Sociocultural effects of epidemics on the Northern Plains

John F. Taylor

The University of Montana

Follow this and additional works at: https://scholarworks.umt.edu/etd
Let us know how access to this document benefits you.

Recommended Citation

https://scholarworks.umt.edu/etd/8873

This Thesis is brought to you for free and open access by the Graduate School at ScholarWorks at University of Montana. It has been accepted for inclusion in Graduate Student Theses, Dissertations, & Professional Papers by an authorized administrator of ScholarWorks at University of Montana. For more information, please contact scholarworks@mso.umt.edu.
COPYRIGHT ACT OF 1976

THIS IS AN UNPUBLISHED MANUSCRIPT IN WHICH COPYRIGHT SUBSISTS. ANY FURTHER REPRINTING OF ITS CONTENTS MUST BE APPROVED BY THE AUTHOR.

MANSFIELD LIBRARY
UNIVERSITY OF MONTANA
DATE: 1983
SOCIOCULTURAL EFFECTS OF EPIDEMICS
ON THE NORTHERN PLAINS: 1735-1870

by

John F. Taylor
B.A., University of Pennsylvania, 1974

Presented in partial fulfillment of the requirements for
the degree of
Master of Arts
University of Montana
1982

Approved by:

Chairman, Board of Examiners

Dean, Graduate School

Date 2/9/83
ABSTRACT

Taylor, John F., M.A. June 1982 Anthropology

Sociocultural Effects of Epidemics on the Northern Plains: 1735-1870 (135 pp.)

Director: Dr. Charline G. Smith

Historical documents from the 1735 to 1870 period relating to the Northern Plains area were examined to determine what sociocultural effects epidemic diseases had on Native American ethnic groups residing there. Descriptions of aboriginal and post-contact diseases were analyzed in comparison with modern medical texts. Documents of Native American origin, those relating to Native American traditions, and journals of Euroamerican travelers and the Bureau of Indian Affairs were surveyed.

Aboriginal disease patterns are characterized as primarily low-order infections with case mortality chiefly among the young and the old. Epidemic diseases are not recognized as such. Post-contact diseases are high-order epidemic diseases with a high and general case mortality. A discussion of aboriginal diseases and an historical overview of the history of epidemic diseases on the Northern Plains is given.

Sociocultural effects of epidemic mortality are discussed in terms of temporary sociocultural disruption, demographic and territorial change, and permanent sociocultural change. Together, these changes promoted polyethnic coresidency and fused ethnicity by eroding the boundaries of ethnic groups. Intraethnic changes tended toward the development of achieved status sets and cognatic kinship networks. A comparison of ethnohistorical data from the Northern Plains with modern ethnographic data from the Amazon Bush of South America indicates a general correlation of sociocultural change influenced by epidemic disease mortality.
As they were standing by the river, the woman said to him, "How is it? Will we always live, will there be no end to it?" He said: "I have never thought of that. We will have to decide it. I will take this buffalo chip and throw it in the river. If it floats, when people die, in four days they will become alive again; they will die for only four days. But if it sinks, there will be an end to them." He threw the chip into the river, and it floated. The woman turned and picked up a stone, and said: "No, I will throw this stone in the river; if it floats we will always live, if it sinks people must die, that they may always be sorry for each other." The woman threw the stone into the water, and it sank. "There," said Old Man, "you have chosen. There will be an end to them."

(Grinnel 1962:138-139)

The second day after this dreadful disease broke out in our camp, and spread from one tent to another as if the Bad Spirit carried it. We had no belief that one Man could give it to another....When at length it left us, and we moved about to find our people, it was no longer with the song, and the dance; but with tears, shrieks, and howlings of despair for those would never return to us.  

(Thompson 1915:337-338)

Little Old Man, a very brave man, donned his war-dress, mounted his war-horse, and rode through the camp with a lance in his hand shouting, "If I could see this thing (the cholera), if I know where it came from, I would go there and fight it!" As he was doing this he was seized with the cramps, fell from his horse, and died in his wife's arms. 

(Grinnell 1972:164-165)
TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABSTRACT.................................................</td>
<td>i</td>
</tr>
<tr>
<td>PREFACE..................................................</td>
<td>ii</td>
</tr>
<tr>
<td>INTRODUCTION.............................................</td>
<td>1</td>
</tr>
<tr>
<td>CHAPTER I - NATIVE AMERICAN DISEASES OF THE NORTHERN PLAINS AREA..........................</td>
<td>4</td>
</tr>
<tr>
<td>Precontact Aboriginal Diseases...........................</td>
<td>8</td>
</tr>
<tr>
<td>CHAPTER II - EPIDEMICS ON THE NORTHERN PLAINS...............</td>
<td>29</td>
</tr>
<tr>
<td>Methodology................................................</td>
<td>30</td>
</tr>
<tr>
<td>Chronology of Epidemics..................................</td>
<td>34</td>
</tr>
<tr>
<td>1733-1735: Smallpox........................................</td>
<td>34</td>
</tr>
<tr>
<td>1750: Smallpox and Measles..................................</td>
<td>37</td>
</tr>
<tr>
<td>1780-1782: Smallpox........................................</td>
<td>37</td>
</tr>
<tr>
<td>1798-1799: Steptococcal Infection, Puerperal Fever........</td>
<td>42</td>
</tr>
<tr>
<td>1800-1803: Pandemic--Smallpox, Measles, and Respiratory Infections......................</td>
<td>45</td>
</tr>
<tr>
<td>1806-1814: Whooping Cough and Streptococcal Infections........................</td>
<td>47</td>
</tr>
<tr>
<td>1819-1820: Measles and Whooping Cough......................</td>
<td>49</td>
</tr>
<tr>
<td>1831-1832: Smallpox........................................</td>
<td>51</td>
</tr>
<tr>
<td>1837-1838: Smallpox........................................</td>
<td>56</td>
</tr>
<tr>
<td>1846-1847: Measles and Whooping Cough......................</td>
<td>60</td>
</tr>
<tr>
<td>1849: Cholera..............................................</td>
<td>64</td>
</tr>
<tr>
<td>1850-1851: Smallpox........................................</td>
<td>66</td>
</tr>
<tr>
<td>1851: Influenza, Cholera...................................</td>
<td>68</td>
</tr>
<tr>
<td>1856-1857: Smallpox........................................</td>
<td>70</td>
</tr>
</tbody>
</table>
INTRODUCTION

The 1945 study of smallpox epidemics by Stearn and Stearn marked the first synthesis of ethnohistorical data relevant to the effects of introduced diseases on New World populations. Because these writers were concerned only with smallpox and were treating the subject on a continent-wide basis, many fine details were lost in this work. Ewers (1973) realized this problem and from secondary sources compiled an impressive list of epidemics that struck Native American ethnic groups in Texas (1973:108-109). From this list, Ewers brought into question the estimates of aboriginal precontact population figures given by Mooney (1929) and Kroeber (1939) and documented that epidemics other than smallpox could and did result in high mortalities. Dobyns (1966) had earlier brought these same estimates into question from epidemic disease records from Mexico and postulated that aboriginal populations before the introduction of Euroamerican diseases were approximately twenty times those of the minimum populations of subject groups during the historic period. From studies of specific ethnic groups bordering the Northern Plains, Bowers (1950, 1965), Deetz (1965), and Hyde (1974) have also postulated precontact population figures for the Mandan, Hidatsa, Arikara, and Pawnee groups to be significantly higher than the Pawnee groups (Mooney 1929).

These high precontact population estimates have a great impact on the conceptualization of the dynamics of culture change and continuity among ethnic groups of the New World generally and
among Northern Plains groups specifically. The extension of a synchronic "timeless" concept to the Northern Plains peoples appears to be somewhat hampered should these ethnic groups be realized as the survivors of major and culturally inexplicable depopulation episodes. Direct trauma, organizational regroupings, and a basic restriction in the social network of individuals within an ethnic group cannot help but have some sociocultural effect on the group, either temporarily, or permanently, or both. This is not to say that cultural continuity and change of Northern Plains ethnic groups occurred solely as a result of depopulation due to introduced Euroamerican diseases, but rather it is one factor in a web of cultural influences including the environment (Oliver 1962), trade in native and Euroamerican items (Blakeslee 1975, Jablow 1950), the acquisition of the horse (Ewers 1955), and general interethnic contacts (Bruner 1961).

Epidemics of introduced Euroamerican diseases afflicting Native American ethnic groups on the Northern Plains fulfilled criteria for sociocultural change by profoundly altering the membership composition of the co-residence units and ethnic groups. The sheer loss of group members and their cultural inventory inevitably had some impact, but greater disruption and subsequent change occurred when sociotechnical demands for personnel to occupy subsistence, defense, and cultural continuity roles led to the incorporation of one or more co-residence groups into a new group or even the fusion of one or more subethnic groups into a new basic group of that order. Refugees and their descendants formed new lines of communication that cut across
ethnic boundaries to form new definitions of personal and group identity.

Endemic diseases and epidemic diseases of low and differential mortality had a similar effect, although much less marked. In diseases of these types, the very old or the very young died, or the fertility of women was impaired. Since these diseases, as well as the high mortality epidemics, were outside native conceptualizations, treatments, and cures, they formed a discordance within belief systems that provided an entree for Euroamerican medicine and, consequently, Euroamerican concepts. A basic result of the minor diseases was a heightened childhood mortality with a concurrent reduction in the replacement population for ethnic groups in the ascendant generations. Coupled with high mortality epidemics, this trend exaggerated the effects of the former with each succeeding generation.

With the apparent intensification of interethnic warfare beginning about 1730, aided by the spread of the horse and the gun "frontier" (Secoy 1953) into the Northern Plains and the progressive extinction of the bison in response to the Euroamerican trade (Roe 1953) into the Northern Plains, this downward populational spiral led to the near extinction of Northern Plains ethnic groups by the 1890s (Wissler 1936) and has been suggested to be a reason for the present-day high fertility rate of their descendants (Liberty 1973).
CHAPTER 1

NATIVE AMERICAN DISEASES OF THE NORTHERN PLAINS AREA

Introduction

Native American diseases of the Northern Plains area have been divided into two categories for the purpose of this discussion: precontact aboriginal disease and those of introduced Euroamerican origin. This division is based both on the medical characteristics of the diseases and the recognition of these diseases within the cognitive sets of the ethnic groups inhabiting this area during the historic period. Such recognition implies a realization of the disease type, course of progression, prognosis, and medical care designed to relieve symptoms and ideally cure the disease within precontact cultural patterns. The precontact aboriginal diseases, in other words, were those regarded by historic ethnic groups as being normal and treatable within their existing cultural inventories, whether curable or not. Since the bulk of data referring to diseases in the Northern Plains area is generated from historic and ethnographic sources rather than from formal pathological studies of mummified cadavers or skeletal remains, this ethnic control becomes extremely valuable in the analysis of documentary sources.

One prime example of the ethnic non-recognition of introduced Euroamerican diseases is the use of the sweat lodge as a cure for those diseases. The actual design of sweat lodge construction appears to have varied slightly from group to group on the Northern Plains, but all share a basic framework of willow

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
poles over which a covering of hides, grass, conifer boughs, or bark was placed. Once the lodge was constructed, heated rocks were carried into the structure and over these rocks was poured a decoction of herbs. Many of these herbs contain active agents recognized as having analgesic, febrifugic, suporific, and decongestant properties (Vogel 1973); in particular, willow bark contains salicin, the active ingredient in aspirin, which works to relieve pain and fevers (Vogel 1973:379-380). After a period of sweating within the sweat lodge, the patient would bathe in the nearest body of water.

From the universality of the usage of the sweat lodge on the Northern Plains, it appears that the sweat lodge treatment was a recognized cure for nearly all aboriginal precontact diseases in this area and especially for respiratory diseases. The medical properties of many of the herbs in the decoctions, as well as those of the willow structure, are recognized in modern medicine. However, when this treatment was applied to the high order infections characteristic of introduced Euroamerican diseases such as smallpox, cholera, measles, whooping cough, epidemic influenza, and others, all which show respiratory involvement as part of the symptomatic progression of these diseases, the sweat lodge treatment was fatal. It is likely the vapor bath portion of the treatment would have been effective had it not been followed by the plunge into cold water. With the high body temperatures accompanying high order infections exaggerated by the high temperatures within the sweat lodge, this plunge appears to have induced hypothermia, a physiological shock reaction which can terminate in cardiac arrest.
After the 1780-82 smallpox epidemic, the cold water plunge was recognized by some Native Americans as being fatal (Thompson 1915:337), yet it continued to be used as a medical treatment for high order infections as late as 1869 with the same disastrous effects (Koch 1896:296). Euroamericans nearly unanimously recognized the futility of the sweat lodge (Denig 1961:116) and, in at least one case (Teit 1930:316), actively prevented its use during a measles epidemic. In another case, the cold water plunge was used as a method in interethnic warfare: the Small Robes Piegan women captured by the Crow in 1845 urged the use of the cold water bath as a measles remedy when they spread this disease to their captors (De Smet 1906:242-243). The sweat lodge is still being used today by Native Americans but largely only in ceremonial contexts.

