Temperature and precipitation levels as predictors of epidemics of western equine encephalitis in horses in Iowa

by

Leo Lawrence Konermann, Jr., DVM

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TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

INTRODUCTION

The primary role of medicine must be to prevent disease. To place primary emphasis on the treatment of disease processes, in human or animal hosts, important as this is, is to be always on the defensive. The real advances come when diseases can be prevented. This most often implies ability to predict the potential presence of the disease processes. Prediction must be done far enough in advance to allow prophylactic measures to be applied before disease processes materially affect the target population.

Given the complex interaction of environment, agent and host, prediction of disease is seldom simple or foolproof. The arboviral infections, with the additional complicating factor of vector hosts, are even more complex. Reeves (1967) lists some fifty-two significant factors or subfactors required for an arbovirus epidemic to occur, many of which are not measurable. The only hope is that certain of these factors can be proven to be significant and consistent indicators of a disease. The subject of this work is an investigation of a combination of two such potential indicators.

It is proposed that epidemic levels of western equine encephalitis (WEE) in horse populations can be predicted in early June by comparing threshold values with a combination of total monthly precipitation and average monthly temperature for certain spring months preceding the active mosquito season.

STATEMENT OF THE PROBLEM

Economic factors have discouraged yearly mosquito abatement programs and even widespread vaccination of horses because of a lack of obvious benefit. Both vaccination, which leads to protective levels of antibodies, and organization and implementation of adequate abatement programs require lead time prior to the expected emergence of epidemics. The primary difficulty is that there are no known parameters to inexpensively determine early enough in the season whether or not a full scale abatement or vaccination program would be needed to abort a potential epidemic of WEE.

In California, Reeves has assessed one such parameter based on the numbers of mosquitoes caught per trap night to predict virus transmission (Reeves 1971). However, attempts to assess the validity of his findings in Iowa retrospectively based on mosquito collection efforts and records of cases of WEE have not been successful. Assuming that with additional research Reeves' theory would apply in Iowa, there still remain problems of extent, time, and cost. Mosquito samplings would have to be quite extensive because there seem to be great local variations in populations. Lead time also becomes a problem in basing vaccination or mosquito abatement on current populations of mosquitoes. By the time that mosquitoes are collected, sorted, totaled, and data compiled, the lead time to equine or human infections will be very brief. Finally, there is considerable expense involved in this process.

What is desirable is one or a combination of inexpensive, timely, and accurate indicators of a potential WEE epidemic which will allow preventive measures to be applied. To determine such indicators a consideration of the time elements and significant quantifiable factors is requisite. Data presented later in this paper will indicate that July is the first month for significant levels of WEE in epidemic years. June incidence rates are not discernibly different for epidemic and non-epidemic years. Given the two to three week horse incubation period for the disease (Thomas 1963), places the time of the virus spillover period in mid-June. This is when insufficient avian hosts are present and the vector, Culex tarsalis, begins to feed in significant numbers on mammalian hosts (Reeves 1971). Indicators at this point are high mosquito populations or sentinel chicken seroconversions in large numbers (Reeves 1971). Both are indicators with good validity but tend to be expensive and give little lead time to prevent equine (or human) infection as noted above.

Prior to this mid-June time frame, virus amplification has had to occur. Using susceptible birds (fledglings), the vectors have produced a large virus pool of infected birds and mosquitoes (Reeves 1971). Because vector competence has increased and the extrinsic incubation period decreased with increasing average temperatures (Hardy 1973), a warmer period in May and early June allows viral amplification to progress to a point where spillover is sufficient to produce epidemic levels. An indicator here would be a threshold temperature level for May and June. Considering that June would

generally be sufficiently warm, an average monthly temperature for May may be the key.

There are, however, other factors that will be significant in a virus amplification cycle. Major measurable factors are vectors and susceptible avian hosts. Nestling population numbers are hard to quantify accurately, but the vector host, C. tarsalis, is quantifiable and has been used as an indicator (Reeves 1971) with the drawbacks noted above. The vector in large numbers is very important to epidemic levels of WEE at all states of the amplification cycle and spillover. Both temperature (Moon 1976) (Chapman 1969) and precipitation (Graham and Collett 1969) have great influence on population levels of C. tarsalis. A time analysis of significant population increases of C. tarsalis in Iowa showed a requirement for a temperature above an undesignated threshold level for two weeks, preceded by high rainfall levels at eighteen and/or thirty four days prior to temperature elevations (Hacker 1973). Given the temperature exceeding some threshold in early May for vector production would mean high rainfall in late March and mid-April as precursors and indicators of a sufficient mosquito population to trigger an equine epidemic of WEE.

Threshold levels for temperature and precipitation in a particular geographic area must be found empirically. However, certain factors from the bionomics of the C. tarsalis can give indications of where such threshold values will be. Water is required for larval/pupal development, and C. tarsalis is capable of using a wide

range of water containments from hoofprints to lakes as breeding sites (Carpenter and LaCasse 1955). Water quantities must be sufficient to keep such temporary catchment breeding sites filled. Although no approximate value can be projected, some threshold value should exist. Temperature values have been better researched. With adequate water available, C. tarsalis reproduced under field conditions within the range 7°C (47°F) to 32°C (90°F) with a maximal reproduction at an average daily temperature of 18°C (65°F) (Fanara and Mulla 1974). The threshold value for temperature can be expected to be in that vicinity. Separate research has determined that short periods of cooler weather 6°C (45°F) will not significantly affect reproduction success. In nature this fits cool nights with warm days.

From the foregoing reasoning, the thrust of this work is evident. Total monthly precipitation for March and April, plus May and June average temperatures will be compared with epidemic levels of WEE in horses in the nine regions of Iowa in an attempt to find reliable predictors of epidemic levels of WEE in horses.

REVIEW OF THE LITERATURE

The Virus

The causative agent of WEE together with eastern equine encephamolyelitis (EEE) and Venezuelan equine encephamolyelitis (VEE) is classified as an alphavirus. It is a member of the family Togaviridae, which is characterized by single-stranded ribonucleic acid (RNA) genetic material, cubic capsid symmetry, and ether sensitive envelopes. The alphaviruses differ from all other members of the Togaviridae, except the rubiviruses, by the use of surface membrane sites for nucleocapsid envelopment and by the size of their virion (Melnick 1978). Distinct constituent antigenic properties also differentiate the alphaviruses from other groups (Henderson et al. 1967).

Although the first recognized epidemic in the United States of what was probably WEE occurred in Kansas and Nebraska in 1912 (Udall 1913), virus isolation was not accomplished until Meyer isolated the virus from a sample from the San Joaquin Valley of California in 1930 (Meyer et al. 1931). Iowa experienced the large epidemic of the 1930s and the virus was isolated by Biester and Schwarte (1940) in 1939. Since that time both virus isolations and clinical signs of equine WEE have occurred regularly in the State of Iowa (Zymet et al. 1966), as in most other areas west of the Mississippi River and on the Canadian plains (Burton et al. 1966).

The normal bird-mosquito-bird transmission cycle for the maintenance and amplification of the WEE virus has been investigated

(Hess and Hayes 1967), and the spillover into mammalian hosts well confirmed (Reeves 1971). However, no overwintering mechanism for the WEE virus in Iowa and other areas inhospitable to viral replication or transmission in the winter has been confirmed (Bellamy et al. 1967). Various overwintering mechanism theories have been proposed using mosquitoes (Blackmore and Winn 1956), snakes and lizards (Prior 1971) and turtles (Hess and Hayes 1967), the cliff swallow bug (O. vicarius) (Hayes et al. 1977), jackrabbits (Lepus californicus), or other small mammals (Reeves 1974) among others. None have garnered sufficient scientific evidence to gain strong proponents, and all have serious drawbacks (Blackmore and Winn 1956) (Rush et al. 1963) (Reeves 1974). The most likely method of maintaining endemicity of the WEE virus in non-tropical areas seems to be a yearly reintroduction through infected migratory birds (Reeves 1974). This mechanism itself is not proven and the absence of WEE viral antibodies in livestock in Hawaii, among other factors, casts some doubt on this method (Scherer et al. 1972).

The Vector

Although WEE virus has been isolated from many invertebrates (Prior 1971) (Hayes et al. 1977), the transmission vectors which have been shown to be capable of transmitting disease in mammals have been limited to the culicine mosquitoes (McLean 1975). Specifically, C. tarsalis must be considered the primary transmission vector in Iowa and the rest of the western United States (Chamberlain 1958)

(Hess and Holden 1958) (Reeves and Hammon 1962) (Hayes et al. 1967). However, the virus has also been isolated from a wide range of mosquitoes including Aedes trivatattus, Anopheles punctipennis, and C. pipiens (complex) in Iowa (Rowley et al. 1973). In Texas, Culiseta melanura and Cu. inornata have been implicated in the transmission of the WEE virus in an epidemic and during cold springs respectively (Hess et al. 1963).

C. tarsalis with a flight range of five miles (Reeves 1976) is an excellent vector for the WEE virus. The viral infection does not seem to affect the longevity of the species, but egg laying has been shown to be somewhat reduced in infective mosquitoes (Thomas 1963). It normally is an avian feeder. If avian populations are inadequate or the birds take action to prevent blood feeding such as wing flapping or feather ruffling, C. tarsalis will feed on other vertebrates including horses and people (Reeves 1976) (Nelson et al. 1976). Reeves et al. (1961) proved the employment and effectiveness of these defensive measures by restraining chickens with nylon stockings and observing a higher proportion of feedings on them. They also noted a higher infection rate in single birds and small flocks than in larger aggregations.

Natural transmission of WEE virus occurs 4-14 days after the infected blood meal (Hayles et al. 1972). An infective dose of at least $10^{2.5}$ chick embryo LD_{50} of the WEE virus is required to infect C. tarsalis. The viral titer in mosquitoes usually stabilizes at about $10^{4.5}$ - $10^{6.5}$ chick embryo LD_{50}, no matter what the initial dose

(Thomas 1963). This matches well with $10^{4.5}$ chick embryo LD_{50} ($10^{4.3}$ PFU) of WEE virus required to infect a horse (Sponsteller 1966). Reeves found the infection rates C. tarsalis in California to be four times the transmission rates because mosquitoes often fed again before the end of the extrinsic incubation period of the virus (Reeves et al. 1961), more fully explaining his previous findings that large vector populations and high vector infection rates did not necessarily equate to high transmission rates (Reeves et al. 1958).

Much evidence exists correlating the incidence of WEE and C. tarsalis populations. Reeves and Hammon (1962) found strong correlations between the incidence of WEE in horses and people with C. tarsalis infection rates. Several large disease outbreaks of WEE in horses and people have been associated with large populations of C. tarsalis (Chamberlain 1958) (Reeves and Hammon 1962) (Graham and Collett 1964). In Iowa, Zymet et al. (1966) found June C. tarsalis populations proportional to disease levels of WEE in Iowa; and although somewhat decreased from 1940 to 1970, they were still an abundant specie statewide. Rowley et al. (1973) found more mosquitoes, and 40% of the statewide C. tarsalis population, in the same western areas that Zymet et al. (1966) identified as containing a major portion of the state totals of WEE in horses and people. Wong et al. (1971) found that even by July and August when their population should have waned greatly, C. tarsalis compromised 2.9% of the mosquito population in 1966, a non-epidemic year. Reeves developed data

on the number of gravid female C. tarsalis caught per night by his New Jersey night traps in a survey area in Kern County, California. He found a threshold value of 10 gravid females per light trap night as a predictor of epidemic levels of WEE in the human population, and that less than one C. tarsalis per light trap night resulted in the virus effectively disappearing from the area (Reeves 1971). The risk of disease was found to be proportional to the C. tarsalis population (Reeves 1976). Graham and Collett (1969) used this concept and the developed values to successfully predict the absence of epidemic levels of the WEE in Utah through a ten-year period.

Vertebrate Hosts

As noted above, C. tarsalis primarily feeds on birds, and avian hosts are the primary vertebrate hosts of the WEE virus. A wide range of avian species have yielded antibody titers and isolations of WEE virus. One implicated in many studies is the house sparrow (Passer domesticus) (Hayes et al. 1967) (Holden et al. 1973) (Reeves 1974). A great asset to the continuance of the cycle, in this species at least, is the fact that parental immunity to WEE virus did not alter the susceptibility of the offspring to infection (Holden et al. 1973).

The chicken has been used as a sentinel animal (Reeves 1971), but it does not seem to be as sensitive a host as some wild birds. It may be that high viremic levels lasting up to ten days in non-domestic birds create an advantage to the virus (Thomas and Eklund 1968). The defense measures against excessive blood feeding have

been mentioned previously (Reeves 1971).

A great number of mammalian hosts exist. Many have been found to have WEE virus or antibodies to it in the search for an over-wintering mechanism. These have included jack rabbits, prairie dogs (Cynomys ludovicianus), thirteen striped ground squirrels (Spermophilus tridecemlineatus) (Hayes et al. 1967), snowshoe hares (Lepus americanus) (Yuill and Hanson 1964) (Kiorpes and Yuill 1975), Richardson's ground squirrels (Spermophilus richardsonii) (Leung et al. 1975), California ground squirrels (Spermophilus beecheii) and gray squirrels (Sciurus griseus) (Hardy et al. 1974). Most of these have been considered short term hosts (Hardy 1973), but relatively high winter viremic levels in snowshoe hares may indicate that they are possible overwintering hosts (Yuill and Hanson 1964).

The two susceptible groups of particular concern are the equine and human populations. The former have been considered more at risk to the vectors, and equine cases of WEE have preceded human cases by two to four weeks (Reeves et al. 1964). Horses contracting this disease have had a mortality rate of 50% (Byrne 1972) and many of those surviving have had permanent sequellae. However, there have been instances of complete recovery (Devine and Byrne 1960).

