sq
temp	22	3	-186.225	379.784	0.000	0.203	0.119
water +temp	22	4	-184.952	380.256	0.470	0.160	0.215
acreage	22	3	-186.779	380.892	1.110	0.117	0.074
water	22	3	-187.021	381.374	1.590	0.092	0.053
acreage + water	22	4	-185.536	381.426	1.640	0.089	0.173
birds	22	3	-187.376	382.085	2.300	0.064	0.022
temp + birds	22	4	-185.906	382.166	2.380	0.062	0.144
acreage + temp	22	4	-186.128	382.610	2.830	0.049	0.127
acreage + water +temp	22	5	-184.644	383.038	3.250	0.040	0.237
water + birds + temp	22	5	-184.729	383.207	3.420	0.037	0.231
acreage + birds	22	4	-186.692	383.737	3.950	0.028	0.081
water + birds	22	4	-186.844	384.040	4.260	0.024	0.068
acreage + water + birds	22	5	-185.527	384.804	5.020	0.016	0.173
acreage + temp + birds	22	5	-185.886	385.522	5.740	0.012	0.146
acreage + birds + temp + water	22	6	-184.552	386.704	6.920	0.006	0.243

Table 10. Cumulative Akaike weights for each variable.

Cumulative Weights of Evidence Support	
Total Acreage	0.358
Spring/Summer Precip	0.823
Summer Min	0.569
Midwinter	0.249

Table 11. AIC results for all models for botulism in the Tulare Basin with (n) number of observations, (k) total number of estimatable parameters, (logL) log-linear likelihood, (AICc) AIC value for small samples, (DAIC) difference in AICc from top model, (wi) Akaike weights and R-squared for the regression.

Botulism: Tulare Basin							
MODEL	n	K	logL	AICc	DAICc	wi	R-sq
Acreage + water	22	4	-180.648	371.650	0.000	0.620	0.718
acreage + water + birds	22	5	-180.375	374.500	2.851	0.150	0.725
acreage + water +temp	22	5	-180.566	374.880	3.233	0.120	0.720
acreage	22	3	-184.854	377.040	5.393	0.040	0.587
acreage + birds + temp + water	22	6	-180.249	378.100	6.449	0.020	0.728
acreage + birds	22	4	-184.207	378.770	7.118	0.020	0.610
acreage + temp	22	4	-184.607	379.570	7.919	0.010	0.596
acreage + temp + birds	22	5	-183.861	381.470	9.823	0.000	0.622
water	22	3	-188.539	384.410	12.762	0.000	0.422
water + birds	22	4	-188.330	387.010	15.365	0.000	0.433
water +temp	22	4	-188.460	387.270	15.625	0.000	0.426
water + birds + temp	22	5	-188.239	390.230	18.579	0.000	0.438
birds	22	3	-194.522	396.380	24.730	0.000	0.005
temp	22	3	-194.566	396.460	24.817	0.000	0.001
temp + birds	22	4	-194.514	399.380	27.733	0.000	0.005

Table 12. Cumulative Akaike weights for each variable.

Cumulative Weights of Evidence Support	
Total Acreage	0.998
Spring/Summer Precip	0.959
Summer Min Temp	0.165
Midwinter Pop	0.197

Table 13. AIC results for all models for cholera in the Sacramento Valley with (n) number of observations, (k) total number of estimatable parameters, (logL) log-linear likelihood, (AICc) AIC value for small samples, (DAIC) difference in AICc from top model, (wi) Akaike weights and R-squared for the regression.

<i>Cholera: Sacramento Valley</i>							
MODEL	n	K	logL	AICc	DAICc	wi	R-sq
temp	22	3	-185.347	378.027	0.000	0.232	0.048
birds	22	3	-185.501	378.335	0.310	0.199	0.034
acreage	22	3	-185.686	378.706	0.680	0.165	0.018
birds + temp	22	4	-184.682	379.716	1.690	0.100	0.103
water	22	4	-184.888	380.129	2.100	0.081	0.086
birds + acreage	22	4	-185.114	380.580	2.550	0.065	0.068
temp + acreage	22	4	-185.162	380.677	2.650	0.062	0.063
birds + temp + acreage	22	5	-184.240	382.229	4.200	0.028	0.139
water + birds	22	5	-184.577	382.903	4.880	0.020	0.112
water + temp	22	5	-184.631	383.012	4.980	0.019	0.108
water + acreage	22	5	-184.794	383.338	5.310	0.016	0.094
water + birds + temp	22	6	-184.214	386.028	8.000	0.004	0.141
water + birds + acreage	22	6	-184.340	386.280	8.250	0.004	0.131
water + temp + acreage	22	6	-184.523	386.647	8.620	0.003	0.116
birds + temp + water + acreage	22	7	-183.920	389.839	11.810	0.001	0.163

Table 14. Cumulative Akaike weights for each variable.