This lack of understanding of the different characteristics of introduced Euroamerican diseases by the Native American ethnic groups on the Northern Plains is based directly on the medical characteristics of these diseases. Aboriginal precontact diseases were either those brought across the Bering Strait by the small nomadic bands peopling the New World between 30,000 to 12,000 years ago, or they developed within the New World. Because of the small numbers of people in each band, only endemic diseases could be carried by these emigrant populations from the Old World; the minimum population necessary to carry high order epidemic infections, estimated at above 500,000 people (Cockburn 1971:51), would not be reached within a limited area until the development of agriculture in the New World. This minimum population is
necessary for the propagation of the epidemic diseases because these diseases either kill the victim or confer immunity with the infection. Thus, a reservoir of uninfected non-immune individuals, usually cohorts of children born after each epidemic, is needed to sustain the epidemic. It is significant that the only known precontact aboriginal epidemic disease in the New World was the Aztec Matlazahuatl, apparently a typhus characterized by spots on the skin, chronic nosebleed, and an epidemic nature. Records and works of art indicate this disease was most active between Mexico and Peru (Newman 1976:669) where there were high population densities. Since population densities on the Northern Plains never reached those of Mexico and Peru, it is doubtful that any epidemic diseases could have been sustained in this area.

Coupled with this lack of the minimum population necessary to sustain an epidemic disease is the fact that the peopling of the New World entailed a passage through subarctic conditions in Siberia and Alaska during the Pleistocene epoch. This "cold screen" may have prohibited the passage of many pathogens into the New World, yellow fever among them (Newman 1976:668). Another inhibiting factor to the importation of Old World diseases was the absence of domesticated animals other than the dog accompanying the early emigrants. Smallpox, measles, and possibly cholera were all probably evolved from bovine or swine prototype infections (Fenner 1970:64-65) and thus could not be expected to develop in the New World. The possible development of specific New World diseases attendant to the domestication of camelids and the turkey has not yet been addressed in print, should such infections ever
have occurred. The possible New World development of syphilis
will be discussed in detail below.

The route of disease infection in contacts between Native
Americans and Euroamericans is thus a largely one-way transfer of
high mortality inducing infections to the aboriginal groups.
With the exception of the urbanized Mesoamerican area with the
epidemic Matlazahuatl, these diseases were completely outside the
conceptualization of disease of the ethnic groups involved. Not
only were mortality rates extremely high for the previously uninfec­
ted peoples, but cultural mechanisms for allocating individuals
to subsistence activities were disrupted by the affliction of
whole camps and, consequently, many people died of starvation and
thirst. In addition to these deaths by natural causes, the ex­
treme disfigurement caused by smallpox, the terrifying swiftness
of cholera mortality, and the loss of friends and kindred con­
tributed to deaths by suicide. As early as 1850, observers of
the Northern Plains ethnic groups found these groups to have
"...a gloomy prospect for the future" (Mitchell 1851:324), par­
tially as a result of the introduced Euroamerican diseases.

Precontact Aboriginal Diseases

A recent overview of precontact aboriginal diseases in the
New World by Newman (1976) suggests that 13 major diseases or
disease types existed in North and South America before Euroameri­
can contact. These diseases are bacillary and ameobic dysentery,
viral influenza and pneumonia, viral arthritides, various rickett­
sial fevers such as Carrion's disease communicated through insects,
various unnamed viral infections, American leishmaniasis, American trypanosomiasis, round worms and other endoparasites, non-venereal syphilis and pinta, a range of nutritional deficiency diseases such as goiter, a range of bacterial pathogens such as streptococcus and staphylococcus, salmonella and other food poisoning agents, and typhus (Newman 1976:669). Many of these diseases are tropical or have been identified in Peruvian mummies; for this reason, much of Newman's list is not applicable to the Northern Plains area. Congenital defects were not examined in this paper. Vogel's overview was more restricted to North America, and he lists (1973:151) rheumatism and arthritis, dysentery and other digestive disorders, intestinal worms, eye disorders, and respiratory infections as being present during the precontact period with rare and localized nutritional deficiencies. Neurological, psychological, and heart diseases were rare, as was cancer. Gregg (1978) suggested that both tuberculosis and syphilis were present on the basis of examinations of skeletal remains in South Dakota and, in particular, the Crow Creek massacre site, present in the Middle Missouri sub-area by the mid-1500s. Gregg's (1978) suggestions are somewhat in conflict with historic data relevant to these last two diseases in the Northern Plains area.

The first useful historical document pertaining to possible aboriginal precontact diseases on the Northern Plains is, paradoxically, fraudulent in a way. Carver (1778) produced a memoir in which he stated that he actually lived with the Sioux after meeting this group at a trade rendezvous at Prairie du Chien. Although this assertion is questionable (cif. Roberts 1937), Carver probably
did receive some accurate medical information while at Prairie du Chien or Michilimackinac, either directly from the Sioux or through another trader. Carver (1778:389-393) noted "pains and weaknesses in the stomach and breast" and attributed these complaints to long fasting, fatigue, and the harsh climate. He found "the disorder to which they are most subject, is the pleurisy, for the removal of which, they apply their grand remedy and preservative against the generality of their complaints, sweating." Dropsy and paralysis were said to be rare, but rheumatism was present. He states that there were the "least traces" of venereal disease among the Sioux and that the disease was unknown to the west but then proceeded to recount the cure of a trader's gonorrhea through the use of prickly ash. As early as Carver's visit, or the visit by his primary source of information in 1768, the Sioux were involved in predatory conflicts with the Pawnee and Arikara (Carver 1778:118). In the early 1800s, Tabeau (1939:151) put venereal diseases as the most common disease among the Arikara, directly contradicting Carver's statement.

The visit of Dr. Edwin James to the Omaha in 1820, as part of Long's expedition, marks the first detailed medical description of the diseases of an ethnic group even peripheral to the Northern Plains (James 1905:15, 20, 43-49). James found the most common Omaha diseases to be syphilis, ulcers and gastrointestinal complaints—which were occasionally fatal—and an influenza-like respiratory disease. Eye diseases were also common. Emphysema, rheumatism, and malaria were present, but rare. In addition to
to the above diseases, James noted a rare occurrence of hare-lip and a few severely congenitally deformed individuals.

Hunter's (1973) narrative of his captivity among the Osage is another important source on Native American diseases of the Northern Plains, despite the fact that the Osage lived on the Central Plains and in close, early contact with French, Spanish, and American traders on the Missouri and Mississippi Rivers. The captivity memoir by Hunter is valuable not only because he presented a list of diseases current before 1816 and a native pharmacology from that period, but also because he gave a view of the Native American perceptions of such diseases and judgments on which diseases were and were not of Euroamerican origin (1973:188). Hunter (1973:210-211) placed pleurisy, cholera morbus, dropsy, rheumatism, diarrhea, malaria, and opthalmic diseases as the most common serious diseases of the Osage. Syphilis (1973:181) was also common and resulted in many fatalities. Swellings, boils, and tumors were rare (1973:210). Hunter made no mention of congenital diseases or the diseases of women which, as an Osage male, he probably would not have been exposed to in Osage society.

Of these diseases, Hunter suggested that syphilis, consumption, and dropsy were of Euroamerican origin, although malaria is also a disease known to have been introduced to the New World (Fenner 1970). Syphilis was said to have been completely unknown until introduced from traders and boatmen on the Mississippi or through Native Americans who had visited Euroamerican settlements (Hunter 1973:181). Consumption, or tuberculosis, was regarded as rare and associated with intemperance (1973:211) and, thus again, with
Euroamerican contacts through the liquor trade, although this medical complaint might well have been a product of the generally poisonous properties of trade rum or whiskey. Hunter's discussion of medical treatments for dropsy provides some insight into the development of medical care for the newly introduced Euroamerican diseases; for dropsy, the Osage "...have more remedies for it than for any other disease, probably, from the uncertainty which attends their operation" (1973:210-211). The materia medica for aboriginal precontact Native American diseases had to be extended by analogy to the new diseases, with a greater degree of experimentation and fewer effective cures.

Comparable data from the Northern Plains from this early period have not been published, even if they should exist in written documents. Tabeau (1939:151) painted a rosy, and probably inaccurate, picture of Arikara health during the early 1800s, stating: "...inflammation of the lungs, ague, jaundice, and other maladies are here absolutely unknown" and that only venereal disease and "accidental illness" were serious problems. In particular, Tabeau (1939:183) found venereal disease, from its description, syphilis, made "more progress in eight days than elsewhere in five or six weeks." The Arikara had no cure for the disease. This fact was also substantiated for both syphilis and gonorrhea by Lewis and Clark (Jackson 1962:505) for all ethnic groups on the Northern Plains. Lewis and Clark (1893), Brackenridge (1904), Bradbury (1904), and Luttig (1964) recorded little information pertinent to precontact diseases on the Northern Plains although all these sources do discuss the effects of smallpox. In addition, Luttig
(1964:106) recorded the death of Sacajawea as a result of "putrid fever," probably puerperal fever, a staphylococcic infection.

To the north, documents left by traders with the Hudson's Bay Company and Northwest Company similarly discussed Native American diseases centered on the effects of smallpox and other epidemics of introduced Euroamerican diseases. Harmon (1973:270-271) provided the best general description in 1807 and found that Native Americans were subject to few diseases. Venereal diseases and tuberculosis were common as were fevers and rheumatism. "Fits," perhaps epilepsy induced by high fevers, trauma or perhaps of psychological origin, were common, "particularly among women" (1973:271). Congential deformities were extremely rare and Harmon attributed this rarity to infanticide at birth. Tanner's (1956) captivity memoir from this early period adds no significant information to Harmon's discussion of disease types. Henry (1897:506, 517, 527) described venereal diseases as both common and uncurable by the Northern Plains ethnic groups. He also described (167:203-204) the appearance of tuberculosis as a new disease in the Red River area in 1800. The diagnosis of this disease is based on the classic symptoms of "...coughing, spitting, and pains in the breast; they linger for a long time, get very lean, and seldom recover" (Henry 1897:203-204). This disease was said by Henry to be the most common and fatal disease, and it reached epidemic proportions between 1800 and 1802 in Native American groups near the Red River.

Maximilian's (1906) narrative of his expedition up the Missouri River in 1833 and 1834 provides the next detailed discussion of
Native American health in the Northern Plains area. At Fort Clark, Maximilian (1906:2: 236-237) observed an epidemic of whooping cough and stated that diarrhea, cholic, and pleurisy were common and sometimes fatal. The pleurisy, or "cattarh," led to the observed symptoms of spitting of blood, which was said to be common, while tuberculosis was rare (1906:359). Rheumatism and eye inflammations were also said to be common as were coughs presumably due to colds (1906:359). Maximilian (1906:360) found that gonorrhea was common among the Hidatsa and Mandan, who stated that the Crow had introduced venereal diseases to the Missouri River ethnic groups. Gout (1906:360) and goiter (1906:257) were present but rare. Only one dwarf was found among the Mandan and Hidatsa as representing the sole congenital defect. Malaria was totally unknown among these groups (1906:237). Maximilian (1906:1: 390-391) also found that periods of starvation were common among the Crees, Assiniboine, and Chippewa due to failures in the seasonal movements of bison.

Perhaps the best single source of information on Native American diseases on the Northern Plains is Edwin Thompson Denig (1930, 1973). Denig worked for the American Fur Company at Fort Union on the Upper Missouri between 1833 and 1856 and had direct or indirect contact with virtually every ethnic group on the Northern Plains and was especially familiar with the Assiniboine and Cree. Denig (1930:425) found diseases uncommon among the Northern Plains ethnic groups except for tuberculosis, rheumatism, and influenza. Fatalities also resulted from staphylococcic and streptococcic infections of wounds and puerperal fever. Hemorrhages
and "pains in the head, heart, and side" were additional causes of death from disease. Pleurisy was fatal also (1930:513). Venereal diseases were common to all groups, but in the Arikara and Crow it reached epidemic proportions (Denig 1973:54, 186). Dysentery, scurvy, and other diseases were present and struck the horticultural groups the hardest because of the lack of hygiene in the sedentary villages (1973:54). Periods of starvation lowered Native American resistance to disease (1930:513), and actual starvation caused mortality (1930:584). Malaria and neurological diseases were unknown (1973:67). Denig made no mention of congenital defects but did state that infanticide was practiced to some degree by all Northern Plains ethnic groups (1973:187).