Susceptible horses receiving an infective dose of at least $10^{4.5}$ chick embryo LD_{50} of WEE virus have shown clinical signs after an incubation period of one to three weeks (Thomas and Eklund 1968). Clinical disease has been initiated by the rapid onset of high fever up to a maximum of $40.7^{\circ}C$ (Sponsteller 1966). Fatal cases have

been evidenced by a progressive paralysis leading to death. Non-fatal cases have been evidenced by less extensive paralysis, and the main signs have been lack of awareness of surroundings, aimless wandering, circling and crashing into objects (Smith and Jones 1966). Such animals have typically shown hypersensitivity and teeth grinding decreasing within four days and a normal gait returning by the end of about seven days after onset (Sponsteller 1966). There have been no significant gross lesions described. Due to the neurogenic nature of the disease, microscopic lesions have been restricted to neuronal necrosis but have not been pathognomonic. Confirmation of the disease in non-fatal cases has been based on a rise in serum neutralizing or complement fixing antibodies (Smith and Jones 1966), but the clinical manifestations have been sufficiently unique in Iowa that presumptive diagnoses have been reported on the basis of clinical signs (Hendricks personal communication 1978)[1]. In an analysis of 156,992 cases reported on clinical bases, paired serum samples were available on only 19,467 (12.4%), 17,247 (88.6%) of these were serologically confirmed as WEE and none were confirmed as another disease (Pinger unpublished 1974)[2]. This has tended to support the accuracy of the reported cases that were not serologically assessed.

The human case fatality rate from WEE has averaged 15% (McLean 1975). In a California study, 52% of the WEE cases occurred in children under one year of age (Longshore et al. 1959). This group

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[1] Hendricks, S. L., Iowa State Department of Health, 1978.

[2] Pinger, R. R., Dept. of Vet. Microbiol. and Prevent. Med., Iowa State University, 1974.

has been particularly sensitive to central nervous system (CNS) damage, with 44% of those contracting the disease at less than three months of age experiencing CNS sequellae. This percentage decreases somewhat in the three months to one year group, but a large drop in CNS sequellae has not occurred until the five year old group (Palmer and Finley 1956). Fortunately, some children in endemic areas have been born with maternal antibodies, which 85% have lost by seven months of age (Longshore et al. 1959). Convalescent patients in all age groups have shown more residual changes, especially learning disabilities, than those in similar age groups which have contracted St. Louis encephalitis (SLE) (Palmer and Finley 1956).

Environmental Factors

The interrelationships between WEE disease incidence, C. tarsalis populations, and temperature and precipitation data have been explored by several investigators. Reeves (1976) found that prolonged spring-time flooding in the Central Valley of California, the only reliable source of water for that area except winter rains and irrigation, has lead to epidemics of WEE in that area. Hess et al. (1963) found a positive correlation between increased incidence of WEE in both horses and people with increased precipitation during the months of April to June. He also noted that cool dry springs led to decreased mosquito populations in summer (Hess et al. 1963). Graham and Collett (1964) correlated increased May or June precipitation with increased mosquito populations and disease incidence in Utah.

The environmental effects of abnormally high spring temperatures have been more complex, being linked to increases in vector longevity and breeding populations (Reeves and Hammon 1962), breeding capacity (Reeves 1970), viral replication, susceptible nestling bird populations (Reeves and Hammon 1962), prevalence of antibodies to the WEE virus in both birds and mammals (Reeves et al. 1964), and extrinsic virus incubation (Thomas 1963). This last observation has been elaborated in several experiments. Both Thomas (1963) and Reeves et al. (1964) found the extrinsic incubation periods for WEE virus to have decreased with increasing temperatures. Rush et al. (1963) found a reduction in the extrinsic incubation of the WEE virus from 21 days in the spring to 5 days in the summer. More efficient natural WEE virus transmission has also been noted, progressing to a maximum at an average daily temperature of 26.7°C (80°F) (Thomas 1963). More efficient and uniform transmission of the WEE virus was found at 23.9°C (75°F) than 20.6°C (69°F) (Hayles et al. 1972), and at 23.9°C (75°F) transmission was frequently accomplished after an extrinsic incubation period as short as four days (Thomas 1963). Laboratory experiments have indicated that C. tarsalis eggs would have at least a prolonged embryonation during colder temperatures 7.2°C (45°F), and many would not hatch or have poor evolution rates to adults (Asman 1975). A linear relationship between temperature and egg development was found between 20°C (68°F) and 30°C (86°F) (Hagstrum and Workman 1971).

The cited data and analyses of weather data and conditions have led to several theories of climatic conditions required for WEE

epidemics. Empirical observations were used to advance a theory of WEE "outbreaks" occurring only north of the 21.1°C (70°F) June isotherm (Hess et al. 1963). Another suggested that cool wet springs followed by hot dry summers were important to the causation of epidemic years for WEE in horses and people (Hess et al. 1963). However, Graham and Collett (1964) observed that in Utah warm wet springs followed by hot dry summers were associated with epidemic levels of WEE in horses and people.

Ever since Ross (1910) utilized the concept of vector critical levels in his studies on malaria, quantification of the precursors of unusual disease incidence has been recognized as a valid source of predictors for those diseases. The relationship of temperature and precipitation to vector population size (Bailey and Gieke 1968), (Fanara and Mulla 1974) and (Moon 1976), of vector population size to disease incidence (Rees et al. 1959), (Reeves 1971), and (Olsen et al. 1979), and of quantifiable temperature levels to incidence of WEE (Hess et al. 1963) have been investigated. The concept is based on assessing meteorobiological conditions that enhance vector populations and in turn initiate transmission of the etiological agent to animal hosts. This paper attempts to combine these procedures and to predict epidemic levels of WEE from temperature and precipitation data.

Such a procedure is not new. Although certainly not utilizing the same epidemiological processes, fascioliasis levels in Anglesy have been predicted by a formula involving minimum temperatures,

amounts of rain and numbers of rainy days in the month (Ollenshaw and Rowlands 1959). Reeves (1967) suggested the possibility of threshold levels of temperature and precipitation for epidemic levels of arboviral disease, and his student Olson (1978) attempted to correlate each temperature and precipitation values with WEE incidence, but not a combination of the two. Olson's data were from Kern County, California, where available water levels are influenced by the spring runoff from the Sierra snowpack and the ever expanding irrigation systems as well as the usually heavy winter and scant spring precipitation. Because of this complex available water situation, he was unable to focus rainfall data sufficiently to quantify values. He was also unable to explain why equine WEE incidence decreased and human WEE incidence seemingly increased with the height of average temperatures in the April to June period. He did not, however, break down the meteorological data to smaller (monthly) units of temperature for correlation with epidemic levels of WEE incidence.

DATA ACQUISITION

The geographical base of this study has been the State of Iowa. The state was divided along county lines into nine roughly equal areas using the northwest through southeast regional designations of the Environmental Data Service (EDS) of the National Oceanographic and Atmospheric Administration (NOAA). (See Figure 1.) The nine areas were chosen as sufficiently large to minimize local influences on WEE case numbers while not being so large that climatological information, especially significant local differences in precipitation levels, would lose its meaning by being averaged over too great an area.

Horse populations were obtained from the 1959, 1969 and 1974 Agricultural Censuses of Iowa (U. S. Bureau of the Census, 1961, 1972, 1977). In the 1964 census, the horse category was inexplicably omitted. The population figures were linearly computed between census years and projected beyond the last census to 1976 and are shown in Table 1. These figures show a decided decline in horse populations over the study period, although a survey in Iowa in 1975 showed an increase in that year (Hendricks unpublished 1976)[1]. It is probable that the actual horse population is somewhat higher than official figures, but in the absence of better population data it must be presumed that the error is proportionally constant for all the census years on which this study is based.

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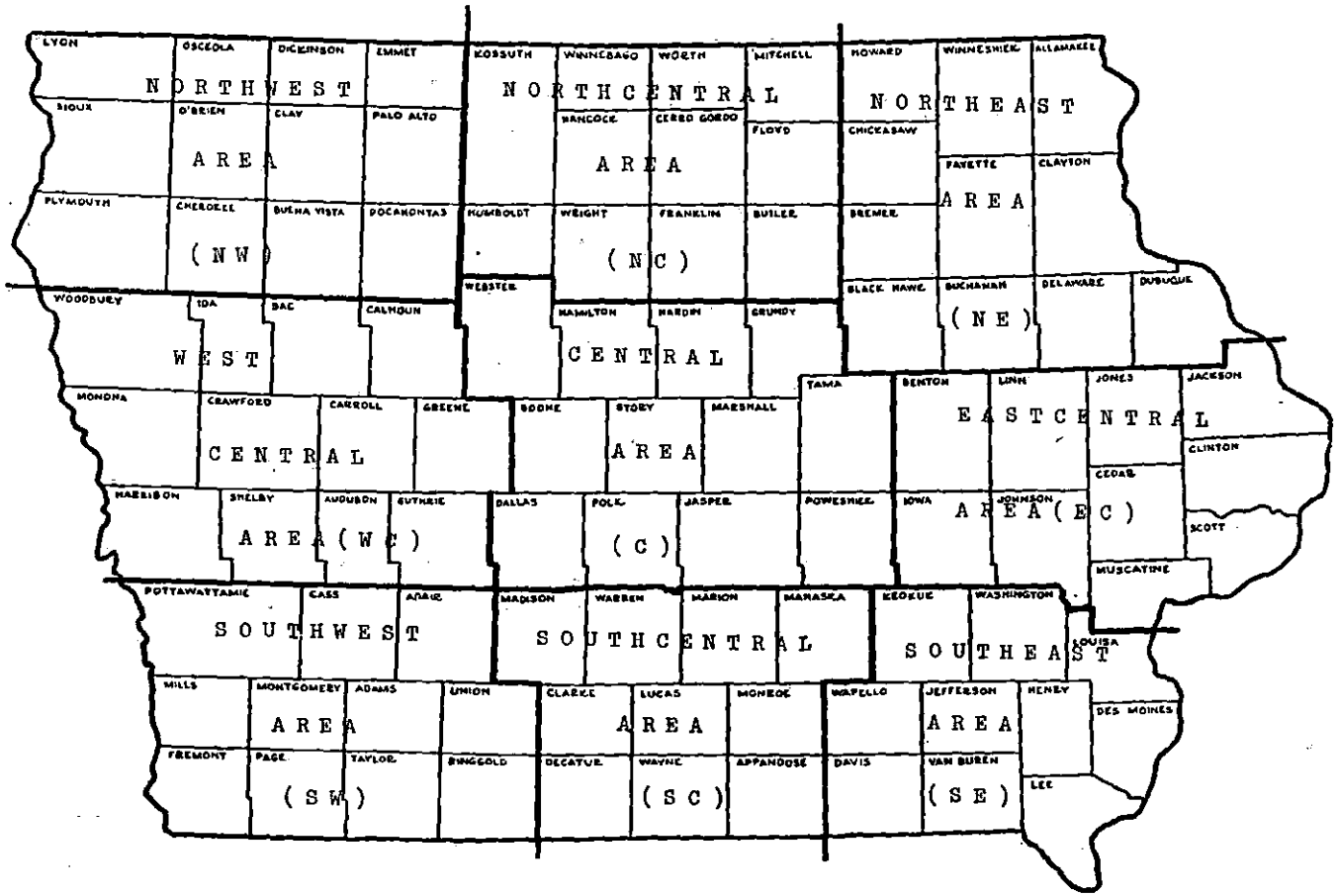
[1] Hendricks, S. L., Iowa State Department of Health, 1976.