Cumulative Weights of Evidence Support	
Total Acreage	0.344
Run-off + Fall/Winter Precip	0.149
Winter Max	0.449
Midwinter Pop	0.421

Table 15. AIC results for all models for cholera in the Grasslands with (n) number of observations, (k) total number of estimatable parameters, (logL) log-linear likelihood, (AICc) AIC value for small samples, (DAIC) difference in AICc from top model, (wi) Akaike weights and R-squared for the regression.

Cholera: Grasslands							
MODEL	n	K	logL	AICc	DAICc	wi	R-sq
acreage	22	3	-175.554	358.442	0.000	0.317	0.106
birds	22	3	-176.212	359.758	1.316	0.164	0.051
temp	22	3	-176.491	360.316	1.874	0.124	0.027
water	22	4	-175.334	361.020	2.578	0.087	0.124
birds + acreage	22	4	-175.489	361.330	2.888	0.075	0.112
temp + acreage	22	4	-175.554	361.461	3.019	0.070	0.106
water + acreage	22	5	-174.182	362.114	3.672	0.051	0.211
birds + temp	22	4	-176.135	362.624	4.182	0.039	0.058
water + birds	22	5	-175.103	363.957	5.515	0.020	0.142
water + temp	22	5	-175.333	364.415	5.974	0.016	0.124
birds + temp + acreage	22	5	-175.487	364.723	6.282	0.014	0.112
water + temp + acreage	22	6	-173.806	365.211	6.770	0.011	0.238
water + birds + acreage	22	6	-174.182	365.963	7.521	0.007	0.211
water + birds + temp	22	6	-175.087	367.774	9.332	0.003	0.144
birds + temp + water + acreage	22	7	-173.804	369.609	11.167	0.001	0.238

Table 16. . Cumulative Akaike weights for each variable.

Cumulative Weights of Evidence Support	
Total Acreage	0.546
Run-off + Fall/Winter Precip	0.196
Winter Max	0.208
Midwinter Pop	0.324

Appendix A:

Year	Total Deaths	DISEASE			TOTAL NUMBER OF DEATHS PER YEAR FOUND IN EACH HABITAT TYPE			
		Cholera	Botulism	Other	Seasonal		Permanent	
					<i>Botulism</i>	<i>Cholera</i>	<i>Botulism</i>	<i>Cholera</i>
1980								
1981								
1982								
1983								
1984								
1985								
1986								
1987								
1988								
1989								
1990								
1991								
1992								
1993								
1994								
1995								
1996								
1997								
1998								
1999								
2000								
2001								
2002								

Appendix A:

Habitat Questionnaire

What was the total wetland habitat acreage on your area in 1980?

Please fill in as much of the lower table as you have the data or the time to look up. The most important is the additional acreage added each year, the breakdown of wetland type is secondary.

Year	Acreage Additions	TOTAL ACREAGE BY YEAR	
		Seasonal	Permanent
1980			
1981			
1982			
1983			
1984			
1985			
1986			
1987			
1988			
1989			
1990			
1991			
1992			
1993			
1994			
1995			
1996			
1997			
1998			
1999			
2000			
2001			
2002			

Appendix B:

<i>Refuge</i>	<i>Region</i>	<i>Responded (Y/N)</i>	<i>Data Collected</i>
Kern NWR	Tulare Basin	Y	Yes
Mendota WA	Tulare Basin	Y	Yes
Los Banos WA	Grasslands	Y	Yes
Sacramento NWR	Sacramento Valley	Y	Yes
Delevan NWR	Sacramento Valley	Y	Yes
Upper Butte Basin WA	Sacramento Valley	Y	Yes
Gray Lodge WA	Sacramento Valley	Y	incomplete
Oroville WA	Sacramento Valley	No	No
North Grasslands WA	Grasslands	Y	Yes
Yolo Bypass WA	Sacramento Valley	Y	Yes
Stone Lakes NWR	Sacramento Valley	Y	Yes
Grizzly Island WA	Suisun Marsh	Y	Yes
San Luis NWR	Grasslands	Y	Yes

Appendix C:

Variable	Description	Disease
Run-off	Total run-off reported by the California Department of Water Resources for the Sacramento and San Joaquin Valleys in million acre-feet.	Botulism and Cholera
Precipitation	Total annual precipitation on biological year. Collected at each weather station	Botulism and Cholera
Spring/Summer Precipitation	Total annual spring and summer precipitation. Collected at each weather station.	Botulism
Fall/Winter Precipitation	Total annual fall and winter precipitation. Collected at each weather station	Cholera
Summer Max	The single greatest observed temperature during summer each year. Collected at each weather station.	Botulism
Summer Min	The single least observed temperature during summer each year. Collected at each weather station.	Botulism
Winter Max	The single greatest observed temperature during winter each year. Collected at each weather station.	Cholera
Winter Min	The single least observed temperature during winter each year. Collected at each weather station.	Cholera
Days Below 5	Total number of days where minimum observed temperature was below 5 degrees centigrade. Collected at each weather station.	Cholera
Days Below 0	Total number of days where minimum observed temperature was below 0 degrees centigrade. Collected at each weather station.	Cholera