As an indication of the frequency of disease, Denig (1930:429) gave the example that in a "large camp the drum can be heard at all hours of the day and night, as there is always some one who is sick, or thinks he is." Denig's definition of a large camp was one with from 50 to 100 lodges (1930:441) and using his average lodge population of 4-1/2 persons per lodge (1930:431), there would be an average of one to three people sick for every 338 inhabitants of an Assiniboine camp. This moderate incidence of disease is misleading, however, since it primarily afflicted children. Denig (1930:513) estimated that two of five infants survived to adolescence, a 60% pre-adult mortality rate. The major mortality from disease was caused by epidemic diseases of introduced Euroamerican origin, smallpox, cholera, measles, and influenza (Denig 1930:625).

The notebooks of Lt. James H. Bradley represent the last major source of information on Native American disease before
the reservation period. Bradley used both Native American informants and information received during conversations with Alexander Culbertson of Fort Kipp. Bradley (1923:278) stated that the most prevalent diseases prior to Euroamerican contact were rheumatism, neuralgia, paralysis, epidemic influenza, and occasionally tuberculosis, "liver complaints," and kidney and bladder stones. The Blackfeet said that venereal disease was first introduced to their ethnic group from the Atsina, or Gros Ventres, who were, in turn, infected through contacts with the Arapaho. During the 1870s, the most common diseases were said to be tuberculosis accompanied by scrofula, rheumatism, venereal diseases, and respiratory infections (Bradley 1917:198, 203).

Bradley (1900:256) also found a higher frequency of goiter on the Yellowstone than elsewhere on the Northern Plains. Palliser (1968:245) reported that goiter was very rare among the Blackfeet, rare among the Cree, and general among the Sarci. The only other nutritional deficiency condition reported on the Northern Plains was scurvy. This disease appears to have afflicted primarily Euroamerican travelers (James 1905:I:283, Maximilian 1906:III:82) and Native Americans in starvation conditions (Matthews 1877:25). Unlike goiter, scurvy was treatable by the consumption of foods and infusions containing high amounts of vitamin C.

The extinction of the bison and the changed residential patterns accompanying the establishment of reservations for the Native American ethnic groups on the Northern Plains resulted in a dramatically altered environment for the development of the spread of disease. At Port Berthold, Matthews saw the beginning
of this pattern as early as the late 1860s and early 1870s. He ascribed (1877:25) scurvy, a tendency to abscesses, a suppurative termination of diseases, and slow healing rates of wounds directly to changed dietary patterns brought about by the consumption of agency food and lowered number of game animals, particularly bison. In addition to the nutritional changes of agency life, the tendency to clustering residences around central food dispersal areas brought a dramatic rise in cases of tuberculosis, a rise brought about in part by starvation itself and a consequently lowered resistance to disease. Among the Crow, Plenty-Coups (Linderman 1962) refused to even talk of the period, and Pretty-Shield (Linderman 1972:249-250) could not believe that "the same sickness that makes people cough to death" ended in an osteological involvement and consequent death of her daughter. Crowfoot (Demsey 1972:63, 181) of the Blood band of Blackfeet lost nearly all of his children to tuberculosis.

As a direct result of starvation, an estimated one-quarter of the Piegan Blackfeet died during the winter of 1883-84 (Grinnell 1962:289). Most ethnic groups on the Northern Plains suffered less than the Piegan during the reservation period before 1900, but Wissler's (1936) discussion of the population dynamics of these groups demonstrates that all groups did undergo demographic change. Because these changes were not a direct product of pre-reservation life, they will not be discussed here.

Another source of precontact aboriginal disease on the Northern Plains was contact with diseased game. Martin (1976) made a very good argument for a tularemia epidemic which spread from beaver to humans among the Chippewa in 1803-04. Tanner (1956:104-
114) described a typhoid-like epidemic which struck the band in which he was living in conjunction with a similar disease observed in impromptu autopsies carried out on beaver he had trapped, and Martin (1976:58-61) has successfully analyzed the symptoms. Martin's attempt to equate an epizootic with the smallpox epidemic of 1781-82 is founded on the musings of David Thompson (1915:110, 327) written many years after this epidemic, and Martin's (1976:53-56) arguments fail due to the basic fact that smallpox is not communicable to dogs or any other animals. The probable presence of sylvanic plague in precontact rodent populations has also been noted (Martin 1976:49). The infection of humans with helminths of animal origin through contact with feces or undercooked food would be another source of precontact human disease on the Northern Plains.

Despite the potential for human contamination from diseased game, there are no data on the Northern Plains, except for the possible typhoidal tularemia and worms, that would suggest such diseases were very serious health factors. Several Sioux winter counts (Mallery 1972:318, 589) indicate that a war party of six men ate the rotting carcass of a bison and apparently died of salmonella poisoning during 1826-27. This is one of the few published examples of such poisoning in the entire literature on the Northern Plains, despite the well-known practice of snaring winter-killed bison floating down the Missouri River by the Mandan and Hidatsa.

Rabies seems to have been the most dangerous disease transmitted from animals to humans on the Northern Plains. The attack
on the participants of the 1833 Green River rendezvous has been discussed by numerous authors, among them Larpenteur (1898:36-41), but the incidence of rabies seems to have been rare. However, an epidemic of rabies in coyotes and probably other animals was recorded by the Sioux in 1772 (Howard 1976:37) and 1838-39 (Cohen 1942b:31). The presence of rabies on the Upper Missouri was recorded by Lewis and Clark (1893:7: 323), but from the very few early references to this disease, as opposed to the high numbers of dogs used by Native American ethnic groups on the Northern Plains, it was uncommon.

This short review of aboriginal precontact diseases among Native American ethnic groups on the Northern Plains reveals several important factors pertinent to the analysis of the effects of introduced Euroamerican diseases. First, with the exceptions of tuberculosis and venereal diseases, the precontact diseases on the Northern Plains appear to have been primarily those of the gastrointestinal and respiratory tracts: intestinal parasites and worms, pleurisy, colds, bronchitis, and pneumonia. Rheumatism and associated viruses were also present. None of these diseases approached the fatal epidemic nature of diseases introduced from Euroamerican sources. Case mortality from these diseases was most probably clustered among infants and the aged, with adult mortality occurring in conjunction with exposure, starvation, or a combination of diseases. With this type of disease mortality, the Native American groups did not need, nor did they have, a high birth rate; nursing, which lasted from two to four years, assured the infant adequate nutrition and resulted in family sizes between four to six

Adult mortality during the precontact and early contact periods seems to have been differentially clustered among males. Part of the higher adult male mortality was certainly a product of intensified interethnic warfare concurrent with the displacement of ethnic groups peripheral to the Plains as Euroamerican technology as spread from the northeast and southwest (Secoy 1953) among other factors, as well as accidental death and injury resulting from male hunting activities. Additional adult male mortality resulted during these male-oriented activities from disease. Hunter (1973:93-94) voiced the often repeated opinion of the Native American superiority of gynecological and obstetrical care of women and the low adult female mortality from purely female-oriented diseases and made the generalization that:

The death of an Indian woman, aside from casualty, is a rare occurrence, except from the ordinary wane of the functions of life. The same cannot be said of the men: their frequent exposure to all varieties of temperature and weather; fatigues from long marches; and long abstinence from food, followed by an inordinate indulgence of the appetite; give rise to many diseases, from which death often times results (1973:94).

A second factor is that Native American concepts of disease and health care were designed particularly to combat the low order aboriginal diseases. Diseases were recognized among the Northern Plains ethnic groups as a spirit or magical object penetrating the body of the ill individual. Thus, spiritual practitioners were called upon to drive away or extract the spirit or thing causing the disease in concert with secondary medical practitioners who
used herbs and other physical treatments to achieve the same re-

sult. The distinction between the two types of medical care has

been chiefly discussed among the Chippewa by Densmore (1928:322-

325). It is noteworthy that while "medicine-men" were males, and

less frequently females, "herbalists" appear to have been virtually

all female. The near total lack of data on female herbal doctors

appears to be a direct result of the differential concern given

female activities by the predominantly male authors of historical

and anthropological literature on the Northern Plains.

This dual treatment of disease in Native American health care

involved both physical treatments of disease with herbs, massage,
lancing and minor surgery, and such treatments as the sweat lodge

and the psychotherapy of shamanistic magic. Denig (1961:116),
among other early observers of this shamanistic curing, reviled
the "drum and rattle" in treating disease, but also recounted in-
stances of miraculous cures. Such a dual treatment of physical
disease is today known as holistic medicine, which seeks to cure
or treat the organic agent of disease and also to treat psycholog-
ic resistance to this process of organic treatment and increase
the psychophysical resistance to the disease itself. The involve-
ment of medical care specific to the given disease in modern hol-
istic medicine appears to parallel Native American medical care
and points out to the failure of Northern Plains ethnic groups to


treat introduced Euroamerican diseases. Native American health
care systems did not have specific treatment to such diseases as
smallpox and measles; consequently, nearly the entire amount of
medical care was concerned with shamanistic "faith healing."
This psychological treatment appears to have been successful for some diseases; Cabeza de Vaca (1973) gained a reputation as a healer as he was attempting to return to Mexico in the early 1500s, but, as Denig indicated for smallpox, this treatment was useless for most diseases. The failure of Native American medicine on the Northern Plains also became a failure of the religious system.

Native American disease theory also failed for the Euroamerican diseases because it contained no concept of disease communication or quarantine aside from the menstrual hut belief system (Lowie 1963:90). The words of a Blackfoot survivor of the 1780-82 smallpox epidemic, "we had no belief that one Man could give it to another, any more than a wounded Man could give his wound to another" (Thompson 1915:337) could be extended to every ethnic group on the Northern Plains during the early contact period. When an individual was ill, whole groups of kin would act corporately to purchase a cure or give a sacrifice to insure his cure (Harmon 1973:303). This process would involve a grouping of several individuals in direct or indirect contact with the person who was ill. Given the types of diseases present during the pre-contact period, such visiting might communicate a minor disease to these visitors. However, with the appearance of epidemic diseases during the post-contact period, this cultural pattern spread epidemics like a prairie fire, especially as many of these diseases are communicable before their overt symptoms appear (Benenson 1970).
Traditional beliefs of infection were based on ritual or magical contamination following a breach of taboo, invasion of a malevolent spirit or thing, or a direct result of sorcery. The precontact aboriginal diseases struck few individuals directly and thus were compatible with the infection theory. The universal infection of whole camps with epidemics of Euroamerican origin strained this theory, especially since the epidemics ended with large numbers of case fatalities. Corporate activities which worked to insure a distribution of food and other goods to families with ill members were disrupted because everyone was ill. The change of the pattern of individual to universal illness is one of the major factors important to the analysis of introduced Euroamerican diseases.

This point is relevant to the discussion of tuberculosis and venereal diseases. Among the historic sources discussed above, these two diseases appear to represent a gradient which climbs from rare or absent during the early contact period to near universal at the beginning of the reservation period. These diseases also share the epidemiological characteristic of being communicable over a long period of time; consequently, they are endemic diseases. For this reason alone, the increase of these diseases through time indicates that tuberculosis and venereal diseases among Native American ethnic groups of the Northern Plains are a result of increased direct and indirect contact with Euroamericans.