Figure 1. Division of Iowa into geographical areas


LYON
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KOSSUTH
WINNEBAGO
WORTH
MITCHELL
HOWARD
WINNESHIEK
ALLAMAKEE
NORTHWEST
NORTHCENTRAL
NORTHEAST
AREA
AREA
AREA
SIOUX
O'BRIEN
CLAY
PALO ALTO
HANCOCK
CERRO GORDO
FLOYD
CHICKASAW
FAYETTE
CLAYTON
PLYMOUTH
CHEROKEE
BUENA VISTA
POCAHONTAS
HUMBOLDT
WRIGHT
FRANKLIN
BUTLER
BREMER
(NW)
(NC)
WOODBURY
IDA
SAC
CALHOUN
WEBSTER
HAMILTON
HARDIN
GRUNDY
BLACK HAWK
BUCHANAN
DELAWARE
DUBUQUE
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WEST
CENTRAL
MONONA
CRAWFORD
CARROLL
GREENE
BOONE
STORY
MARSHALL
TAMA
BENTON
LINN
JONES
JACKSON
EASTCENTRAL
CENTRAL
AREA
CLINTON
HARRISON
SHELBY
AUDUBON
GUTHRIE
DALLAS
POLK
JASPER
POWESHIEK
IOWA
JOHNSON
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MARION
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KEOKUK
WASHINGTON
LOUISA
SOUTHWEST
SOUTHCENTRAL
SOUTHEAST
MILLS
MONTGOMERY
ADAMS
UNION
CLARKE
LUCAS
MONROE
WAPELLO
JEFFERSON
HENRY
DES MOINES
AREA
AREA
AREA
FREMONT
PAGE
TAYLOR
RINGGOLD
DECATUR
WAYNE
APPANOOSE
DAVIS
VAN BUREN
LEE
(SW)
(SC)
(SE)

Table 1. Estimated equine population for Iowa by geographic areas 1962-1976

YEAR	NW	NC	NE	WC	C	EC	SW	SC	SE
1962	7018	7510	9482	8849	10884	10036	7158	9905	9160
1963	7128	7478	9454	8840	10902	10101	7029	9773	9039
1964	7038	7446	9426	8830	10920	10166	6899	9642	8917
1965	7047	7415	9399	8820	10939	10231	6770	9511	8796
1966	7057	7383	9371	8811	10957	10296	6641	9379	8675
1967	7067	7352	9343	8801	10975	10361	6512	9248	8554
1968	7077	7320	9316	8792	10994	10426	6383	9116	8432
1969	7087	7288	9288	8782	11012	10491	6254	8985	8311
1970	6573	6804	8931	8120	10256	9993	5758	8392	7779
1971	6060	6320	8575	7458	9500	9495	5263	7799	7247
1972	5546	5835	8218	6795	8744	8997	4767	7205	6715
1973	5033	5351	7862	6133	7988	8499	4272	6612	6183
1974	4519	4867	7505	5471	7232	8001	3776	6019	5651
1975	4005	4383	7148	4809	6476	7503	3280	5426	5119
1976	3492	3899	6792	4147	5720	7005	2785	4833	4587

The number of cases of equine WEE and their locations were obtained from the Iowa State Department of Health (ISDH 1963-1977), and is summarized in Table 2. These yearly summaries were based on reports of veterinarians and a small percentage of reported cases

Table 2. Reported equine cases of WEE by areas for Iowa, 1962-1976

YEAR	NW	NC	NE	WC	C	EC	SW	SC	SE
1962	33	20	15	23	49	53	47	13	15
1963	59	48	6	83	100	39	120	119	43
1964	145	219	65	168	178	131	99	110	172
1965	122	92	34	106	89	90	121	72	86
1966	23	24	9	35	48	16	35	49	23
1967	42	30	20	38	41	23	46	33	16
1968	43	47	14	42	106	50	32	33	27
1969	38	40	10	22	48	28	39	22	8
1970	50	34	28	51	55	39	50	89	53
1971	15	18	6	16	23	14	20	25	10
1972	14	18	5	13	22	37	15	16	15
1973	21	11	3	8	17	7	11	9	8
1974	33	27	5	19	18	11	15	13	12
1975	77	51	52	79	50	34	34	33	24
1976	4	2	2	6	6	9	10	2	0

were serologically tested and seroconfirmed (Pinger unpublished 1974)[1]. However, these reported clinically diagnosed cases were considered to be sufficiently accurate for use in this study as in a previous one

[1]Pinger, R. R., Dept. of Vet. Microbiol. and Prevent. Med., Iowa State University, 1974.

(Zymet et al. 1966). Attack rates were computed for all years using reported case numbers and the projected yearly population as shown in Table 3. Epidemic levels considered to be periods of disease incidence in excess of expected frequency (Schwabe et al. 1977) were identified for the purpose of this study on the basis of frequency polygon charts for each area of the state with peak attack rates clearly above the usual level for the area being considered epidemic years.

Precipitation and temperature data were obtained from EDS summaries (EDS 1961-1976). The total monthly precipitation for each area was obtained for March and April of each year separately and combined (Tables A-1 thru A-3). Average monthly temperatures for each area were compiled for May each year (Table A-4).

The Iowa State Department of Health also had available for the years 1969-1975 a compilation of reported cases of WEE in the State of Iowa by months (ISDH 1969-1975) though these were not broken down by county or area.

Table 3. Attack rate of WEE in horses in Iowa, 1962-1976

YEAR	NW	NC	NE	WC	C	EC	SW	SC	SE
	Reported cases per 1000 horses by area								
1962	4.70	2.66	1.58	2.60	4.50	5.28	6.57	1.31	1.64
1963	8.39	6.42	0.63	9.37	9.17	3.86	17.07	12.18	4.75
1964	20.60	29.41	6.90	19.03	16.30	12.89	14.35	11.41	19.29
1965	17.31	12.41	3.62	12.02	8.14	8.80	17.87	7.57	9.78
1966	3.26	3.25	0.96	3.97	4.32	1.55	5.27	5.22	2.65
1967	5.94	4.08	2.14	4.32	3.74	2.22	7.06	3.57	1.87
1968	6.08	6.42	1.50	4.78	9.64	4.80	5.01	3.62	3.20
1969	5.36	5.49	1.08	2.51	4.36	2.67	6.24	2.45	0.96
1970	7.61	5.00	3.14	6.28	5.36	3.90	8.68	10.63	6.81
1971	2.48	2.85	0.70	2.15	2.42	1.47	3.80	3.21	1.38
1972	2.52	3.08	0.61	1.91	2.52	4.11	3.15	2.22	2.23
1973	4.17	2.06	0.38	1.30	2.13	0.82	2.57	1.36	1.29
1974	7.30	5.55	0.67	3.47	2.49	1.37	3.97	1.96	2.12
1975	19.23	11.64	7.27	16.43	7.72	4.53	10.37	6.08	4.69
1976	1.15	0.51	0.29	1.45	1.05	1.28	3.95	0.41	0
$\bar{x}$	7.74	6.72	2.10	6.11	5.59	3.97	7.73	4.88	4.18
s	6.01	6.84	2.25	5.41	3.87	3.12	4.84	3.75	4.72

RESULTS

In this study, equine WEE attack rates, total monthly precipitation for March and April, and average monthly temperatures for May and June were compared by areas of the State of Iowa for the period 1962-1976 to assess threshold levels for precipitation and temperature, and epidemic attack rates. These data are shown in Figures 2-10. Precipitation and temperature data for other months in the first half of the year were also screened with no correlations evident.

The graphs of attack rates in all areas by years indicated the epidemic threshold rates shown in Figure 11. Epidemic years were statewide in 1964, also in 1965 with the exception of the northeast and south central sections. The southwest and south central sections experienced epidemic levels in 1963, as did the southeast and south central in 1970, and the west central plus northern tier in 1975. Noteable also was the increase in attack rates in endemic years from east to west, with the unglaciated northeast the lowest.

The graphed temperatures showed a clear gradation from north to south. A threshold value of 16.7°C (62°F) existed in the northern tier of areas with 16.9°C (64°F) the threshold in the central areas and 17.1°C (66°F) in the southern tier.

Graphed total monthly precipitation for March and April combined for each area by year showed generally similar precipitation conditions throughout the state and projected a threshold of 5.8 inches.

The necessary conditions for high vector mosquito populations were a wet spring (March and April) followed by a hot May. A

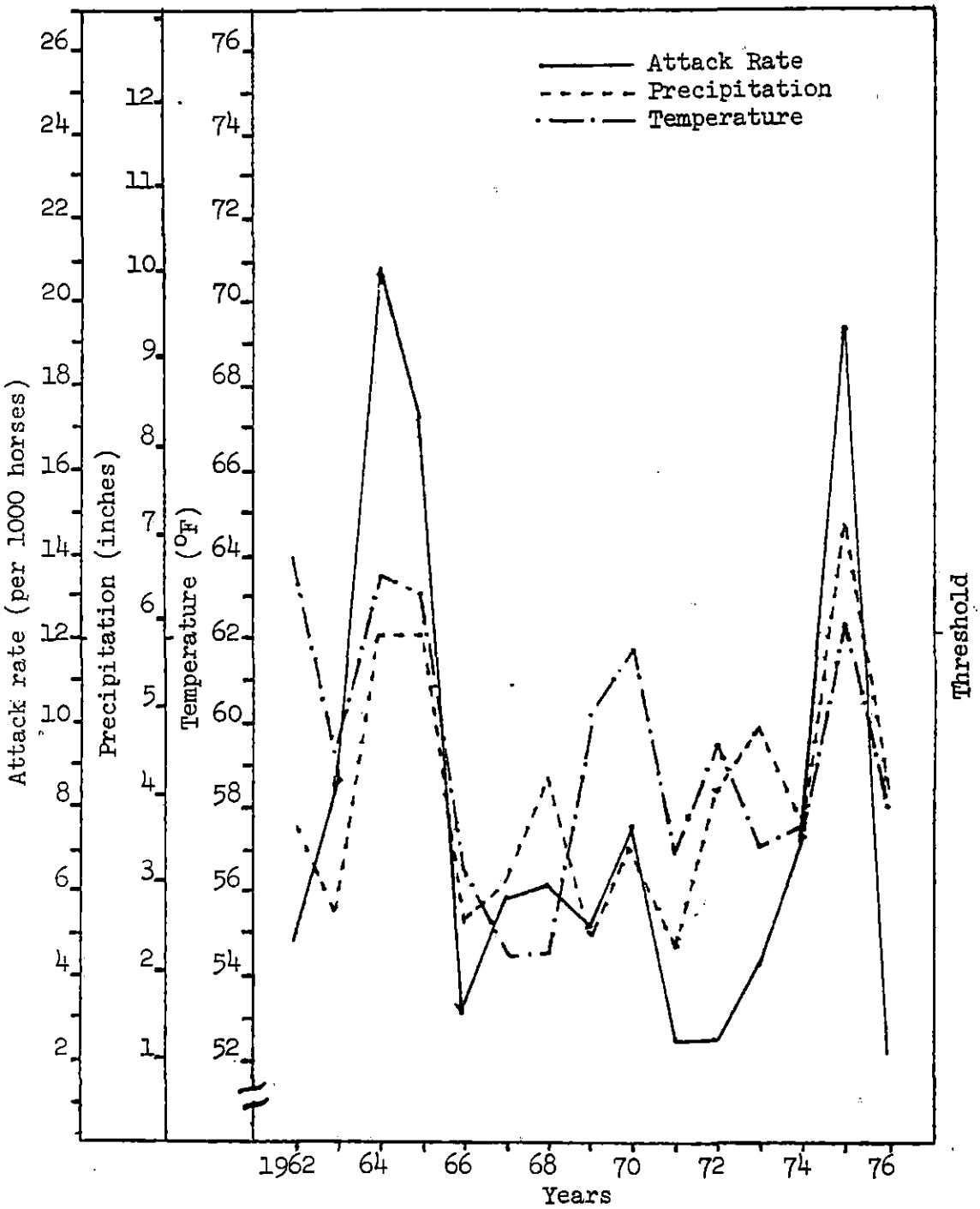


Figure 2. WEE attack rate, March and April total precipitation, and average May temperature, 1962-1976, Northwest Iowa

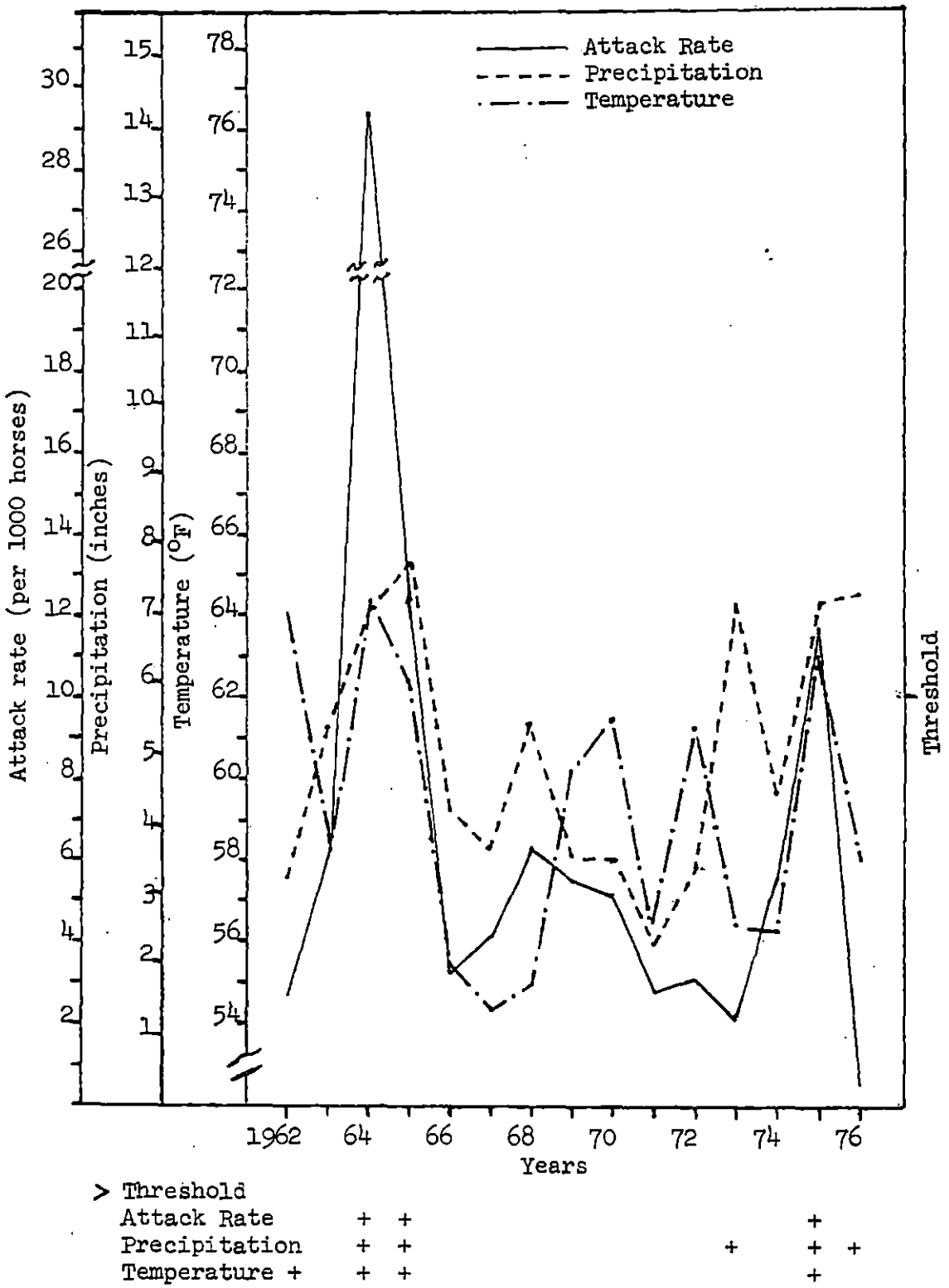


Figure 3. WEE attack rate, March and April total precipitation, and average May temperature, 1962-1976, North Central Iowa