In his discussion of venereal disease, Vogel (1973:143-144) presented several early sources which pointed to these diseases as having been introduced through Euroamerican contact. Two early
sources relevant to the Northern Plains, Carver and Hunter, made
the same generalization: Carver (1778:392-393) implied that in-
fection spread from centers to the northeast and Hunter (1973:181)
specifically stated that the source of the venereal diseases was
from boatmen at trade centers. Interethnic contacts with Native
American groups trading to the southwest was another portal of
infection for venereal diseases on the Northern Plains (Bradley
1923:278, Maximilian 1904:2:360). The spread of venereal dis-
ease on the Northern Plains was facilitated by ceremonial sexual
intercourse (Kehoe 1970) as well as societal sexual mores. Mem-
ers of the Lewis and Clark Expedition (1893:I:255) contracted
venereal disease from the Mandan and Hidatsa while at Fort Mandan,
and Thompson (1916:234-235) complained that employees of Northwest
Company posts sold their goods cheaply to secure sexual access to
females among these ethnic groups. Brackenridge (1904:129-130)
described the loaning of wives, daughters, and sisters for sexual
purposes as part of Arikara hospitality and noted that refusal of
such favors enhanced the status of the individual. The "common
boatmen" were evidently held in low esteem as an Arikara chief
asked: "...Whether you people have any women among you..."
because "your people are so fond of our women, one might suppose
they have never seen any before" (Brackenridge 1904:130). Henry
(1897:526, 734-735) found the offering of wives and daughters to
be prevalent among the Blackfeet and Atsina as a trading custom
and also with the expressed desire for a child fathered by an
Euroamerican. Sexual contacts between Euroamericans and Native
Americans spread venereal disease to the Native Americans and visa
versa, either by direct contacts on the Northern Plains or from indirect contacts by other Native American groups trading outside this area.

Tuberculosis on the Northern Plains appears to follow the same pattern, although at least one strain of this disease was present in the New World before Euroamerican contact. Gregg (1978) has presented conclusive evidence of a hunch-back tuberculosis condition among the skeletal population at the Arikara Crow Creek Creek site, dating at before 1600 AD. Despite the presence of this disease, it was rarely encountered by early Euroamerican travelers or, if encountered, it was described in such a vague manner as to be diagnosed as any one of several respiratory complaints. Maximilian (1904:2:359) succinctly stated that "spitting of blood is said to be frequent, but not pulmonary consumption." Morgan (1959:102) was informed by Martin, a Fort Union trapper, that tuberculosis took three years to kill a Native American on the Northern Plains and found the incidence of the disease to increase as contacts with Euroamericans did. Because of this association, Martin felt that it was a disease introduced by Euroamericans. This same association was also voiced by Hunter (1973:211). Henry's (1897:167, 203-204) description of tuberculosis as a newly widespread disease on the Red River between 1800 and 1802 tends to support an Euroamerican influence in the spread of this disease, either by influencing an increased contact between infected and uninfected individuals or by the importation of a new and virulent strain of the bacilli, or both. The disease agent which caused often fatal hemorrhages
of the respiratory tract is unknown; however, even nosebleeds could be fatal (Audobon 1960:2:108,121).

An insight into the identity of this agent may be found in the papers of the Palliser Expedition (1968:240) whose members found a disease "which resembled pleuropneumonia" among bison in 1856. They were assured by guides that this disease was common during summer and there was no human danger in eating the meat. This assurance of safety is questionable as it probably referred to immediate danger. Tuberculosis remains a significant disease among remnant bison herds (Meagher 1978:216) and a strain of this bovine infection possibly was conferred to pre- and post-contact Native Americans through consumption of under-cooked meat or other contacts with diseased animals. The relationship between North American and Euroamerican bison and bovine tuberculosis has not been examined.

A last factor in precontact Native American health and disease on the Northern Plains is the very low number of congenital deformities among the ethnic groups of this region. Maximilian (1904:2:257) identified a few cases of dwarfism and several cases of congenital deafness. Henry (1897:736) described only congenital deafness. Harmon (1973:271), Kelly (1871:181), and Schoolcraft (1953:154) directly associated the rarity of congenital deformities to infanticide. Although Denig (1961:186) found infanticide to be most prevalent among the Crow, he inferred it to be present among all ethnic groups on the Northern Plains. It may be significant that no source in the Northern Plains region found the relatively high frequency of congenital deformities noted by James (1905:2:20).
among the Omaha, nor is there any indication of a spiritual power associated with congenital deformities such as was recorded by Murie (1914) in the Pawnee poor hero cycle. The only archaeological evidence of a severely deformed individual comes from the Larson Site (32BL9), a protohistoric Mandan site which was abandoned prior to 1750 AD. Here, a cranium was discovered showing developed acromegaly and an absence of mandibular alveolar tissue. Unlike other burials at the site, this individual was buried with substantial amounts of red ochre, indicating perhaps that the deformity was regarded as being imbued with spiritual power. In the case of this individual, as that of the dwarf noted by Maximilian, infanticide at birth would not have eliminated the development of the deformity. Most defects, however, were eliminated by infanticide or by natural deaths.

Overall, the health of Native Americans on the Northern Plains during the precontact period can be inferred to have been generally good. Should an individual have survived infancy and early childhood, his resistance to extant diseases would have insured him a long life if he did not die from accident, warfare, or starvation. The dichotomy between precontact and post-contact disease patterns is illustrated in Denig's (1973:125) discussion of burial mounds.

Ordinarily Indians are not buried in heaps because when they are not visited by fatal epidemics they rove in quest of game, do not remain long in one place, and are healthy people and seldom more than one or two graves are seen near any of their transient encampments. Even where large villages have wintered the interments do not often exceed ten to a dozen bodies. But when Pestilence such as smallpox prevails, and attacks the whole nation at the same time, they are disabled from

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
travel and obliged to remain stationary until the disease finishes. Thus hundreds are consigned to the same general burial ground.

Although Denig was talking about Woodland Period mounds in terms of his personal experience with the Assiniboine and other ethnic groups of the mid 19th century, his remarks do provide a vital insight. A large camp was defined by Denig (1930:441) as having an average of 75 lodges with an average lodge population of eight individuals. During the most severe weather in a given year, only about 12 of 600 individuals died, or two percent of the population, not counting replacement births. Carrying this maximum death rate throughout the year, a maximum of eight percent of a given camp population would lie, or a total of 48 individuals. To compensate for this death rate were the reproductive efforts of married women; and using Fidler's 1815 statistics (Ray 1974:112), together with Denig's (1930:513) 1840-50 average birthrate of 4.5 children to each woman during the 20-year fertility period, there would be an average of 33 children born during this same period, given no multiple births. As Fidler estimated a secondary sex ratio near 100, this anomalously low replacement birthrate is probably a result of poor statistics. With the tertiary sex ratio of 50 more commonly estimated for Northern Plains ethnic groups, there would be a replacement birthrate equal to and slightly above the death rate. The high death rate reported by Denig was also during a period after introduced Euroamerican diseases had begun to make serious impacts on Native American health. The effects of epidemic disease upon this declining birthrate were to create a descending spiral of depopulation on the Northern Plains.
"We had no belief that one Man could give it to another, any more than a wounded Man could give his wound to another," stated a Blackfoot survivor of smallpox (Thompson 1915:337). It was as if the Bad Spirit himself had visited the band. This reaction to first contact with a malignant epidemic infection can be duplicated in the literature on virtually any people in the Americas or the Pacific Islands. Because these peoples were isolated from the interlocking population network of the Old World, they had no prior contact with such diseases as chickenpox, measles, influenza, typhoid, smallpox, and cholera. As, by definition, malignant epidemic infections provide partial to total immunity to subsequent infection, these isolated populations had neither resistance nor immunity to the newly introduced diseases. Medical belief systems oriented to indigenous low-order infections did not cure the new high-order infections; and in many cases, they actually contributed to a higher mortality rate. The end result was many deaths and, consequently, a significant impact upon native cultures. The peoples living in the Northern Plains area were no exception. In this chapter, an historic chronology of epidemics on the Northern Plains is presented, with a listing of groups involved, probable locus of infection, and estimates of mortality where possible.
Methodology

The study methodology for the data presented here differs from similar studies by Stearn and Stearn (1945) for North America as a whole and by Ewers (1973) for Texas. Stearn and Stearn appear to have culled historic documents for a diagnosis of smallpox and to have accepted this diagnosis without examining other sources to determine whether the disease may have been measles, or even cholera. Ewers utilized secondary sources, and again no cross-examination of data was apparently made. To avoid such problems in this study, research controls were established. The first control was basic and required that more than one primary source be used to confirm an epidemic. Due to the limited number of source materials dealing with the early historic period on the Northern Plains, this control was extended so that two sources were not required for a specific ethnic group but for groups that were known to have come into regular contact during the period. The best example of this control in operation is in the discussion of the 1750 smallpox epidemic given below.

The second control was the diagnosis of the epidemic disease types through a comparison of documented symptoms with those described in a Public Health Service manual on infectious diseases (Benenson 1970). Whenever no symptoms or only a few were mentioned in the historical source, the diagnosis presented was tentatively accepted. Some control on the diagnoses was also provided by the immunological properties of the diseases. Smallpox, measles, chickenpox, and erysipelas all produce the symptom of eruptive pustules, or "spots," but each disease produces specific immunity.
Hence, by tracing back through time from a known immunity to a disease, the disease type of an earlier epidemic can be suggested. This diagnostic control was particularly useful in the evaluation of winter-counts in which pictoral mnemonic clues by the Native American author portrayed principal yearly events of a band. The depiction of a face with spots on it can easily be any of the diseases named above; thus, the diagnosis may rest upon the informant who is interpreting the winter-count. However, when the spotted figure is shown in a position indicating severe cramps, a symptom of smallpox, diagnosis can be more readily ascertained. The association of measles with respiratory disease is a similar diagnostic clue. Differentiation between measles and chickenpox cannot be made from winter-counts unless respiratory disease is also indicated as being present.

The question of whether an epidemic was smallpox or measles is very real. Smallpox is a known killer, and measles can produce nearly the same rate of mortalities among a non-resistant population. For example, among the Kalopalo in central Brazil, a 1954 measles epidemic was fatal to 26.6% of the population (da Silva 1972:267). Several scholars have postulated that the high frequency of "smallpox" epidemics in historical literature may represent a mis-diagnosis of measles. This is probably correct, but on the Northern Plains smallpox epidemics were frequent. A portion of this frequency arises from the fact that, as late as the late Eighteenth Century, smallpox was a childhood disease which resulted in from 10% to 20% of child deaths in Europe (Cockburn 1971:52). A second factor is the great difference in the period...
of communicability between the two diseases. Measles is communi-
cable for little more than a week through contact with respiratory
discharges or articles contaminated with the above, while smallpox
is communicable for months through the same process, and even by
touching an infected individual (Benenson 1970). The long communi-
cability of smallpox increased the chances of its being transferred
to non-immune individuals and resulted in long-term, widespread
epidemics that were readily carried across the Atlantic to the
New World.

Respiratory diseases are more difficult to diagnose from his-
torical sources than are smallpox, measles, or cholera due to the
wide range of infections which share common symptoms. Discrete
respiratory epidemics can be differentiated from the pulmonary
complications of other diseases according to whether these diseases
appear singly in an epidemic or diffuse to different populations
from a pandemic. The distinctions between whooping cough, some
streptococcal infections, and influenza are not clear in historical
discussions. Whooping cough may be diagnosed with reasonable
accuracy by the paroxysmal cough and the degree to which coughing
is stressed. The symptoms of streptococcal infections may vary
widely, depending on the portal of entry and the tissue of local-
ization of the disease. Wounds might become infected with strep-
tococci, or the infection may be epidemic in nature, such as
occurs with streptococcal sore throat, scarlet fever, erysipelas,
or puerperal fever. With the exception of erysipelas, strepto-
occal infections do not confer immunity upon infection, and they
also have exceptionally long periods of communicability. Such
Smallpox

Whooping Cough

Measles

Puertual Fever

Figure 1
Sioux Winter-Count Depictions of Disease (from Mallery 1972)
infections can lead to degenerative complications and a lingering death. An epidemic puerperal fever appeared among the Sioux in 1799 (Curtis 1905:169; Mallery 1972:589-590), and by 1850 infections due to streptococci were endemic on the Northern Plains (Denig 1930:425).

Influenza also produces symptoms of this same general type. Influenza complicated by pneumonia may last a month and result in serious complications but does provide some resistance with infection. Ray (1976) has detailed the diffusion of several influenza epidemics into central Canada during the 1830s, but these appear not to have spread beyond the fur trade river posts to the Native Americans on the Northern Plains. During the 1806-14 period, two discrete epidemics characterized by respiratory symptoms occurred in the Northern Plains area, but even the best accounts (Henry and Thompson 1897:167, 202-204, 225, 228, 232-233) do not provide the accuracy needed to distinguish between the major respiratory diseases.

The "chronology of epidemics" section, below, presents a discussion of each epidemic. This discussion is brief, as it would be beyond the scope of this work to provide a full account of each epidemic.

Chronology of Epidemics

1733-1735: Smallpox

This smallpox epidemic is well documented in Sioux winter-counts (Curtis 1905:164; Howard 1976:33; Mallery 1972:300). Although the High Hawk account edited by Curtis calls the epidemic
measles, all the counts indicate cutaneous eruptions and severe stomach cramps, characteristic symptoms of smallpox. This diagnosis is confirmed by French documents (Thwaites 1906:2:172-175) which show the presence of smallpox in the Old Northwest and detail its spread east to Montreal by Iroquois warriors. The locus of infection may have been from the Mississippi River, but this is speculation. It is certain that this is not the same epidemic noted among the Woods Cree by La Verendrye (1927:256-259, 282) in 1737. That epidemic occurred two years later, and the infection was restricted to those Cree bands trading at York Factory. Many Sioux are reported to have died, but no mortality estimates can be assigned.

Inferential data suggest that this epidemic also struck the Arikara. In 1795, Truteau (1952:299) was told that smallpox had struck three times with great mortality. Deetz (1965:101) concluded, from an analysis of changes in Arikara ceramic styles, that the time period from 1720 to 1750 was one of maximum cultural stress. Additional inferential information is suggested by comparison of the 1717 Vermale Map with the 1795 Soulard Map (Wheat 1957:Fig. 98, Fig. 235a). The earlier map shows "40 villages Panis ou Ricaras" near the mouth of the Cheyenne River, while the later map shows only "7 villages Ris" at that location. This evidence, along with the fact that Sioux-Arikara interaction had occurred at this early date, make it almost certain that the Arikara did experience this epidemic. No data exist to indicate its extent.
1733-1735

Figure 2: Smallpox

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
1750: Smallpox and Measles

The only historical document directly identifying this epidemic is a report from the French posts in southwestern Wisconsin written by La Jonquiere (Thwaites 1908:3:87). The report states that the Osage had contracted smallpox and measles on the Missouri River, where that group had been warring against the Pawnee and Wicheta. The alliance of the Comanche with these latter groups was noted, and that group may have carried the disease or diseases from the Texas area where at least smallpox was present the same year (Ewers 1973:108). There is little evidence to suggest spread of the epidemic into the Upper Missouri. The inferential data for Arikara infection, cited for the previous smallpox epidemic, may be applied again here, but no Sioux winter-count mentions the epidemic. Houghton's (1832:175) Chippewa data indicate that smallpox epidemic occurred the same year among that ethnic group but was brought from Montreal by a band of young men and was characterized by no widespread contagion. Due to the lack of descriptive data for analysis, La Jonquiere's diagnosis of the epidemic will be accepted. No mortality estimates can be assigned this epidemic.

1780-1782: Smallpox

This epidemic of smallpox first appeared in the Spanish settlements of the Southwest in 1779 (Ewers 1973:100). In 1780, the disease spread to the Comanche either through direct raids or through contacts with infected bands. Through interethnic contact, the epidemic spread to the ethnic groups on the western margins of
1750

Figure 3: Smallpox

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
the Northern Plains: the Shoshoni, Nez Perce, Salish, Kutenai, and Crow. Nez Perce informants told Spalding (Spalding and Smith 1958:137) that the disease struck a polyethnic Nez Perce and Salish bison hunting camp during the winter, probably the winter of 1780-81. The Salish have the same tradition (Mengarini 1977:193-194). During that summer, the epidemic continued in the Shoshoni camps. The Blackfeet caught the disease by plundering a stricken Shoshoni camp (Thompson 1915:322-323). The epidemic was still raging among the Blackfeet when Hudson's Bay Company employees first heard of the epidemic on October 22, 1781 (Tomison and Walker 1952:262-263). Smallpox victims did not reach Cumber­
land House until December 11 (1952:223) but, once there, spread smallpox to other ethnic groups trading at the post, thence radiating the disease north and east during the winter. Based upon Hudson's Bay Company Reports, smallpox among the Blackfeet ended by March 1782 (243, 279).

The mortality caused by this epidemic was high and general. At least one major camp of Nez Perce and Salishans was annihilated. Estimated of Salishan mortality range upward to a population equi­
valent to six major bands (Teit 1930:315-316). Blackfeet mortality was high among the camps that contracted the disease, but several camps escaped infection (Tomison and Walker 1952:288). The Crow, as a unit, experienced a high mortality rate, but fatalities were largely restricted to one great encampment of 400 lodges, which constituted approximately half the Crow population, at the conflu­
ence of the Clark's Fork with the Yellowstone River. Nearly every­
one died, and the location became known as the "Place of Skulls."
The other half of the Crow were near the Powder River and escaped infection (Bradley 1899:166, 179; 1917:197). The Shoshoni were all but exterminated by this epidemic and its aftermath. Fatalities were extremely high; following the epidemic, the Blackfeet, blaming the Shoshoni for the onset of smallpox, armed themselves with guns and crushed the survivors (Thompson 1915:336) along with other Salish groups to the west (Teit 1930:315-316).

The epidemic also spread from the Comanche through the Pawnee to the ethnic groups of the Upper Missouri. The Cheyenne (Berthong 1963:10) suffered a high mortality. The Arikara were reduced from about ten villages to only three, consolidating into two villages by the 1790s in the face of Sioux attacks (Bowers 1950:24-26). The Hidatsa did not merge into a single political group until after the 1780-82 smallpox epidemic, but data suggest that the Hidatsa-proper and Awatixa groups had a lower mortality rate than did the Mandan, while the Awigaxa suffered the same percentage of fatalities as the Mandan (Bowers 1965:362-366). The incredibly high mortality rate of the village horticulturalists can be attributed directly to their sedentary settlement pattern. While scattered camps of nomads could avoid contagion, a large village, once contaminated, could not.

Smallpox spread east and north from these groups. Sioux winter-counts record that the smallpox epidemic lasted for two years. One account by Batiste Good (Mallery 1972:308) lists the years 1779-81 for the epidemic but is contradicted by other Sioux counts of the same period which record the years 1780-82. A possible source of infection for at least some Sioux groups was
the Sioux attack which destroyed the Mandan villages on the west side of the Missouri during the midst of the epidemic. Sioux winter-counts indicate that mortality was high.

The spread of the smallpox epidemic to the Assiniboine, Cree, and Ojibwa also occurred through warfare and raiding. Warren (1885:47-48) gave the Ojibwa account of the spread of the smallpox epidemic. A polyethnic war party of the three groups traveled westward from the Cree village at the confluence of the Dead and the Red Rivers to attack the "Gros Ventres." These "Gros Ventres" were apparently the Gros Ventres des Prairies, or Atsina, judging from the western direction of the war party's travel. Members of the enemy camp were attacked, and the party met a surprisingly feeble resistance. Scalping those they killed, the war party entered a village filled with the smallpox dead. As the party returned to their own village, the symptoms of the disease appeared among them. Smallpox destroyed this village; and the tributary to the Red River was thereafter known as the Dead River. From this locus, smallpox continued to spread north and east. Warren estimated the Ojibwa mortality to be between 1,500 and 2,000 individuals. Both McDonell (1933:112) and Denig (1961:114-115) estimated a fifty percent Cree mortality rate. Denig (1930:399) placed the Assiniboine mortality rate at one-third of their total population.

This discussion of the 1780-82 smallpox epidemic does not agree in one point with that given by Thompson (1915:302) and most recently by Ray (1974:107). Thompson proposed that the epidemic entered the Northern Plains from the northeast, and Ray accepted this source. This is incorrect, as the Assiniboine, Cree,
and Ojibwa accounts all indicate a western locus, as does the Chippewa account given by Houghton (1832:177). Additional information is given by Henry (1897:293-294) about a Mr. Bruce, who had been at Fort de Tremble when it was attacked by the Cree in 1781. Bruce related that during the evacuation of the post, two young Native American men were permitted to ride in the canoes to Fort Dauphin. It was later discovered that both had smallpox, and the disease spread onward.

1798-1799: Streptococcal Infection, Puerperal Fever

Sioux winter-counts (Curtis 1905:169; Mallery 1972:312, 589-590) characterize this year as the year many women died in childbirth, an extremely good description of puerperal, or child-bed, fever. This unique occurrence marks the appearance of widespread streptococcal infections on the Northern Plains and possibly the beginning of the endemic nature of this pathogen in the area, increasing post-partum mortality and degenerative wound infection. The locus of infection may have been from Canada, but the most likely source was a trading party which entered the area from St. Louis. Both Sioux and Euroamerican accounts (Nasatir 1952) record the beginning of the St. Louis trade a few years previously. No other Native American source records this epidemic, but as the Sioux and Arikara had formed a trade partnership by this date, the Arikara could have also been infected.
Figure 4: Smallpox

1780-1782

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
1798-1799

Figure 5: Pupueral Fever

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
1800-1803: Pandemic--Smallpox, Measles, and Respiratory Infections

Between the years 1801 and 1803, a pandemic appears to have occurred in the Northern Plains area. Smallpox entered the area from the south and a respiratory disease from the northeast. A third disease, perhaps measles, was present in the east.

Smallpox struck St. Louis for the first time in 1799 (Houck 1980:62). During the winter of 1799-1800, this disease infected the Omaha, Iowa, and other groups living on the lower Missouri River (Delassus 1952:631). Shortly afterward, the Pawnee contracted the disease (Dougherty 1931b). By summer, smallpox had spread into the Central Plains to the Arapaho who infected the Atsina by 1801. Atsina mortality was "upwards of 100 all young people" (Fidler 1976:294) but did not spread to other ethnic groups to the north and east (1976:317). The Crow also contracted smallpox during this epidemic from their allies, the Arapaho (1976:321; Larocque 1911:59), but the only estimates of mortality for this ethnic group also include those for the 1780-82 smallpox epidemic. Teit (1930:315-316) reported that the Crow infected the Salish and smallpox spread westward into the Columbia as a severe epidemic. Spalding (Spalding and Smith 1958:137) was told of this epidemic by Nez Perce, and he saw the smallpox scars. Because he was told this epidemic was much less severe, Spalding hypothesized that the epidemic was alastrim rather than smallpox. Alastrim, or Variola minor, is a much less severe form of smallpox. The difference between the effects of this epidemic on the Salish and the Nez Perce probably does not lie in a difference in diseases but rather in a perception of the disease. The Nez Perce
possibly had a greater number of immune individuals in the population than did the Salish, many of whom appear to have relocated to the eastern margin of the Salishan-speaking territory to more easily engage in bison hunting.

Sioux winter-counts record that some Sioux bands contracted smallpox during this epidemic, while others did not. The infection was spread to the Sioux after that group attacked the Omaha during the epidemic (Tabeau 1939:100). However, the epidemic among the Sioux appears to have been more complex. Sibley (1832:721-722), a medical doctor, found measles to be present with smallpox in a pandemic centered in the Red River of Texas about 1803. An indication of measles is also present in the Sioux winter-counts. In the No Two Horns, Blue Thunder, and Blue Thunder variant winter-counts, a high mortality for the epidemic of 1800-01 is shown; but during the 1818-20 measles epidemic, only children showed a significant number of fatalities (Howard 1960:326-363, 352). This would indicate that a number of Sioux adults were immune to measles. On the other hand, two winter-counts edited by Cohen (1939, 1942a) do not mention the 1800-03 epidemic but indicate high general mortality during the 1818-19 measles epidemic. As smallpox does not confer immunity to measles, it would have been that measles was present during the 1800-03 pandemic.

On the northern margins of the Northern Plains, Henry (Henry and Thompson 1897) provided the only available record of a highly infectious respiratory disease which reached epidemic proportions. Henry (1897:202-204) described its symptoms as: "...coughing, spitting, and pains in the breast; they linger a long time, get
very lean, and seldom recover. This is the most common and fatal disease among them." Henry first mentioned this disease in 1801 and by 1803 found camps of Assiniboine and Cree so ill that they were unable to hunt (1897:167, 288). These symptoms appear to be diagnostic of tuberculosis, but Henry himself caught the infection and recovered in about three weeks (1897:288). A part of this respiratory disease was probably tuberculosis, but influenza or some kind of streptococcal infections constituted some of this respiratory disease array during the 1800-03 period. Martin (1976), using the captivity memoir of John Tanner, has diagnosed this epidemic as tularemia contracted from beaver by the Chippewa. This diagnosis appears to be unlikely because the disease was widespread across the northern rim of the Northern Plains. Many fatalities were indicated, but mortality never reached catastrophic levels.