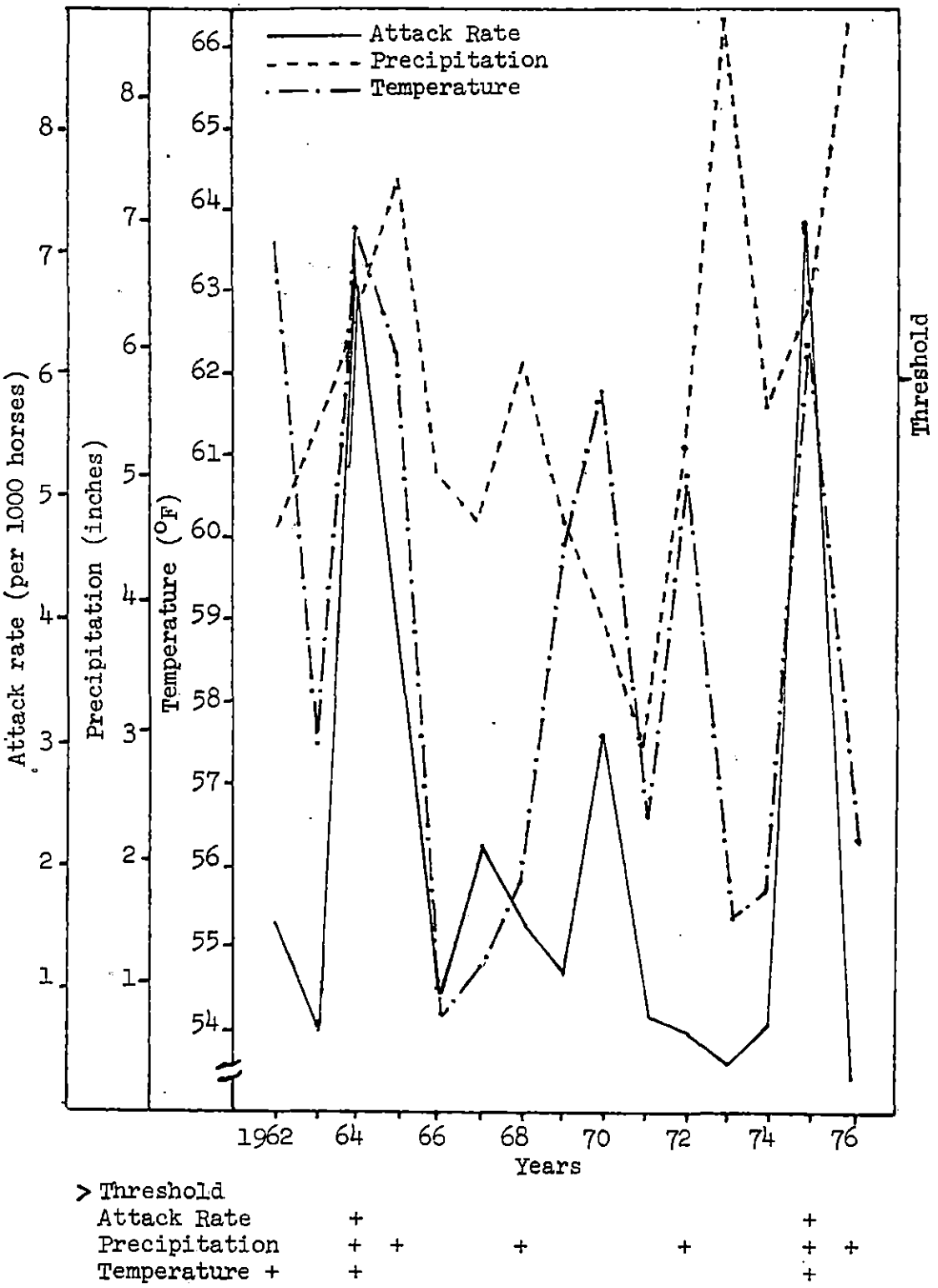


Figure 4. WEE attack rate, March and April total precipitation, and average May temperature, 1962-1976, Northeast Iowa

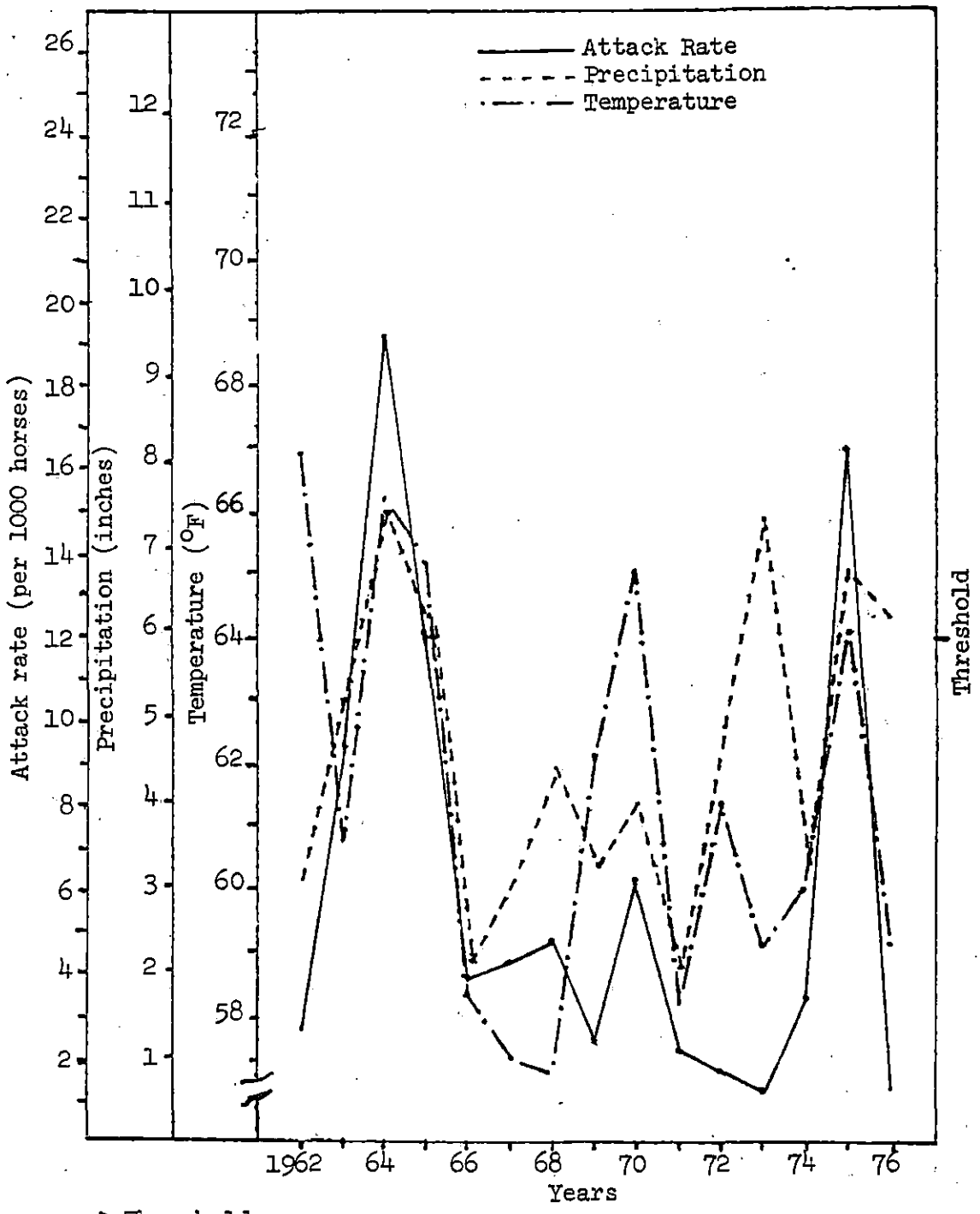


Figure 5. WEE attack rate, March and April total precipitation, and average May temperature, 1962-1976, West Central Iowa

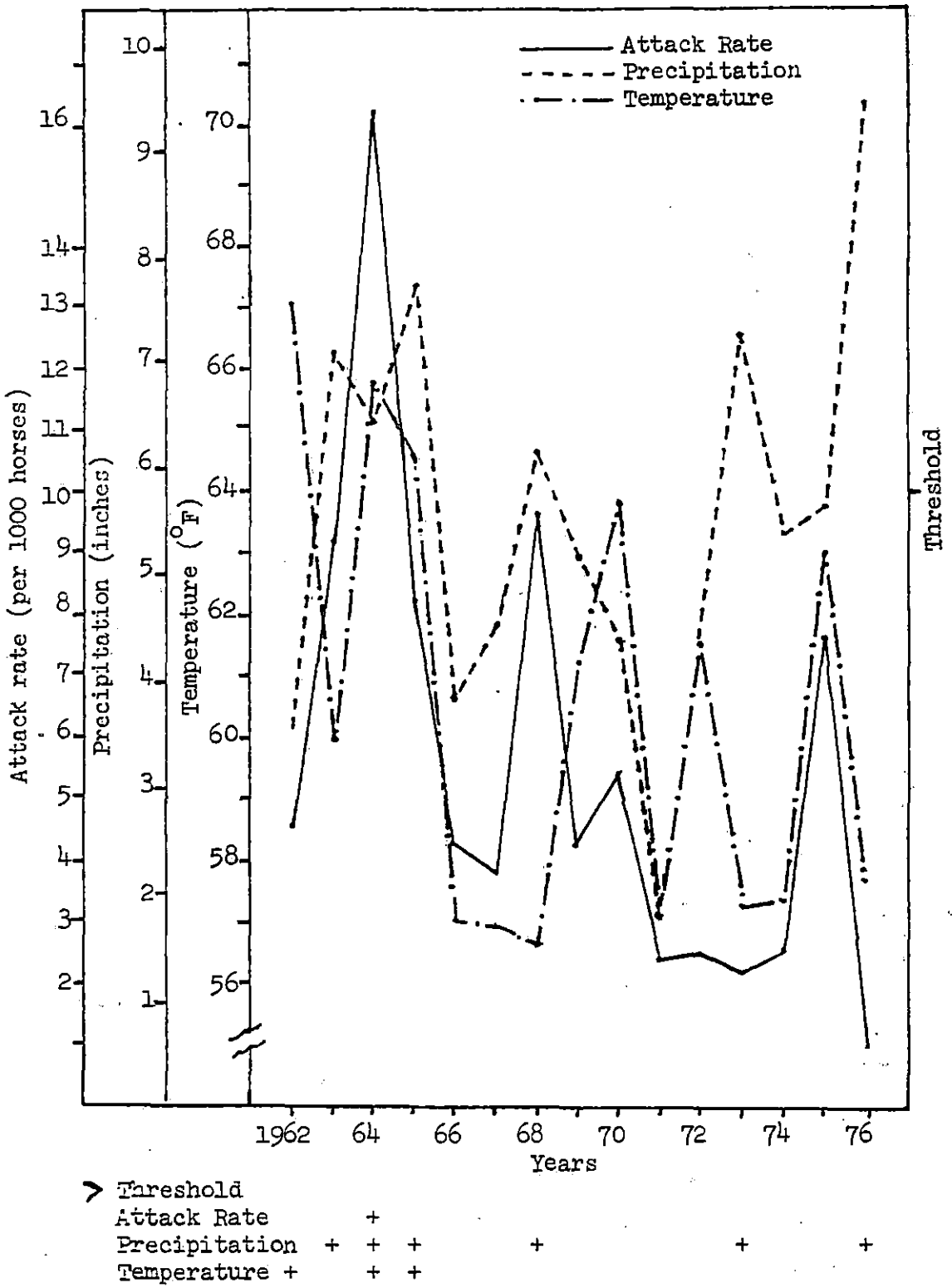


Figure 6. WEE attack rate, March and April total precipitation, and average May temperature, 1962-1976, Central Iowa