1806-1814: Whooping Cough and Streptococcal Infections

This period of epidemic respiratory infections appears to be a continuation of similar infections noted by Henry in Canada in 1801. The disease or diseases were endemic in Canada by 1806 when Henry visited the Mandan. At the Mandan villages, he found the same disease, which he (Henry and Thompson 1897:343) described as "a kind of whooping-cough" and indicated that infirm elderly individuals and children died. Mackenzie (1889:371-372) was with Henry and reported 130 deaths among the Mandan and Hidatsa during the month of June. Sioux winter-counts document the epidemic among some Sioux bands in 1807 (Cohen 1942a:20) and a second
1800-1803

Figure 6: Pandemic

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
similar epidemic among the Sioux during 1812-14 (Mallery 1972:276, 588). In 1810, Henry (1897:656, 662) found the disease among the Piegan Blackfeet.

The symptoms described in accounts of the disease suggest that streptococcal infections were also present with the whooping cough. Neither disease confers immunity after infection, and both have long periods of communicability. Henry (1897:225, 662) contracted the infection twice, the first time with severe pulmonary congestion and later, in 1810, with merely a bad cold and a sore throat. That this sore throat may have been strep-throat is suggested by the death of Sacajawea from puerperal fever in 1812 (Robinson 1924:69-71), a medical condition caused by the same pathogen. Both diseases were spread by Euroamerican traders from trade centers in Canada but resulted in relatively few fatalities.

1819-1820: Measles and Whooping Cough

Franklin (1824:1:92-93) was a witness to this epidemic at Cumberland House during the winter of 1819-20 and emphatically stated that both epidemics occurred together. This association is also recorded by Provencher (Nute 1942:242), at the newly established Earl of Selkirk's Red River Settlement. Provencher noted that only a few children died, but this was not the case for the Native American ethnic groups which had never been exposed to measles. Ray (1974:108), working with unpublished Hudson's Bay Company records, reported that over 2,400 Assiniboine and perhaps 800 Cree died in the Brandon area. Dempsy (1965:7)
1806-1814

Figure 7: Pandemic

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
has estimated from these records that one-third of the Blackfeet and Atsina groups died from the epidemic. Many of these deaths may have been from whopping cough as a Blood Blackfeet winter-count records the disease as the "Coughing epidemic" (Dempsy 1965: 7). Sioux accounts have been discussed above; some indicate high undifferentiated mortality and others, those describing high mortality during the 1800-03 epidemic, indicate high numbers of deaths among children only.

1831-1832: Smallpox

This smallpox epidemic was restricted to the ethnic groups living south and west of the Pawnee villages on the Platte River. Only one, and possibly two, of the Northern Plains ethnic groups were infected. Dougherty (1931b) first discovered the epidemic among the Pawnee during an inspection tour of his agency in the fall of 1831 and found that over one-half of the population had died. The immediate vaccination efforts Dougherty (1831a) organized probably prevented the spread of this epidemic into the Northern Plains.

The only Northern Plains groups that were infected by this smallpox epidemic were the Atsina and possibly the Cheyenne. In both cases, the disease was transmitted through contacts with the Arapaho. Bradley (1900:226) stated that the Atsina and Arapaho were living together when the smallpox epidemic struck and that both groups suffered high mortalities. For this reason, only about 200 Atsina children died during the 1837-38 smallpox epidemic; the children were the only individuals not immune through
1819-1820

Figure 8: Measles

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
prior infection. The possibility of Cheyenne infection results from the close interethnic ties of this group with the Arapaho. Lavender (1954:138-139, 387-388) provided contradictory evidence in his discussion of Bent's Fort however. When the crew constructing the fort developed smallpox through contacts with Santa Fe in 1832, William Bent had no control over the men cutting wood for the post who were camped in the vicinity of the Cheyenne. Later in 1832, the Cheyenne consolidated with the Sutaio group (Grinnell 1962:87), a fact which suggests that the Cheyenne needed a political and social alliance to replace group members lost to epidemic fatalities.

Denig (1961) has complicated the historical record of epidemics by his insistence that the Crow and the Arikara suffered high mortality during this epidemic. No strong evidence suggests this happened. Denig (1961:58) stated that 200 Arikara died from smallpox and this exposure conferred immunity to the group as a whole, lowering the Arikara mortality rate during the 1837-38 smallpox epidemic. Perhaps some Arikara did contract smallpox during this epidemic, but half the group did die during the later epidemic (Pilcher 1838d). Denig's (1961:169-183) detailed account of the group disorganization of the Crow is excellent, but his disorganization did not result from the 1831-32 smallpox epidemic. From Crow informants, Bradley (1899:179-181) gave a near exact duplication of Denig's account but correctly attributed the historical event to a massive polyethnic Sioux and Cheyenne attack on a large, undefended Crow camp during the 1820s. Denig probably equated the Crow disorganization with that suffered by the Assiniboin during
the 1837-38 smallpox epidemic and visualized the similar mechanics of trauma response to indicate a similar causal agent.

The 1831-32 smallpox epidemic is important in that it prompted the first effective effort by the United States to vaccinate western Native American groups against smallpox. Jefferson ordered the Lewis and Clark expedition to carry vaccine; but before the commencement of the expedition, Lewis experimented with the vaccine and decided it was useless (Lewis and Clark 1893:1:xxix; Jackson 1962:36). In 1819, Long received the same orders for his expedition, but the vaccine was ruined in a canoe accident (Long 1906:1:202). Shortly after the 1818-20 measles and whooping cough epidemic, the Hudson's Bay Company instituted a program which vaccinated most Cree and several Assiniboine for smallpox.

During the 1831-32 epidemic, Dougherty independently commissioned one Dr. Bryant to vaccinate the Pawnee and other ethnic groups on the Lower Missouri, and his reports (Dougherty 1831a) led directly to an Act of Congress, passed on May 5, 1832, providing general funds for this purpose (United States Documents, Serial 233, Document 2:162). Two other doctors, Davis and Martin, were later hired; but despite Dougherty's suggestions, no attempt was made to vaccinate groups living in the Upper Missouri. Maximilian was told by an Indian Agent accompanying Dr. Davis that the total number of Native Americans vaccinated was in the neighborhood of 2,600 and that efforts stopped at the Sioux agency where the doctors were told: "Now we are well; if we should become sick it will be time enough to submit to the operation" (Maximilian 1906:1:286).
1831-1832

Figure 9: Smallpox

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
**1837-1838: Smallpox**

The smallpox epidemic of 1837-38 is the best known of all the epidemics which infected the Native Americans on the Northern Plains. Mortality was high because the youngest individuals surviving the 1800-03 epidemic were 39 years old; and for the majority of ethnic groups infected by the 1780-82 epidemic, the youngest immune survivor was over 55 years of age. Other epidemics, as well as deaths from warfare and other causes, reduced this immune reservoir to only a few individuals. The only other method to prevent smallpox was vaccination. Only the Cree, Assiniboine, and Sioux had been exposed to this preventative and then in limited numbers. The Atsina had contacted the 1831-32 smallpox epidemic and consequently suffered few fatalities. Pilcer, Indian Agent for the Upper Missouri, placed the aggregate mortality from this epidemic at over 17,200 individuals and indicated (1838d), "I have no doubt that if the above number were doubled, that it would be within bounds."

A number of stories in the literature dealing with this epidemic recount that an angry trapper placed infected clothing on the steamboat St. Peter and that the Mandans caught and spread the disease through a blanket stolen from a smallpox victim on that boat. No element of this story is historically demonstrable. The trapper most often accused of placing the contaminated articles upon the St. Peter is William P. May. It is doubtful that this man intentionally spread the disease as he was the one who brought news to Fort Clark that the epidemic was active on the Little Missouri (Chardon 1932:122). This news may have become garbled
on its way upriver: Culbertson (in Bradley 1900:221) believed May to be the author of the epidemic. From the incubation and development stages of smallpox, it is also unlikely that the Mandan contracted the disease directly from the St. Peter. Smallpox begins the pustulation phase about the 15th day after infection, but the first case of the epidemic at Port Clark was not noted until July 14, 1837, 24 days after the St. Peter left the Mandan village (Chardon 1932:118, 121). In the following discussion of the 1837-38 smallpox epidemic, only the most authoritative sources will be used.

Sixty miles above Fort Leavenworth, smallpox symptoms first appeared on the St. Peter. An employee of the Bureau of Indian Affairs (name unknown) suggested the victim, a mulatto deckhand, be put ashore. Pratte, the captain, refused, and several more cases developed before the St. Peter reached Council Bluffs (Pilcher 1838a). Count Arese (1934:65) was a passenger and was told that the disease was a mild form of "cholera" then present in St. Louis. At Council Bluffs, three Arikara women were among the embarking passengers; these women were later to infect the Arikara, Mandan, and Hidatsa. The St. Peter continued upriver, dropping off trade goods and annuities at various stops. Pilcher (1838a) disembarked at Fort Pierre. The St. Peter reached the Mandan village at Fort Clark on June 18, 1837 (Chardon 1932:188). The Arikara women probably disembarked to join an Arikara lodge group camped outside the Mandan village. Some 250 lodges of Arikara were invited to join the Mandan for their mutual defense early in 1837 (1932:80, 109).
The St. Peter arrived at Fort Union on June 24. Halsey, then the head of the post, was a passenger on the boat and had smallpox. He was recovering at the time, but several deckhands had died. In an attempt to halt the disease before the Assiniboine and Cree visited the post on their annual fall trade, Larpenteur (1898:131-132) attempted to variolate the four men and twenty women at Fort Union who had neither contacted smallpox nor been vaccinated. Variolation involves vaccinating a patient with smallpox rather than cow-pox, and great care must be taken to only use old scabs which contain antibodies and thus produce a mild form of the disease. Halsey's scabs apparently were too freshly formed as all the women and one man died of smallpox, and the disease consequently spread to the Assiniboine and Cree.

At the mouth of the Yellowstone River, the St. Peter halted, and keel-boats were dispatched to the upriver posts, Forts Benton and Cass. Harvey was in charge of the Fort Benton boat at the mouth of the Judith River and sent word to Culbertson that he had smallpox cases onboard. Culbertson ordered the boat in, and it arrived in early July, carrying two terminal smallpox victims. Despite warnings by Culbertson, the Blackfeet demanded trade, traded, and left Fort Union. After two months with no word from the Blackfeet, Culbertson went out to find them. A few days travel from Fort Benton, he found the remains of a camp of 60 lodges, the survivors of which were only a few old women. Smallpox also appeared at Fort Cass, but there was no trade at that post, and the Crow escaped infection (Bradley 1900:222-226).
The 1837-38 smallpox epidemic first broke out among the Santee Sioux (Pilcher 1938a). Pilcher (1938d) put the total Sioux mortality at about 1,200 individuals. The few Siux winter-counts even showing the disease indicate few fatalities (Howard 1960:374). Count Arese (1934:32-98) traveled through the Sioux territory east of the Missouri and indicated that mortality was higher in this region than west of the river. A possible explanation for the low Sioux mortality lies in the 1832 vaccination effort; and more importantly, the vaccination and variolation efforts carried out during 1837 by the American Fur Company (Pilcher 1838c). This effort minimized the infection of Sioux lands west of the Missouri River.

Apart from Larpenteur's abortive efforts at variolation at Fort Union, there were no other attempts to limit the disease as it spread across the Northern Plains. From a total pre-epidemic population of about 1,600, only 300 Mandan survived. The Arikara and Hidatsa each lost 1,200 to 1,300 individuals, a mortality rate of near fifty percent (Pilcher 1838d). The higher Mandan mortality resulted from their great numbers of suicides (Pilcher 1838d) and their refusal to scatter to avoid contagion as did the Arikara and Hidatsa (Chardon 1932).

The Assiniboine were reduced from 1,000 to 400 lodges; the inhabitants of the 200 of the surviving lodges had been vaccinated in Canada and only lost children. The dominant Assiniboine band headed by Le Gauche had only 150 survivors in 30 lodges from a population of 250 lodges before the epidemic (Denig 1961:72, 77). A further loss of 64 men and 8 women was sustained when La Gauche...
attacked the Mandan villages on July 12, 1838 only to be ambushed by the Arikara (Chardon 1932:115). The Assiniboine spread the disease to the Cree, but there were many fewer fatalities due to the Canadian vaccination program (Denig 1961:115).

To the west, the Blackfeet were particularly hard hit by this epidemic, with mortality estimates ranging from 6,000 (Bradley 1900:225) to 8,160 (Pilcher 1838d) individuals. The confluence of the Belly and St. Mary's Rivers became known as the Grave Yard or Whoop-up due to the especially high numbers of fatalities there (Dempsy 1965:9). It is evident from the description of this locale (Bradley 1900:225) that a large camp was stricken by the disease and did not fragment to avoid general contagion. Culbertson estimated that two-thirds of the Blackfeet died. Even the American trappers in the Three Forks, Montana area found evidence of the smallpox epidemic among the Blackfeet (Russell 1972:86-89). The adult Atsina were largely immune to the disease, and only 200 children died, while the Crow escaped uninfected (Bradley 1900:226). The Northern Cheyenne sustained no known fatalities (Grinnell 1962), but the Southern Cheyenne suffered high mortality when the smallpox epidemic spread south through the Pawnee to the Central and Southern Plains between 1838 and 1840 (Mooney 1898:274-275).

1846-1847: Measles and Whooping Cough

This epidemic entered the Northern Plains from the east from several centers of infection: immigrant trains, Missouri trading posts, and the Red River Settlement. Mortality seems to have been
Figure 10
Sioux Winter-Count Depictions of Disease (from Mallery 1972)
1837-1838

Figure 11: Smallpox

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
restricted to the old and the young, the former succumbing to the pulmonary infection and the latter to both components of the epidemic (Denig 1961:115). The highest mortality appears to be among the Metis (Denig 1961:115) and the Arikara (Moore 1846:290). The Cree and Assiniboine avoided the disease for the most part (Denig:115); and while the Mandan and Hidatsa caught the disease, they scattered to avoid contagion (Beckwith 1938:312). The Blackfeet also suffered limited infection, perhaps in part because a Cree war party carrying the epidemic turned back when they developed measles symptoms (Kane 1971:145). The Piegan Small Robes band did contract measles and spread the disease to the Crows when they conquered the group. De Smet (1906:2:242-243) recorded that when Crow children developed measles, the Piegan women urged the Crow to bathe victims in cold water, increasing Crow fatalities through hypothermic shock.

The Oregon Trail was a major center of infection for this epidemic. Winter-counts of western Sioux groups (Cohen 1942c; Curtis 1905:175; Howard 1960:371-378; Mallery 1972:322) record high mortality, and the Cheyenne also suffered greatly (Berthrong 1963:101). Among the Northern Plateau ethnic groups, the disease was particularly virulent and fatal as these groups had never been exposed to measles before (Mengarin 1977:196). Teit's (1930:316) informant stated that a primary cause of death was the use of cold baths and sweating to combat the disease and that Euroamerican opposition to these medical practices lowered the high mortality among the Salish. One band of Cayuse suffered about a fifty percent mortality, sparking a general uprising (Spalding and Smith
1958:342). The epidemic spread among the Shoshoni to the Crow and resulted in a more general epidemic than resulted from the Small Robes infection. The Crow suffered a significant mortality from this epidemic (Denig 1961:185-186).

1849:  Cholera

Cholera was present in the Mississippi drainage since 1833 but had never before been communicated to the Native Americans on the Northern Plains. Maximilian (1906:2:14, 107-108) reported the disease at posts along the Lower Missouri and that, at Dougherty's Indian Agency, seven of ten infected Americans died. Because of the quick course and highly fatal nature of this disease, high population density is necessary to create an epidemic. The prerequisite was satisfied when gold was discovered in California.

When news of the California gold discoveries reached the eastern United States, massive numbers of men gathered at St. Louis to await the spring traveling season. Cholera was brought to St. Louis by steamboats from New Orleans. The Oella No. 2 arrived at Fort Smith with cholera cases on board on April 12, 1949 followed by the Robert Morris on May 5 (Marcy 1939:142, 36). The first groups of Forty-niners escaped cholera, but later groups were infected. Billington (1962:226-228) hypothesized that of the 55,000 Forty-niners traveling to California over 5,000 died of cholera.

Native American bands and ethnic groups bordering the trails became infected with cholera through contaminated water and goods.
1846-1847

Figure 12: Measles

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
taken from abandoned wagons. In two months, an estimated 1,284 Pawnee died, one-quarter of their population (Barrow 1850:1078). Some 600 Crow died, including several prominent men (Denig 1961:186). An unknown, but probably large (Mitchell 1850:49), number of Sioux on the Upper Platte died. Major mortality occurred among ethnic groups south of the Platte. One major center of contagion was the polyethnic Kiowa Sun Dance camp, where the Kiowa had gathered with many Wichita, Arapaho, and Cheyenne. When the first victims of cholera died without any visible symptoms, the groups scattered in the face of horrible, invisible, sudden death (Grinnell 1962:2:165). All the ethnic groups present at the Kiowa camp reported a mortality of half the group population from the disease or disease-related suicides (Mooney 1898:289). Among the Cheyenne, three bands were virtually exterminated by this epidemic (Grinnell 1962:1:92). Western Sioux groups were also infected (Vestal 1934:188).

1850-1851: Smallpox

Smallpox was again spread across the southern rim of the Northern Plains during these years—in this case, from the Lower Missouri and from emigrant trains. Among the Mississippi Sioux, Sacs, and Foxes, mortality rates reached twenty percent (Lea 1851:265). Sioux winter-counts record significant numbers of fatalities, and De Smet (1905:2:649) inferred that deaths were primarily restricted to children among some bands. Vaughn (1853:354) recorded that 30 Crow lodges or 400 individuals died when infected Shoshoni spread the epidemic to them. In November 1851, Omaha
1849

Figure 13: Cholera

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
trading at Larpenteur's (1898:299) post passed the epidemic to his family. This occurred during the last months of the epidemic, and contagion within the Northern Plains was extremely limited.

1851: Influenza, Cholera

Influenza and cholera were brought into the Northern Plains by the Picotte steamboat St. Agne in 1851. Father De Smet (1905:640-643) was a passenger and recorded that despite several deaths from cholera, the boat did not turn back. At the Fort Pierre Sioux Agency, an influenza epidemic was encountered as were the recovering survivors of the smallpox epidemic of the same year (1905:649-650, 678-679). At the Arikara village at Fort Clarke, influenza and cholera joined to form a pandemic.

Kurz (1937) was present at Fort Clark and Fort Union and recorded the spread of the diseases on the Upper Missouri. After the arrival of the St. Agnes, influenza and cholera struck the Arikara and shortly afterwards the Hidatsa and Mandan (1937:71). Kurz, a painter, was told by the Native Americans to stop preparing sketches because, as with Catlin and Bodmer, he was causing an epidemic (1937:75-76). The Picotte traders at Fort Clark were said to have helped spread this rumor, perhaps to divert blame from their own company, but the Arikara learned the truth, killed a trader, and burned the Picotte post after looting it (1937:196-198). Kurz himself was forced to flee to Fort Union (1937:107). Only the influenza portion of the pandemic spread to the Crow, who lost from 150 (Kurz 1937:215) to 250 (Denig 1930:425) individuals.
1850-1851

Figure 14: Smallpox

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
Deaths among the Arikara were in excess of 300, and the Hidatsa and Mandan lost 20 warriors, "women and children not counted" (Kurz 1937:196).

1856-1857: Smallpox

The steamboat Clara, again of the Joseph Picotte Company, brought this epidemic upriver from St. Louis in a macabre duplication of the 1837-1938 smallpox epidemic. The Arikara, Mandan, Hidatsa, Assiniboine, Cree, Crow, Shoshoni, Nez Perce, and some Sioux bands contracted the disease. Mortality estimates of this epidemic indicate smaller numbers of fatalities among individual ethnic groups than during the previous epidemic. Given the excellent witness reports by Vaughn (1856, 1857), Warren (1875), and Redfield (1857), this lowered mortality rate resulted from the presence of a larger number of smallpox-immune individuals within the populations. The Arikara, Hidatsa, and Mandan suffered a mortality rate of about one-quarter of their populations (Redfield 1857:416). Assiniboine fatalities reached from 1,200 (Vaughn 1857:408) to 1,500 (Warren 1875:50), again one-quarter of the population (Redfield 1857:416). The Assiniboine spread smallpox to the Cree at Fort Ellice, where half of the Cree around the post were reported to have died (Palliser 1968:141). Major mortality occurred among the Crow.

Paradoxically, it was Agent Vaughn who was responsible for the Crow contagion. The Crow was warned by a Platte trader "that those of them who wished to die had only to visit Fort Union and receive their presents, as these contained the germ of smallpox"
Figure 15: Influenza, Cholera
It was "with much difficulty" that Vaughn (1856:632) convinced a group to collect the Crow annuities. The Crow also collected smallpox from infected Assiniboine at the fort. The following year, Redfield (1857:417) could not find the Crow to deliver annuities as they had scattered to avoid contagion after experiencing "considerable deaths." A conservative estimate of Crow mortality puts fatalities at over 1,000 (Warren 1875:50). Nez Perce bands contracted the disease with mild symptoms among at least one group (Stuart 1957:1:129), and 166 Sioux were also estimated to have died. This Sioux mortality was primarily among the Blackfeet Sioux (Warren 1875:50), but De Smet (1905:2:723) found an additional high child mortality rate among Oglala bands. The Blackfeet and Atsina escaped this smallpox epidemic.

1861-1864: Smallpox, Whooping Cough, Scarlet Fever

The epidemic which occurred during this period were, for the most part, restricted to the Upper Platte. One localized epidemic did occur among the Blackfoot band of the Blackfeet in 1864. Two factors explain the poor quality of the historical documents discussing Native American health through these years. One prime reason is that the Civil War and, particularly, the first Sioux War limited American contact with groups on the Northern Plains. With this limited contact, there were fewer sources to provide even gross data on the known epidemics. A second major factor is that fatalities from these epidemics were largely among children and, thus, of little importance to adult male-oriented Native American historians. Given these restraints, a discussion of epidemics during this period is presented with no separate heading.
1856-1857

Figure 16: Smallpox

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
In 1861 and 1862, a few Sioux winter-counts record that smallpox among western bands resulted in many fatalities among children (Cohen 1939:18; Mallery 1972:325). Greater and more general mortality was experienced by the Arapaho and Cheyenne, among other ethnic groups in the Southern Plains (Lovee 1862:275). Whooping cough also infected the Cheyenne in 1863 and again produced a high childhood mortality (Evens 1863:275). The captivity memoir of Fanny Kelly (1871:135) also recorded a high child fatality rate to disease among the Sioux, but the account does not specify which disease was present.

The year 1864 also brought smallpox and scarlet fever to the Northern Plains. Agent Wildenson (1864:417) found smallpox aboard the steamboat Yellowstone, but the disease apparently did not spread to Native American groups. Smallpox also appeared on the Platte, and several Cheyenne bands were vaccinated (Ketcham 1864:400-401). In a separate epidemic of scarlet fever, an estimated 1,100 Blackfoot died near Edmonton House (Dempsey 1965:14). This epidemic also struck the Piegan and caused an unknown number of fatalities (Curtis 1911:78).

1869-1870: Smallpox

The smallpox epidemic of 1869-1870 was communicated to the Northern Plains by the Metis. Only those ethnic groups which had escaped the smallpox epidemic eleven years earlier were affected to a major degree by this epidemic. The Atsina and Assiniboine were both infected by a Metis camp near Fort Peck in June 1869. The Assiniboine scattered and suffered little illness. The Atsina,
1861-1864

Figure 17: Various
however, had few immune individuals and suffered greatly with 741 deaths (Reed 1870:664), one-third of the ethnic group (Sully 1870b:654). Many of these fatalities resulted from the use of sweat baths and from suicide (Kock 1896:296). A Crow woman spread the disease to the River Crow band, but inoculation and fragmentation of the group limited mortality to only 30 deaths (Reed 1870). Ten lodges of Arapaho joined the Atsina during the epidemic and most of the visitors died of smallpox (1870:665). Among all the groups, the young adults composed the greatest numbers of fatalities (Sully 1870b:654).

Mortality among the Blackfeet groups reached considerable proportions. Because of the state of war which existed between the Blackfeet and the Americans in Montana, no government attempt was made to combat the disease; in fact, the Baker Massacre was perpetrated upon a camp that had suffered an average of six deaths daily for the previous two months (Sully 1870b:654). By July 1870, an estimated 1,400 Piegan and Blood Blackfeet had died of the epidemic, with "the greatest number of young men and women" victim to the disease (Reed 1870:660). More than 2,000 Piegan, Blood, Blackfeet, and Sard eventually died (Dempsy 1972:59-60).