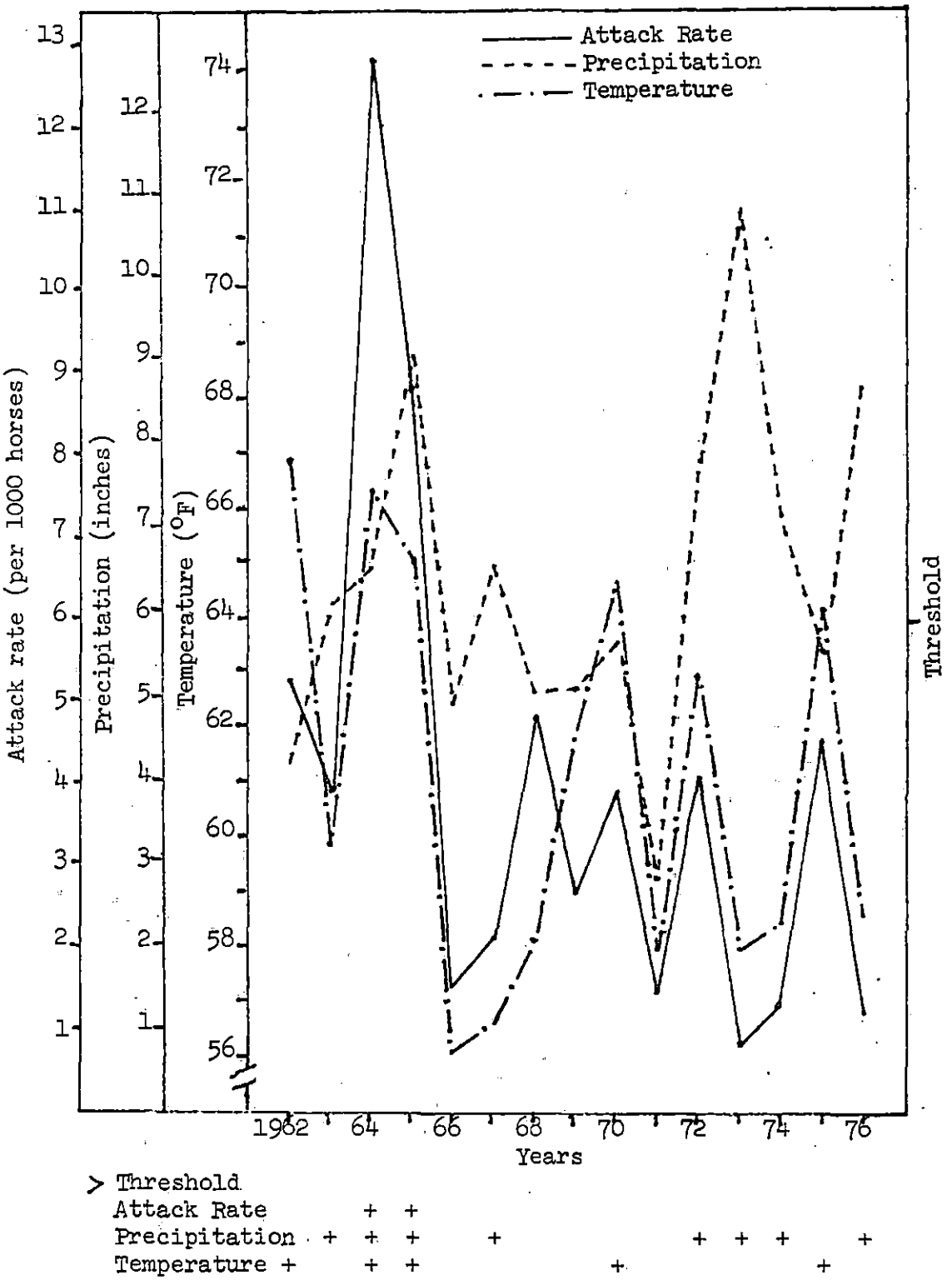


Figure 7. WEE attack rate, March and April total precipitation, and average May temperature, 1962-1976, East Central Iowa

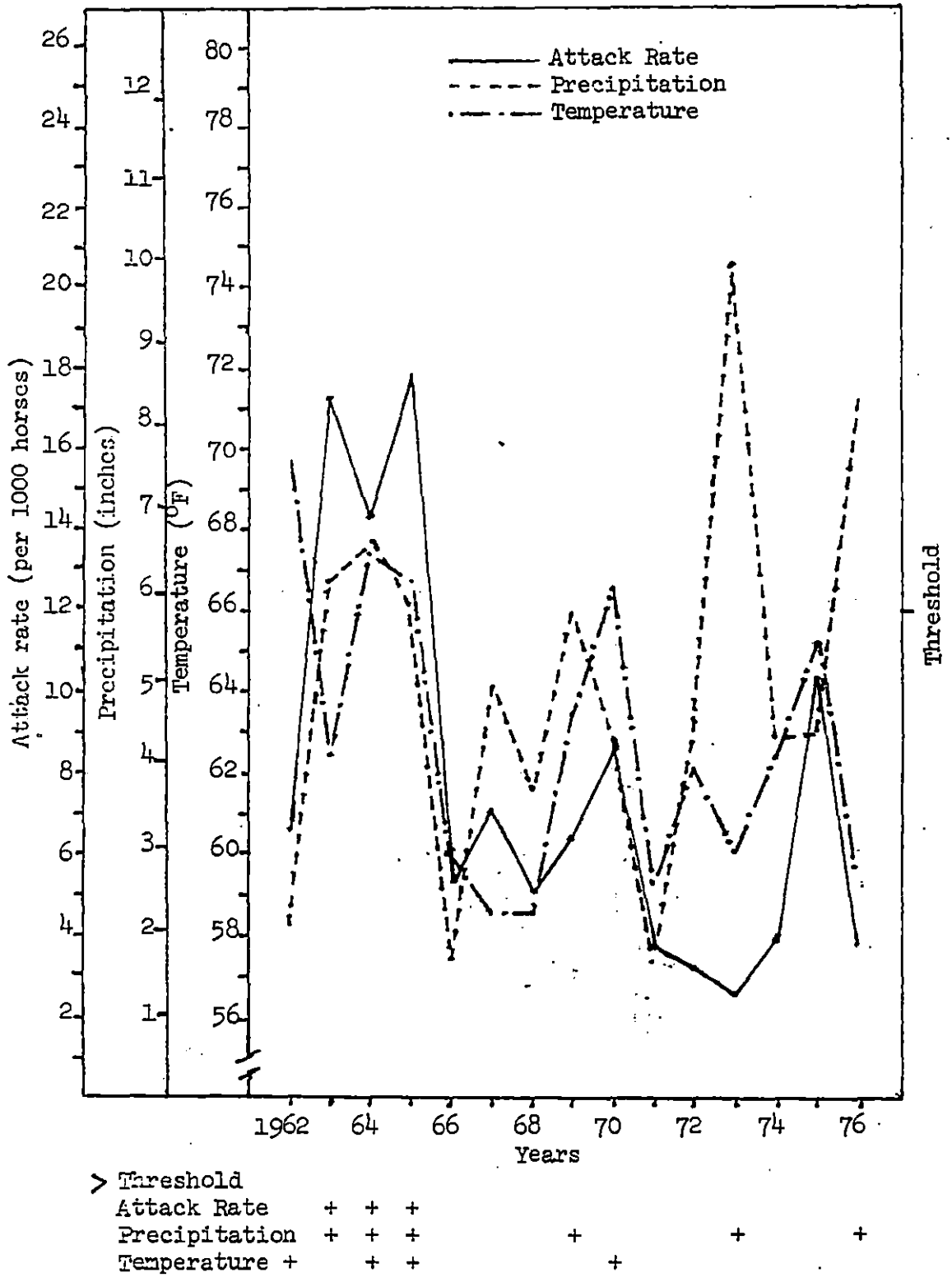


Figure 8. WEE attack rate, March and April total precipitation, and average May temperature, 1962-1976, Southwest Iowa

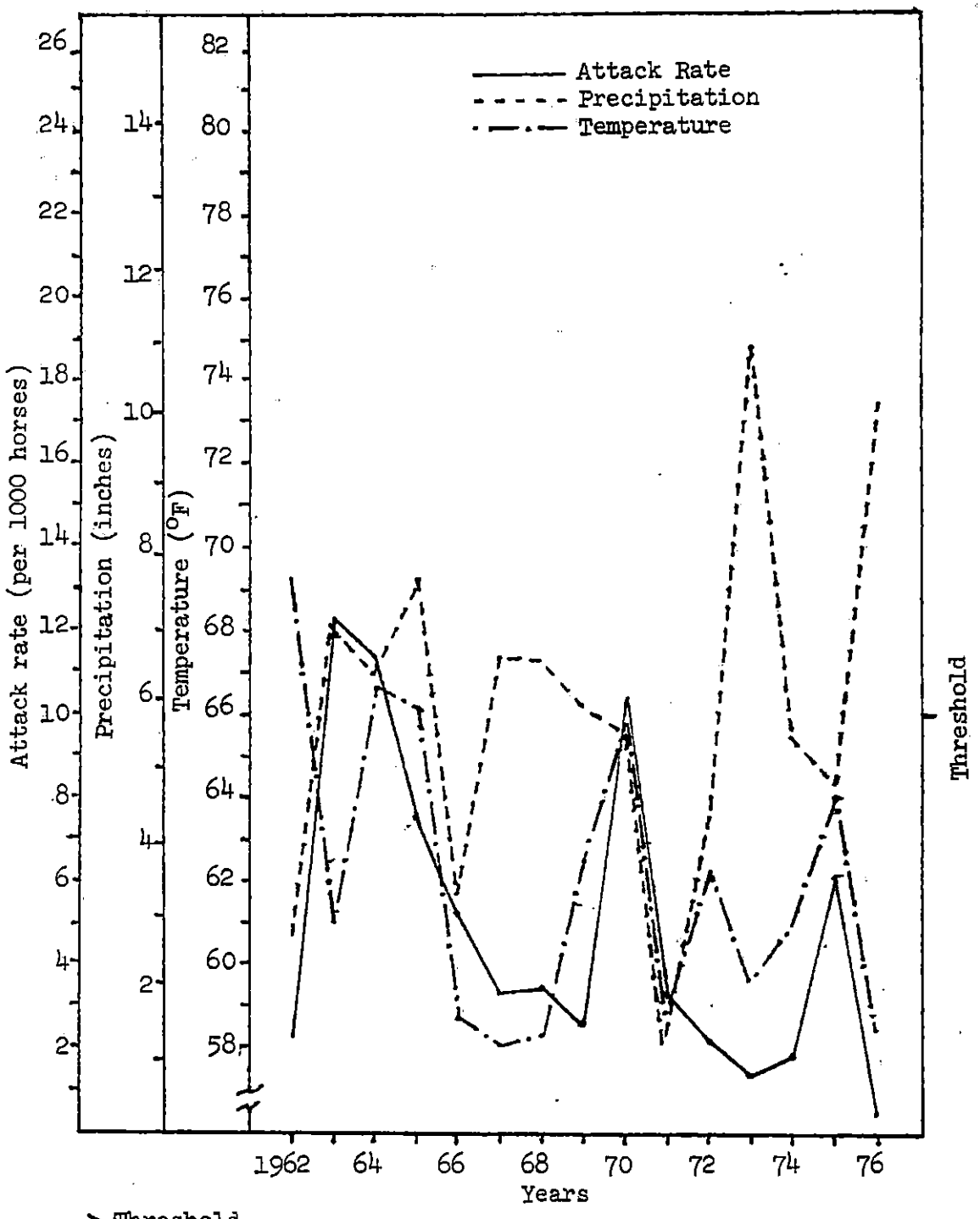


Figure 9. WEE attack rate, March and April total precipitation, and average May temperature, 1962-1976, South Central Iowa

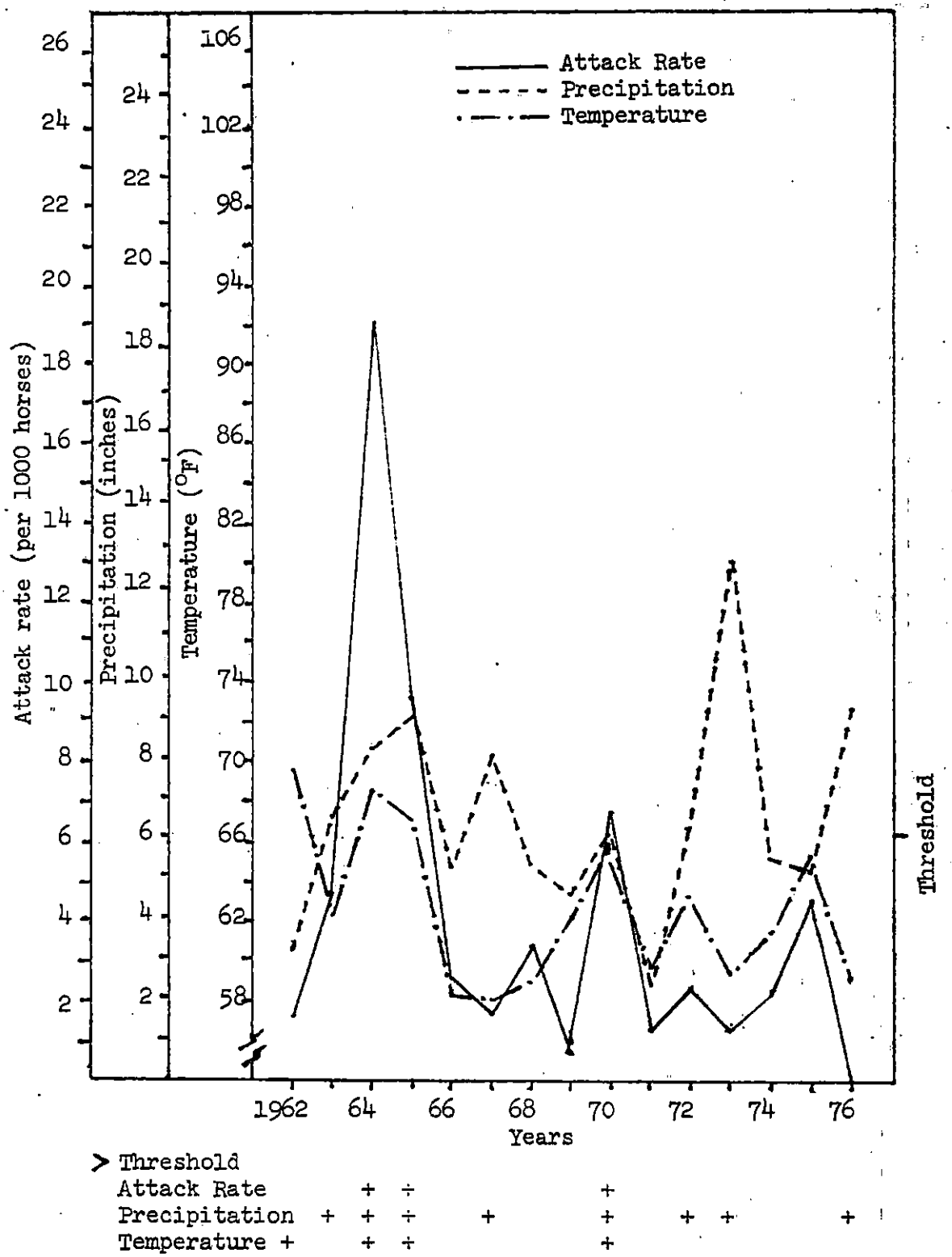
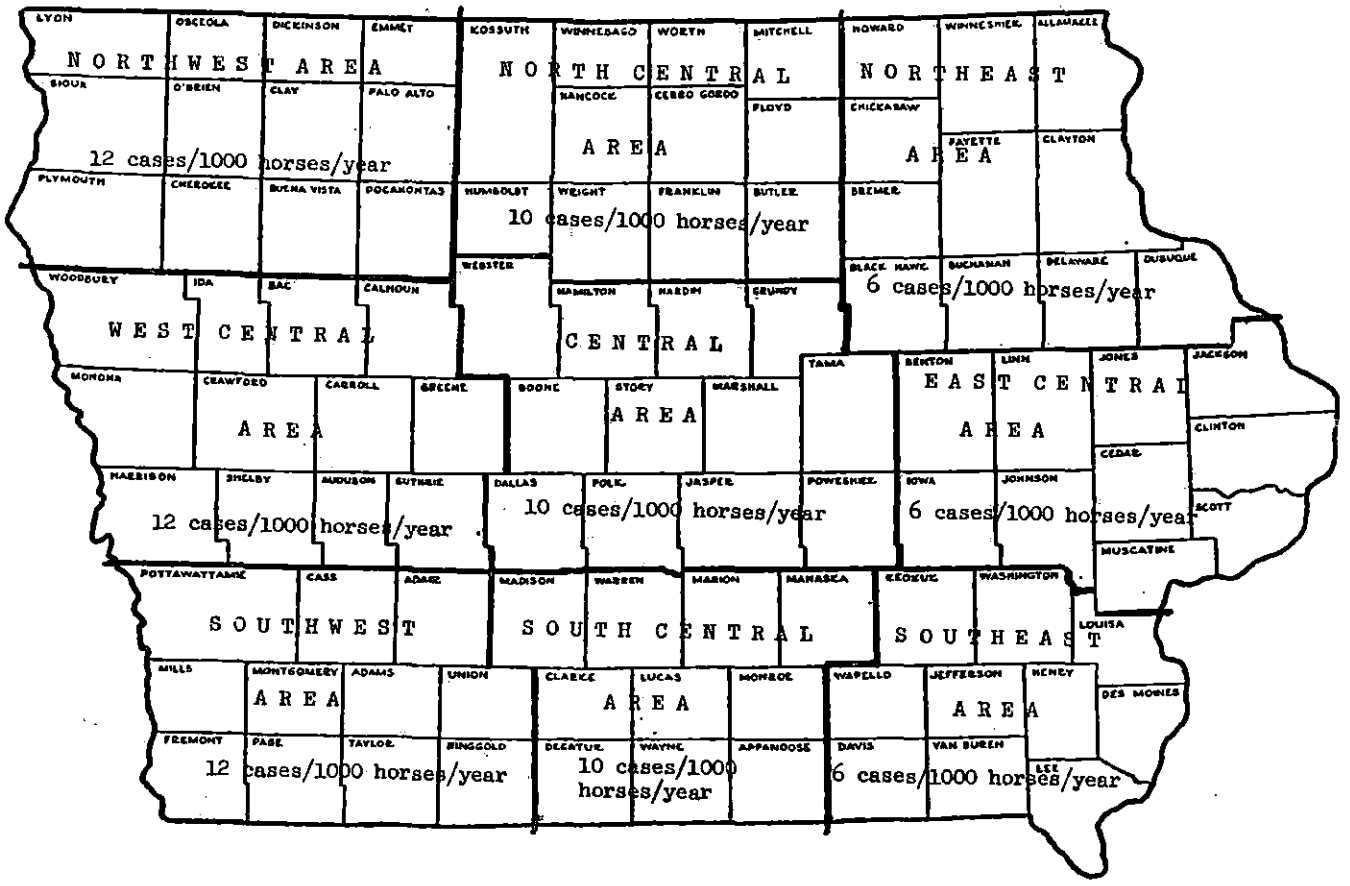


Figure 10. WEE attack rate, March and April total precipitation, and average May temperature, 1962-1976, Southeast Iowa

Figure 11. WEE epidemic level attack rate thresholds for the geographical areas of Iowa


NORTHWEST AREA
12 cases/1000 horses/year
NORTH CENTRAL AREA
10 cases/1000 horses/year
NORTHEAST AREA
6 cases/1000 horses/year
WEST CENTRAL AREA
12 cases/1000 horses/year
CENTRAL AREA
10 cases/1000 horses/year
EAST CENTRAL AREA
6 cases/1000 horses/year
SOUTHWEST AREA
12 cases/1000 horses/year
SOUTH CENTRAL AREA
10 cases/1000 horses/year
SOUTHEAST AREA
6 cases/1000 horses/year

combination of the three factors thresholds is shown at Table 4.

Table 4. Agreement of threshold precipitation and temperature with WEE attack rates, 1962-1976

Area	Factor	Threshold	62	63	64	65	66	67	68	69	70	71	72	73	74	75	76
NW	Precip	5.8"			+	+										+	
	Temp	62°	+		+	+										+	
	A.R.	12/1000			+	+										+	
NC	Precip	5.8			+	+								+		+	+
	Temp	62	+		+	+										+	
	A.R.	10			+	+										+	
NE	Precip	5.8			+	+			+					+		+	+
	Temp	62	+		+											+	
	A.R.	6.0			+											+	
WC	Precip	5.8			+	+								+		+	+
	Temp	64	+		+	+					+					+	
	A.R.	12			+	+										+	
C	Precip	5.8		+	+	+			+					+			+
	Temp	64	+		+	+											
	A.R.	10			+												
EC	Precip	5.8		+	+	+		+					+	+	+		+
	Temp	64	+		+	+					+					+	
	A.R.	6			+	+											
SW	Precip	5.8		+	+	+				+				+			+
	Temp	66	+		+	+					+						
	A.R.	12		+	+	+											
SC	Precip	5.8		+	+	+		+	+	+				+			+
	Temp	66	+		+	+											
	A.R.	10		+	+						+						
SE	Precip	5.8		+	+	+		+			+			+	+		+
	Temp	66	+		+	+					+						
	A. R.	6			+	+					+						

Regression analysis was used as an alternate means of predicting attack rates. Its use as a tool to predict outcomes of an event which is a function of several variables is well-documented (Draper and Smith 1966).

After some trial and error model attempts in one geographical region (NW Iowa), the model $y = B_o + B_1 X_1 + B_2 X_2$ with y = attack rate, x_1 = precipitation, and x_2 = temperature yielded results not quite as good as the "Threshold" or graphical procedure. Further consideration suggested that trigonometric functions might be appropriate (e.g. sinusoidal curves).

The use of trigonometric functions, however, produces accuracy problems when the magnitude of the numbers is large. For this reason data in each region were divided by the "threshold value". This produced an additional benefit in that the response variable, y, was now a function of the epidemic threshold. That is, a value $y \geq 1$ indicated epidemic levels. Thus, the model $y = B_o + B_1 X_1 + B_2 X_2 + B_3 X_3$ with (y = attack rate/threshold, x_1 = sin (temperature/threshold), X_2 = sin (precipitation/threshold), and X_3 = cos (precipitation/threshold)) was the most parsimonious; the factor cos (temperature/threshold) was discarded as it is highly correlated with sin (temperature/threshold) and added little to the regression. Adjustment of the present variation due to regression (R^2) for degrees of freedom was considered, as well.

The detailed analyses in Appendix B indicate the trigonometric prediction equations for each of the nine geographical regions,

along with a test of the significance of the regression (e.g. do these variables predict attack rate?). Statistical values for all areas are given in Table 5.

Table 5. A summary of regression analysis values by area

Region	S	R^2%	R^2(Adj)%	F_o	Significant at α =
NW	.321	69.8%	61.6%	8.50	.005
NC	.607	42.1%	26.3%	2.67	.100
NE	.544	43.6%	28.2%	2.83	.100
WC	.447	50.0%	36.4%	3.66	.050
C	.351	39.7%	23.3%	2.41	.250
EC	.404	55.8%	43.8%	4.64	.025
SW	.347	45.6%	30.8%	3.08	.100
SC	.503	35.1%	17.4%	1.99	.250
SE	.631	54.5%	40.9%	3.99	.050

The ability of these models to predict epidemics was not dependent upon the normality of the residuals or of any other variables in the model; the hypothesis tests were, however, and although no residual analyses were included, the assumptions were not hard to support. A summary of prediction ability in each of the nine regions for these methods compared to the threshold approach is shown in Table 6. These data were used to compute the specificity and sensitivity of the three methods in predicting epidemic rates using standard

Table 6. Number of epidemic years predicted by utilized methods

Region	Threshold Method				Linear Regression				Transformed Regression			
	TP	FP	FN	TN	TP	FP	FN	TN	TP	FP	FN	TN
NW	3	0	0	12	3	0	0	12	3	0	0	12
NC	3	0	0	12	3	0	0	12	3	0	0	12
NE	2	0	0	13	2	0	0	13	2	1	0	12
WC	3	0	0	12	1	0	2	12	1	0	2	12
C	1	1	0	13	0	0	1	14	0	0	1	14
EC	2	0	0	13	2	4	0	9	2	3	0	10
SW	2	0	1	12	1	0	2	12	2	0	1	12
SC	1	1	2	11	0	0	3	12	0	0	3	12
SE	3	0	0	12	3	2	0	10	3	1	0	12
Total	20	2	3	110	15	6	8	106	16	5	7	108

TP = True Positive - epidemic predicted, epidemic occurred.

FP = False Positive - predicted epidemic, no epidemic occurred.

FN = False Negative - no epidemic predicted, one occurred.

TN = True Negative - no epidemic predicted, none occurred.

statistical methods. This was done only for each region and for all regions combined. That has been summarized in Table 7 to show the range for the regions and then using total values for each method.

Table 7. Comparison of predictive methods

Test	Threshold Method		Linear Regression		Transformed Regression	
	Range	Average	Range	Average	Range	Average
Sensitivity	.33-1	.870	0-1	.652	0-1	.696
Specificity	.87-1	.982	.69-1	.946	.77-1	.955

DISCUSSION

The data as presented matched well with the mechanics of the disease and of the vector amplification processes as presented in the introduction, and showed excellent epidemic predictive value. Using the threshold method, false predictions are not difficult to explain in terms of the disease mechanics. The two false positives both took place in 1965 in the south central and central areas. The preceding year had been an epidemic one with high rates. The rate of drop off was not a greater percentage than most other areas. However, it did place them below the epidemic threshold. The most probable explanation would have been a great reduction in susceptibles due to acquired immunity through disease or immunization. Given the many factors involved, certainly others might also be possible.

The three false negatives all occurred in the southwest or south central regions. One from each area in 1963 involved average monthly temperatures in the area of 16.7°C (62°F) instead of the required 17.1°C (66°F). The third in 1970 in the south central region has precipitation .39 inches below the threshold of 5.80 inches and .1°C (.2°F) below the threshold of 17.1°C (66°F), so close that the local variations would allow this.

Regression analysis indicated that there were definite correlations between the precipitation plus temperature thresholds used, and epidemic attack rates. This certainly agreed with the multifactorial basis of the disease (Reeves 1967). The regression

analysis was clearly less sensitive and specific as an indicator. This can be attributed to the inherent difficulty in making a regression model that can predict as accurately as the threshold values. The fact that a trigonometric function best fit the data coincided well with a large body of evidence, as yet not completely explained, on sinusoidal variations in weather patterns and mathematical models of weather prediction (DeLacey, personal communication)[1].

The only other basic data for analysis were the statewide reported cases by month for the years 1969-1975 (Table 8).

Table 8. Reported equine cases of WEE and percentages of the yearly reported totals in each month in Iowa, 1969-1975

YEAR	MAY		JUNE		JULY		AUGUST		SEPTEMBER		OCTOBER		TOTAL	ATTACK RATES
	#	%	#	%	#	%	#	%	#	%	#	%	#	Cases/ 1000
1969	2	(0.8)	15	(5.9)	45	(17.6)	102	(40.0)	79	(31.0)	10	(3.9)	255	3.29
1970	5	(1.1)	19	(4.2)	65	(14.5)	184	(41.0)	157	(35.0)	16	(3.7)	449	6.18
1971	0	(0)	14	(9.5)	22	(15.0)	64	(43.5)	37	(25.2)	0	(0)	147	2.17
1972	17	(11.0)	6	(3.9)	27	(17.4)	57	(36.8)	37	(23.9)	4	(2.6)	155	2.47
1973	5	(5.3)	5	(5.3)	27	(28.4)	34	(35.8)	19	(20.0)	5	(5.3)	95	1.64
1974	4	(2.6)	9	(5.8)	19	(12.3)	57	(37.0)	60	(39.0)	6	(3.9)	154	2.90
1975[a]	0	(0)	13	(3.0)	69	(15.9)	192	(44.4)	130	(30.0)	22	(5.1)	434	9.01

[a]Epidemic year for the state overall.

[1]Delacey, P. D., Dept. of Math., U.S. Military Acedemy, 1981.

Analysis of this information indicated that there was one statewide epidemic year (1975) and one year of high incidence statewide in the six-year period, based on those definitions already discussed. August was the month of most cases of each year by a large margin, and from 35% to 44% of the total cases. September was the month with the next highest number of cases, and from 20% to 39% of the total. July was the third highest month. In 1973, July had more cases than September due to an exceptionally low number of September cases that year.

These data were analyzed for predictors of WEE in epidemic levels in horses. The first month when there were clear indicators of high levels of equine cases of WEE was July. Because the cases for August were already incubating by the time the data for July were available, this does not seem to be a timely indicator.

Several other theories proposed in the literature for the prediction of epidemic levels of WEE (primarily in the human population) were also applied to the data from the nine regions of Iowa for applicability to this state. The theory that the incidence of epidemic levels of WEE occurred above (north of) the 70°F June isotherm (Hess et al. 1963) was investigated. The 70°F June isotherm is the line of equal 70°F average monthly temperature for June (Thessen 1946). Thus, those regions with average June temperatures above 70°F would be considered to lie below (south of) the isotherm, and those with average June temperatures below 70°F to lie above (north of) the isotherm. Average June temperatures (Table A-5) were examined

for relationship to the 70°F June isotherm in all areas over the fifteen years studies. The results are summarized at Table 9.

Table 9. Relationship of average June temperatures to 70°F June isotherm

Relationship to 70°F June Isotherm	Total Years	Epidemic Years
Below	68	13
Above	67	10
Total	135	23

The data of this study did not support Hess' theory. However, this concept was based on human disease "outbreaks" with no clear definitions in terms of attack rates or epidemic levels (Hess et al. 1963). The isotherm was present in the state eleven of the fifteen study years. In 1963 and 1973 it was to the north, and in 1969 and 1974 it was to the south.

In the same article in which Hess (1963) cited his 70°F June isotherm theory, he also reported correlations between WEE disease incidence and cool wet springs with increased precipitation in the April to June period followed by hot dry summers. The April to June time period showed no consistent correlation pattern with disease at epidemic levels in the period of this study. The cool spring-hot summer proposal also did not show correlations with the Iowa data. The attack rates recorded in the study areas in Iowa

showed no particular agreement with temperature patterns in March and April. Likewise, the epidemic summers were overall not significantly warmer than the non-epidemic ones; the important temperature period appearing to be the month of May. Graham and Collett (1964) correlated high incidence of WEE with increased precipitation in May or June in Utah, a pertinent finding in the light of the evidence presented here for the importance of rain earlier in the year in Iowa.

It is improbable that the epidemic cycle would operate differently in different locations. The same factors of water and temperature availability are generally the primary variables in that cycle wherever it occurs. There may be some variation in the exact months of rainfall and temperature but the same pattern will exist. Re-examination of the data in these other studies may well show patterns similar to this study.

SUMMARY AND CONCLUSIONS

This study has utilized data from a number of sources to find indicators in regularly published meteorologic data for epidemic levels of WEE in horses in Iowa. These meterologic data and a number of theories from authors in other geographic areas were assessed for reliability in predicting the epidemic years represented in the disease data. The following conclusions were reached:

1. No existing theories reported from other geographic areas precisely fit the Iowa disease incidence and meteorological data.

2. A definite correlation exists between the defined epidemic levels of WEE in horses in Iowa for the period 1962-1976, and values over threshold level for precipitation totals for March plus April in the same year with average May temperatures over a threshold level.

3. That the proposed threshold method is more sensitive and specific than the mathematic models tested.

4. That the potential for epidemic levels of WEE will exist when the combined total precipitation for the months of March and April surpasses 5.8 inches and the May monthly average temperature exceeds 62-66^{o}F.

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APPENDIX A:

METEOROLOGICAL DATA

Table A-1. Total precipitation in inches by areas for Iowa for March, 1962-1976

YEAR	NW	NC	NE	WC	C	EC	SW	SC	SE
1962	1.78	1.31	1.96	1.74	1.53	2.27	1.04	1.09	1.74
1963	1.27	2.19	3.21	2.31	2.62	2.66	2.79	3.77	3.64
1964	1.44	1.28	1.48	1.41	1.14	1.99	1.63	1.47	2.15
1965	2.24	3.69	2.70	2.73	3.14	2.68	2.90	2.62	2.66
1966	1.17	2.59	2.72	1.41	2.19	1.93	0.81	1.22	1.55
1967	0.36	1.30	2.11	0.79	1.69	2.07	1.90	2.41	2.02
1968	0.47	0.66	1.16	0.63	1.26	1.17	0.50	0.94	1.77
1969	1.53	0.97	0.85	0.88	1.07	1.07	1.25	1.39	1.22
1970	2.08	1.62	2.05	2.32	2.81	2.97	1.74	1.97	2.27
1971	1.09	1.25	1.46	0.83	0.85	0.86	0.50	0.58	0.76
1972	0.89	1.05	1.89	0.91	1.09	2.08	0.76	1.05	1.98
1973	2.60	3.42	3.16	2.97	3.31	4.31	6.26	5.81	4.73
1974	1.34	1.65	2.18	0.87	1.39	2.61	0.56	1.66	3.01
1975	1.89	2.09	2.59	1.76	2.35	2.85	1.75	1.93	2.07
1976	3.07	3.72	3.37	3.05	3.54	4.00	2.46	3.45	3.83

Table A-2. Total precipitation in inches by areas for Iowa for April, 1962-1976

YEAR	NW	NC	NE	WC	C	EC	SW	SC	SE
1962	1.94	2.04	2.61	1.41	2.02	1.89	1.11	1.73	1.35
1963	1.28	3.20	3.15	2.68	4.42	3.50	3.66	3.26	3.03
1964	4.35	5.72	4.82	6.17	5.27	4.65	5.17	4.65	5.96
1965	3.57	4.02	4.63	3.50	4.73	6.33	3.09	5.16	6.56
1966	1.43	1.67	2.27	0.79	1.63	3.03	0.94	2.15	3.72
1967	2.53	2.47	2.59	2.48	2.86	4.41	3.17	4.32	6.05
1968	3.70	4.95	4.77	3.84	4.88	3.86	3.47	5.67	3.81
1969	1.09	2.54	3.85	2.55	4.08	4.09	4.69	4.61	3.48
1970	1.67	1.81	1.69	1.75	1.72	2.79	2.72	3.44	4.05
1971	1.09	1.00	1.44	1.21	1.11	1.87	1.33	1.70	1.39
1972	3.12	2.36	3.11	3.99	2.98	5.70	3.94	3.34	4.38
1973	2.17	3.79	5.45	3.49	4.08	6.51	3.79	5.14	7.87
1974	1.89	2.97	3.41	2.50	4.06	4.64	3.93	3.85	2.54
1975	5.13	5.01	3.69	5.11	3.39	2.80	2.78	2.95	2.96
1976	1.60	3.73	5.11	3.02	5.96	4.70	5.94	6.63	5.43

Table A-3. Total precipitation in inches by area for the months of March and April, 1962-1976

YEAR	NW	NC	NE	WC	C	EC	SW	SC	SE
1962	3.72	3.35	4.57	3.15	3.55	4.16	2.15	2.82	3.09
1963	2.55	5.39	5.36	4.99	7.04	6.16	6.45	7.03	6.67
1964	5.79	7.00	6.30	7.58	6.41	6.64	6.80	6.13	8.11
1965	5.81	7.71	7.33	6.23	7.87	9.01	5.99	7.78	9.22
1966	2.64	4.26	4.99	2.20	3.82	4.96	1.75	3.37	5.27
1967	2.89	3.77	4.70	3.27	4.55	6.48	5.07	6.73	8.07
1968	4.17	5.61	5.93	4.47	6.14	5.03	3.97	6.61	5.58
1969	2.62	3.51	4.70	3.38	5.15	5.16	5.94	6.00	4.70
1970	3.75	3.43	3.74	4.07	4.53	5.76	4.44	5.41	6.32
1971	2.18	2.25	2.90	2.04	1.86	2.73	1.83	2.28	2.15
1972	4.01	3.41	5.00	4.90	4.07	7.78	4.70	4.39	6.36
1973	4.77	7.21	8.61	7.46	7.39	10.82	10.05	10.95	12.60
1974	3.23	4.62	5.59	3.37	5.45	7.25	4.49	5.51	5.55
1975	7.02	7.10	6.28	6.87	5.74	5.65	4.53	4.88	5.03
1976	4.67	7.45	8.48	6.07	9.49	8.70	8.40	10.08	9.26

Table A-4. Average monthly May temperatures (^{o}F) by areas for Iowa, 1962-1976

YEAR	NW	NC	NE	WC	C	EC	SW	SC	SE
1962	64.0	64.1	63.7	67.0	67.0	66.9	69.6	69.3	69.7
1963	59.1	58.5	57.6	60.8	60.0	59.9	62.5	61.2	61.6
1964	63.6	64.4	63.8	66.1	65.8	66.4	67.3	67.0	68.7
1965	63.0	62.6	61.9	65.2	64.9	65.0	66.9	66.1	67.1
1966	56.5	55.4	54.2	58.7	57.0	56.3	60.1	58.8	58.2
1967	54.4	54.4	54.7	57.3	56.9	56.8	58.7	58.1	58.0
1968	54.6	55.0	55.9	57.1	56.6	58.1	58.7	58.4	59.1
1969	60.8	60.2	59.9	62.1	61.2	61.8	63.4	62.6	62.8
1970	61.9	61.6	61.7	65.1	63.8	64.7	66.6	65.8	66.1
1971	57.0	56.3	56.6	58.2	57.2	58.0	59.2	58.4	59.5
1972	60.6	61.3	61.7	61.5	61.5	63.0	62.1	62.3	63.6
1973	57.2	56.6	55.8	59.2	57.3	58.0	60.2	59.5	59.6
1974	57.3	56.4	55.7	60.0	57.4	58.5	62.6	61.0	61.1
1975	62.6	63.1	62.1	64.2	63.0	64.3	65.1	64.3	65.1
1976	58.0	57.9	56.9	59.0	57.7	58.7	59.5	58.6	59.2

Table A-5. Average monthly June temperatures by areas for Iowa, 1962-1976

YEAR	NW	NC	NE	WC	C	EC	SW	SC	SE
1962	67.3	67.7	67.2	68.9	69.4	69.7	70.5	70.3	71.5
1963	73.0	72.2	70.9	73.9	73.6	73.2	75.5	74.4	75.1
1964	69.5	69.4	68.7	69.8	69.6	70.8	70.6	70.4	72.0
1965	68.9	68.5	67.2	69.8	68.9	69.1	70.3	69.4	70.4
1966	69.0	69.3	68.5	69.8	69.3	69.9	70.2	69.3	70.4
1967	67.4	67.8	68.4	68.7	68.8	70.9	69.2	69.5	71.0
1968	70.4	69.7	69.0	72.2	71.2	71.8	73.6	72.7	73.9
1969	63.4	62.7	62.8	65.6	65.0	65.9	67.7	67.1	67.5
1970	70.9	70.1	69.2	71.7	70.8	70.3	71.9	71.2	71.4
1971	73.1	73.8	73.0	75.2	75.3	75.8	76.4	76.1	77.1
1972	68.6	67.7	66.7	70.4	68.3	68.7	71.8	70.2	70.3
1973	70.6	71.1	70.1	72.0	71.5	72.1	73.0	71.9	72.9
1974	67.6	66.8	65.0	68.8	66.8	67.0	69.6	67.6	67.7
1975	68.0	69.3	68.9	69.0	69.3	71.4	71.2	70.8	72.4
1976	67.8	69.9	68.9	70.6	69.1	70.5	71.2	70.1	70.9

APPENDIX B:

REGRESSION ANALYSIS DATA

Regression analysis data, northwest area

THE REGRESSION EQUATION IS
Y = 3.34 - 3.28 X1 - 5.33 X2
+ 4.08 X3

THE ST. DEV. OF Y ABOUT REGRESSION LINE IS
S = .321
WITH (15- 4) = 11 DEGREES OF FREEDOM

R-SQUARED = 69.8 PERCENT
R-SQUARED = 61.6 PERCENT, ADJUSTED FOR D.F.

ANALYSIS OF VARIANCE

DUE TO	DF	SS	MS=SS/DF
REGRESSION	3	2.627	.876
RESIDUAL	11	1.135	.103
TOTAL	14	3.762	

FURTHER ANALYSIS OF VARIANCE
SS EXPLAINED BY EACH VARIABLE WHEN ENTERED IN THE ORDER GIVEN

DUE TO	DF	SS
REGRESSION	3	2.627
SINP	1	1.716
COSP	1	.757
SINT	1	.155

ROW	X1 SINP	Y ATKRT	PRED. Y VALUE	ST.DEV. PRED. Y	RESIDUAL
1	.598	.392	.615	.188	-.223
2	.426	.699	.455	.142	.246
3	.841	1.717	1.189	.153	.527
4	.842	1.442	1.178	.147	.264
5	.446	.272	.341	.135	-.069
6	.478	.495	.234	.162	.261
7	.659	.507	.321	.192	.186
8	.437	.447	.509	.158	-.062
9	.602	.634	.545	.144	.089
10	.367	.207	.428	.189	-.222
11	.636	.210	.531	.125	-.321
12	.735	.347	.567	.153	-.220
13	.529	.608	.344	.104	.265
14	.936	1.602	1.650	.283	-.247
15	.721	.096	.571	.138	-.475

Regression analysis data, north central area

THE REGRESSION EQUATION IS
Y = - 11.2 + 2.85 X1 + 1.24 X2
+ 11.0 X3

THE ST. DEV. OF Y ABOUT REGRESSION LINE IS
S = .607
WITH (15- 4) = 11 DEGREES OF FREEDOM

R-SQUARED = 42.1 PERCENT
R-SQUARED = 20.3 PERCENT, ADJUSTED FOR D.F.

ANALYSIS OF VARIANCE

DUE TO	DF	SS	MS=SS/DF
REGRESSION	3	2.955	.985
RESIDUAL	11	4.059	.369
TOTAL	14	7.014	

FURTHER ANALYSIS OF VARIANCE
SS EXPLAINED BY EACH VARIABLE WHEN ENTERED IN THE ORDER GIVEN

DUE TO	DF	SS
REGRESSION	3	2.955
SINP	1	1.342
COSP	1	.013
SINT	1	1.601

ROW	X1 SINP	Y ATKRT	PRED. Y VALUE	ST.DEV. PRED. Y	RESIDUAL
1	.546	.27	.91	.34	-.65
2	.801	.64	.80	.27	-.15
3	.935	2.94	1.45	.32	1.49
4	.971	1.24	1.24	.36	-.00
5	.670	.32	.27	.27	.06
6	.609	.41	.04	.29	.37
7	.623	.64	.44	.31	.20
8	.569	.55	.59	.23	-.04
9	.556	.50	.70	.26	-.20
10	.378	.28	-.24	.52	.52
11	.355	.31	.67	.25	-.36
12	.947	.21	.67	.31	-.46
13	.715	.55	.45	.26	.10
14	.941	1.16	1.33	.28	-.16
15	.959	.05	.77	.32	-.72

Regression analysis data, northeast area

THE REGRESSION EQUATION IS
Y = - 7.02 + 1.88 X1 + .930 X2
+ 6.60 X3

THE ST. DEV. OF Y ABOUT REGRESSION LINE IS
S = .318
WITH (15- 4) = 11 DEGREES OF FREEDOM

R-SQUARED = 43.6 PERCENT
R-SQUARED = 28.2 PERCENT, ADJUSTED FOR D.F.

ANALYSIS OF VARIANCE

DUE TO	DF	SS	MS=SS/DF
REGRESSION	3	.856	.285
RESIDUAL	11	1.109	.101
TOTAL	14	1.965	

FURTHER ANALYSIS OF VARIANCE
SS EXPLAINED BY EACH VARIABLE WHEN ENTERED IN THE ORDER GIVEN

DUE TO	DF	SS
REGRESSION	3	.856
SINP	1	.062
COSP	1	.206
SINT	1	.588

ROW	X1 SINT	Y ATKRT	PRED. Y VALUE	ST.DEV. PRED. Y	RESIDUAL
1	.707	.263	.628	.153	-.364
2	.798	.105	.337	.111	-.232
3	.883	1.150	.742	.159	.408
4	.953	.603	.612	.149	-.008
5	.758	.160	.084	.166	.076
6	.722	.357	.089	.152	.268
7	.653	.250	.256	.141	-.006
8	.735	.160	.422	.102	-.242
9	.601	.523	.399	.171	.125
10	.471	.117	-.071	.259	.188
11	.759	.102	.559	.118	-.457
12	.996	.063	.114	.222	-.050
13	.821	.112	.228	.142	-.116
14	.883	1.212	.646	.134	.565
15	.994	.048	.202	.204	-.154

Regression analysis data, west central area

THE REGRESSION EQUATION IS
Y = - 3.18 - .913 X1 - 1.70 X2
+ 6.65 X3

THE ST. DEV. OF Y ABOUT REGRESSION LINE IS
S = .372
WITH (15- 4) = 11 DEGREES OF FREEDOM

R-SQUARED = 50.0 PERCENT
R-SQUARED = 36.4 PERCENT, ADJUSTED FOR D.F.

ANALYSIS OF VARIANCE

DUE TO	DF	SS	MS=SS/DF
REGRESSION	3	1.525	.508
RESIDUAL	11	1.524	.139
TOTAL	14	3.049	

FURTHER ANALYSIS OF VARIANCE
SS EXPLAINED BY EACH VARIABLE WHEN ENTERED IN THE ORDER GIVEN

DUE TO	DF	SS
REGRESSION	3	1.525
C6	1	.897
C7	1	.149
C9	1	.479

ROW	X1 C6	Y C4	PRED. Y VALUE	ST.DEV. PRED. Y	RESIDUAL
1	.517	.217	.653	.257	-.437
2	.758	.782	.430	.151	.352
3	.965	1.586	1.206	.254	.381
4	.879	1.002	.870	.162	.131
5	.370	.351	.165	.219	.185
6	.534	.360	.088	.166	.272
7	.697	.398	.143	.203	.255
8	.550	.209	.387	.153	-.178
9	.646	.523	.591	.183	-.068
10	.545	.179	.161	.247	.019
11	.748	.159	.462	.148	-.302
12	.900	.108	.777	.246	-.669
13	.549	.289	.261	.125	.028
14	.926	1.369	.941	.165	.428
15	.806	.121	.478	.175	-.357

Regression analysis data, central area

THE REGRESSION EQUATION IS
Y = - 5.34 + 1.89 X1 + .931 X2
+ 4.85 X3

THE ST. DEV. OF Y ABOUT REGRESSION LINE IS
S = .351
WITH (15- 4) = 11 DEGREES OF FREEDOM

R-SQUARED = 39.7 PERCENT
R-SQUARED = 23.3 PERCENT, ADJUSTED FOR D.F.

ANALYSIS OF VARIANCE

DUE TO	DF	SS	MS=SS/DF
REGRESSION	3	.892	.297
RESIDUAL	11	1.353	.123
TOTAL	14	2.244	

FURTHER ANALYSIS OF VARIANCE
SS EXPLAINED BY EACH VARIABLE WHEN ENTERED IN THE ORDER GIVEN

DUE TO	DF	SS
REGRESSION	3	.892
SINP	1	.264
COSP	1	.290
SINT	1	.338

ROW	X1 SINP	Y ATKRT	PRED. Y VALUE	ST.DEV. PRED. Y	RESIDUAL
1	.575	.450	.706	.218	-.256
2	.937	.917	.666	.124	.251
3	.894	1.630	.920	.170	.710
4	.977	.814	.823	.193	-.009
5	.612	.432	.523	.156	.109
6	.706	.374	.419	.155	-.045
7	.872	.964	.515	.168	.449
8	.776	.436	.676	.113	-.240
9	.704	.536	.724	.137	-.188
10	.315	.242	-.083	.297	.325
11	.646	.252	.566	.121	-.314
12	.956	.213	.525	.157	-.312
13	.807	.249	.525	.151	-.276
14	.856	.772	.790	.127	-.018
15	.998	.105	.289	.298	-.184

Regression analysis data, east central area

THE REGRESSION EQUATION IS
Y = - 9.69 + 1.05 X1 + .357 X2
+ 11.4 X3

THE ST. DEV. OF Y ABOUT REGRESSION LINE IS
S = .404
WITH (15- 4) = 11 DEGREES OF FREEDOM

R-SQUARED = 55.8 PERCENT
R-SQUARED = 43.8 PERCENT, ADJUSTED FOR D.F.

ANALYSIS OF VARIANCE

DUE TO	DF	SS	MS=SS/DF
REGRESSION	3	2.270	.757
RESIDUAL	11	1.797	.163
TOTAL	14	4.067	

FURTHER ANALYSIS OF VARIANCE
SS EXPLAINED BY EACH VARIABLE WHEN ENTERED IN THE ORDER GIVEN

DUE TO	DF	SS
REGRESSION	3	2.270
SINP	1	.128
COSP	1	.296
SINT	1	1.846

ROW	X1 SINP	Y ATKRT	PRED. Y VALUE	ST.DEV. PRED. Y	RESIDUAL
1	.66	.88	1.16	.25	-.28
2	.87	.64	.61	.14	.04
3	.91	2.15	1.26	.19	.89
4	1.00	1.47	1.08	.22	.38
5	.75	.26	.15	.26	.11
6	.90	.37	.28	.20	.09
7	.76	.80	.35	.16	.45
8	.78	.44	.75	.13	-.31
9	.64	.65	1.07	.16	-.42
10	.45	.24	.11	.33	.14
11	.97	.68	.94	.15	-.25
12	.96	.14	.21	.35	-.07
13	.95	.23	.47	.17	-.25
14	.83	.75	1.03	.15	-.27
15	1.00	.21	.46	.18	-.25

Regression analysis data, southwestern area

THE REGRESSION EQUATION IS
Y = - 5.89 + 1.41 X1 + .741 X2
+ 6.25 X3
THE ST. DEV. OF Y ABOUT REGRESSION LINE IS
S = .347
WITH (15- 4) = 11 DEGREES OF FREEDOM

R-SQUARED = 45.6 PERCENT
R-SQUARED = 30.8 PERCENT, ADJUSTED FOR D.F.

ANALYSIS OF VARIANCE

DUE TO	DF	SS	MS=SS/DF
REGRESSION	3	1.111	.370
RESIDUAL	11	1.324	.120
TOTAL	14	2.435	

FURTHER ANALYSIS OF VARIANCE
SS EXPLAINED BY EACH VARIABLE WHEN ENTERED IN THE ORDER GIVEN

DUE TO	DF	SS
REGRESSION	3	1.111
SINP	1	.239
COSP	1	.405
SINT	1	.468

	X1	Y	PRED. Y	ST.DEV.	
ROW	SINP	ATKRT	VALUE	PRED. Y	RESIDUAL
1	.362	.547	.751	.260	-.205
2	.897	1.422	.779	.123	.644
3	.922	1.196	1.025	.168	.171
4	.959	1.489	1.009	.152	.480
5	.297	.459	.177	.210	.282
6	.767	.588	.525	.178	.063
7	.632	.417	.434	.171	-.016
8	.954	.520	.825	.119	-.305
9	.693	.723	.915	.139	-.191
10	.310	.317	.140	.212	.177
11	.725	.262	.697	.117	-.434
12	.987	.214	.328	.300	-.114
13	.699	.331	.708	.112	-.377
14	.704	.864	.845	.119	.019
15	.993	.329	.505	.182	-.176

Regression analysis data, south central area

THE REGRESSION EQUATION IS
Y = - 4.01 + 1.59 X1 + .729 X2
+ 3.59 X3

THE ST. DEV. OF Y ABOUT REGRESSION LINE IS
S = .352
WITH (15- 4) = 11 DEGREES OF FREEDOM

R-SQUARED = 35.1 PERCENT
R-SQUARED = 17.4 PERCENT, ADJUSTED FOR D.F.

ANALYSIS OF VARIANCE

DUE TO	DF	SS	MS=SS/DF
REGRESSION	3	.739	.246
RESIDUAL	11	1.366	.124
TOTAL	14	2.105	

FURTHER ANALYSIS OF VARIANCE
SS EXPLAINED BY EACH VARIABLE WHEN ENTERED IN THE ORDER GIVEN

DUE TO	DF	SS
REGRESSION	3	.739
SINP	1	.092
COSP	1	.485
SINT	1	.163

ROW	X1 SINP	Y ATKRT	PRED. Y VALUE	ST.DEV. PRED. Y	RESIDUAL
1	.467	.131	.488	.263	-.357
2	.936	1.218	.606	.128	.612
3	.871	1.141	.782	.166	.359
4	.974	.757	.727	.174	.030
5	.549	.522	.261	.183	.261
6	.917	.357	.506	.174	-.149
7	.908	.362	.516	.168	-.154
8	.860	.245	.645	.117	-.400
9	.803	1.063	.715	.139	.348
10	.363	.321	.047	.255	.274
11	.667	.222	.517	.114	-.295
12	.950	.136	.088	.278	.048
13	.813	.196	.571	.122	-.375
14	.746	.608	.629	.117	-.021
15	.986	.041	.221	.210	-.180

Regression analysis data, southeastern area

THE REGRESSION EQUATION IS
Y = - 11.7 + 1.96 X1 - .0800 X2
+ 13.4 X3

THE ST. DEV. OF Y ABOUT REGRESSION LINE IS
S = .631
WITH (14- 4) = 10 DEGREES OF FREEDOM

R-SQUARED = 54.5 PERCENT
R-SQUARED = 40.9 PERCENT, ADJUSTED FOR D.F.

ANALYSIS OF VARIANCE

DUE TO	DF	SS	MS=SS/DF
REGRESSION	3	4.782	1.594
RESIDUAL	10	3.986	.399
TOTAL	13	8.768	

1
FURTHER ANALYSIS OF VARIANCE
SS EXPLAINED BY EACH VARIABLE WHEN ENTERED IN THE ORDER GIVEN

DUE TO	DF	SS
REGRESSION	3	4.782
SINP	1	2.120
COSP	1	.018
SINT	1	2.644

ROW	X1 SINE	Y ATKRT	PRED. Y VALUE	ST.DEV. PRED. Y	RESIDUAL
1	.51	.27	.89	.46	-.61
2	.91	.79	.83	.22	-.04
3	.99	3.21	1.79	.55	1.43
4	1.00	1.63	1.66	.34	-.03
5	.79	.44	.14	.30	.30
6	.98	.31	.54	.34	-.22
7	.82	.53	.32	.27	.21
8	.72	.16	.58	.21	-.42
9	.89	1.13	1.29	.24	-.16
10	.36	.23	-.56	.48	.79
11	.89	.57	1.02	.21	-.64
12	.82	.21	.48	.59	-.27
13	.82	.35	.56	.21	-.21
14	.76	.76	.92	.22	-.14