Synthesis

The common characteristic of the epidemic diseases is that all were introduced to the New World by Euroamericans either as new disease types or as virulent new strains of an existing disease. The ethnic groups on the Northern Plains formed a "virgin-soil" soil population for the epidemics in that they had never
1869-1870

Figure 18: Smallpox

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
been exposed to the infections before. The lack of immunological resistance and effective medical treatment contributed to the high case mortality. The chronology of these epidemics is of some importance, since major epidemics struck many Northern Plains ethnic groups about every generation or, more specifically, when cohorts of women who had been born after the previous epidemic entered their reproductive periods. These women and their children, and the children of the women who had survived an epidemic when they themselves were children, would have no immunity to the epidemic diseases.

The pattern of diffusion of epidemics on the Northern Plains was necessarily a result of the patterns of social interaction among human populations as only humans can carry these infectious diseases. The geographic distribution was largely a product of social distance. Three general chronological periods mark sub-phases in this pattern. Before 1800, only smallpox appeared on the Northern Plains, diffused northward from the Spanish settlements in Texas and New Mexico. These smallpox epidemics were spread to the Northwestern Plains entirely by Native American interethnic contact; of these, only the 1780-82 epidemic became widespread over the whole region. The limited spread of the 1800-01 smallpox epidemic marks the end of this phase. Smallpox is communicable for a long time because of the infectious nature of pustule scabs and sporadic interethnic contact was sufficient to spread the disease. The pattern of distribution of the 1780-82 smallpox epidemic, however, indicated a high degree of such interaction at that time.
The second and third phases mark the appearance of other infectious diseases in the region, spreading from Euroamerican centers within the Northern Plains margins. The second phase, dating from the late 1790s to about 1820, relates to the inclusion of the Upper Missouri River into the Canadian trade network. Ray's (1976) study of the diffusion of Euroamerican epidemic diseases in the western interior of Canada provides insight into the disease pattern of both the second and third phases. Of the eight epidemics found to have occurred on the rivers of the Canadian plains between 1830 and 1850, only two were widespread on the Northern Plains. Both of these epidemics, the 1837-38 smallpox epidemic and the 1845-46 measles epidemic, also had major loci of infection on the Missouri River. Euroamerican fur brigades, infected at the major trade disbursement centers, carried the diseases to trading posts in the Northern Plains and to the Native American bands they contacted en route. These brigades reached the Canadian posts during the summer or early fall; it was during these seasons that disease contamination would have occurred. It was also during summer and fall that the Native American groups who frequently traded at the Canadian posts were near the Missouri River where they exploited the seasonal movements of bison herds (Moodie and Ray 1976). Due to the relatively short period for communicability of most epidemic diseases, epidemics of influenza, whooping cough, and some streptococcal infections could quickly end within a locality.

The establishment of American posts in the 1830s and the Oregon Trail in the 1840s made Euroamerican contact within the
United States a more active locus of epidemic disease after 1820, and particularly after 1849 with the opening of the gold fields. A growing number of contacts between infected Euroamericans and large numbers of non-immune Native Americans made this area ripe for the infection and spread of epidemic diseases as well as other infections.

The specific social processes involved in the spread of epidemic diseases are well documented in the historical and anthropological literature. Chardon (1932), Culbertson (Bradley 1900), Denig (1961), Henry (Henry and Thompson 1915) provide excellent first-hand observations of the spread of epidemics. These accounts indicate that, in general, interethnic contacts made through trade and warfare and through the flight of individuals from stricken populations were active in the spread of epidemics between ethnic groups. Within an ethnic group and between close allies, epidemics were spread through kith and kin relationship. Attempts to fell or to avoid the epidemics failed because of the incubation period of each disease, when infection is communicable but before disease symptoms have appeared.

The population estimates and other demographic information in the historical literature dealing with the Northern Plains are insufficient to permit the use of demographic statistics, but a general survey of the data does permit a few generalizations on population trends. The total populations of the Pawnee, Arikara, Hidatsa, Mandan, Shoshoni, and Salish ethnic groups dropped throughout the historic period. Those of the Blackfeet groups, Atsina, and Assiniboine peaked during the 1830s and thereafter
declined. Crow and Cheyenne estimates remain constant, although they do indicate a slow population growth with the population doubling approximately every 36 years among the group as a whole but with a range of variance between subethnic groups. Lodge populations, the expression of nuclear family groups, drop an average of twenty percent for all ethnic groups, indicating the growing effects of tuberculosis mortality and infertility, from infertility due to venereal disease, in addition to the catastrophic mortality from disease, warfare, and starvation.
CHAPTER III
SOCIOCULTURAL EFFECTS OF EPIDEMICS

The catastrophic and extensive mortality caused by epidemics on the Northern Plains resulted in sociocultural change, particularly as the surviving members of ethnic group populations regrouped into new co-residence units often times cross-cutting ethnic boundaries. Consequently, the ethnic identity of the newly formed co-residence groups became blurred. Frederick Barth (1969:11-12) has defined an ethnic unit, or ethnic group, as "a population which 1) is largely biologically self-perpetuating; 2) shares fundamental cultural values, realized in overt unity in cultural forms; 3) makes up a field of communication and interaction; 4) has a membership which identified itself, and is identified by others, as constituting a category distinguishable from other categories of the same order." According to Barth's idealization, ethnic groups are operating networks of culture and status-bearing individuals who define themselves as a distinct population on the basis of a set of defined and communicable symbols. The symbols assume a variety of cultural forms that are limited in the way they can be expressed by the environment, technology, and ideology of the ethnic group members involved. In contacts between different ethnic groups, these symbols and cultural forms are communicated and ideas exchanged, which tends to increase similarities between the self-identified populations within a geographical area. The medium of this communication is the co-residence group, an aggregation of individuals who may, or

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
may not, belong to the same ethnic group, but who do form a unified, cooperative local social group (Sharrock 1974:98). Any variable that alters the cultural symbols, symbolic arrays, communication, or limiting factors to cultural expression of an ethnic group or co-residence group will result in sociocultural change.

Epidemics on the Northern Plains fulfilled these criteria for sociocultural change by profoundly altering the membership composition of the co-residence units and ethnic groups. The sheer loss of group members and their cultural inventory inevitably caused change, but an even greater impact was probably felt when the socio-technical demands for personnel to fulfill subsistence, defense, and cultural continuity roles led to the incorporation of one co-residing ethnic subgroup into another, or the fusing of some of the members of different ethnic groups into one co-residence group. On a more individual level, refugees from stricken co-residence groups, and the relatives and descendants of such refugees, were incorporated into new co-residence groups, and formed lines of communication between the subethnic groups within an ethnic group and between different ethnic groups. Movements of individuals from one co-residence group to another resulted in increased interethnic contact and the diffusion of cultural forms across ethnic boundaries. The trauma resulting from the impact of epidemics, the incorporation of ethnic groups and subgroups, the regrouping co-residence units, and the new avenues of communication across group boundaries led to new definitions of personal identity.
It must be noted that catastrophic mortality from warfare can promote the same effects as epidemic disease mortality. However, such catastrophies were rare on the Northern Plains on a major ethnic group scale. The only example of catastrophic warfare mortality that occurred in this region before 1850 involved the Crow in the early 1820s when this ethnic group was attacked by a large group of Sioux and Cheyenne warriors. Denig (1961: 169-183) found the similarities between the effects of this warfare mortality and the epidemic disease mortality he witnessed to be so great that he erroneously attributed the sociocultural reaction to be one from a smallpox epidemic.

Epidemics were not the sole catalyst for sociocultural change on the Northern Plains, but they acted in concert with other factors as part of a web of new influences diffusing into the Northern Plains from Euroamericans in North America. Unlike the positive additive effects of the innovations possible with the advent of the horse, the gun, and increased trade, the sociocultural effects of epidemics were reducing in nature, the negation of a possible fluorescence of culture on the Northern Plains during the 19th century.

The sociocultural effects of the epidemic diseases introduced by Euroamericans to the Northern Plains ethnic groups fall into three levels: 1) temporary sociocultural disruption, 2) demographic and territorial change and, as a result, 3) permanent sociocultural change.
Temporary Sociocultural Disruption

Temporary sociocultural disruption was the immediate result of an epidemic, but its effects often continued for a decade or more. The disruption found its basis in differential survivorship from the epidemics. Death resulted from disease infection and complications, medical treatments such as the use of the sweat lodge and cold water plunge, and epidemic-related suicide; but as a rule, proportionately more men died than women, and more adults died than elderly individuals and young children. To escape the epidemic diseases, small groups of people often fragmented from larger populations; some ethnic group segments escaped with only a few fatalities, while others were decimated. The immediate post-epidemic concern was to regroup co-residence units to redefine the social organization of the ethnic group and to provide for the needs of the survivors.

A factor exacerbating the sociocultural disorganization caused by epidemic disease mortality was suicide. In this historical literature on the Northern Plains, suicide is found to be commonly associated with smallpox and cholera, but it occurred even as a result of influenza complications. Overt factors that led to suicide were the incomprehensible nature of epidemic diseases and the failure of religious and medical belief systems to halt the diseases. The disfigurement caused by smallpox pustules was viewed with universal horror because of beliefs surrounding the ritual mutilation of enemies, i.e., mutilation which occurred during life would be replicated in the afterworld. Four Bears, a Mandan chief, in a speech to the Mandans and Arikaras during the...
1837-38 smallpox epidemic announced, "I do not fear death my friends. You know it, but to die with my face rotten, that even the Wolves will shrink at horror at seeing Me...." (Chardon 1932: 125). The appearance of cholera, which shows no overt symptoms until the beginning of often fatal convulsions, was viewed with the same suicidal fear. A Cheyenne warrior rode through a polyethnic Arapaho and Cheyenne Sun Dance camp shouting, "If I could see this thing (cholera), if I knew where it came from, I would go there and fight it!" (Grinnell 1962:2:164-165), but many other Cheyenne and Arapaho committed suicide (Mooney 1898:289).

The inability of Native American belief systems to deal with epidemic diseases must have had some consequences for the existing ideologies; the historical documents record one attempt to control epidemics through shamanistic methods of sending disease to enemies (Tabeau 1939:185), and one destruction of a religious shrine after an epidemic (Thompson 1915:324). By the 1830s, the role of Euroamericans in introducing the epidemics was understood, and soon thereafter these diseases seem to have been classified by the Indians as beyond the reach of the Native American religious and medical belief systems.

Suicide also resulted from the loss of kith and kin and from the disruption of the societal role-playing network. To some degree these factors are related. Suicide can be an emotional response to the death of friends and kinsmen; the basis of this response can be seen, in part, as the loss of personal identity. Chardon (1932:132) recorded the case of a boy who refused to live after the death of his friends. Pilcher (1838b) suggested that
the more than ninety percent Mandan mortality during the 1837-38 smallpox epidemic was related to a high suicide rate. Pilcher attributed these Mandan suicides directly to the loss of friends and the fear of disfigurement; but on another level, it is likely that suicide resulted from the loss of personal and ethnic identity because of the destruction of the Mandan social organization. During the period of immediate and extreme sociocultural disorganization occasioned by epidemics, the fear of imminent death and the irrelevancy of playing social roles in a disintegrating social network led to identity fragmentation. Disfigurement by smallpox scars or physical impairments produced by the epidemic infections were causes of suicide since they created a dissonance between ideal and actual perception of identity (cif. Tanner 1956:96-98).

Following the immediate shocks of epidemics, there was a period of reorganization. The salient features of this reorganization have been outlined by Denig (1961:72) in his description of the Assiniboine following the 1837-38 smallpox epidemic:

Among the rest relationship was nearly extinguished, all property lost or sacrificed, and generally very old or very young persons were the only ones who recovered. Most of the principal men having died, it took years to recover from the shock. Young men had to grow up, remnants of bands had to be collected, new leaders to be formed, property to be had.

Strong leadership was an essential factor in minimizing the trauma of ethnic group reorganization. Such leadership could be provided by surviving authority figures (Thompson 1915:339-240) or could occur within a highly structured social movement. Good Boy of the Mandans (Bowers 1950) and Rotten Belly of the Crows (Denig 1961:170) achieved the statuses of culture heroes because
of their charismatic leadership during the reorganization period following catastrophic mortality. Failure to redefine the authority structure of co-residence units and ethnic groups resulted in sociocultural chaos, such as occurred among the Arikara after the 1780-82 smallpox epidemic. As detailed by Tabeau (1939), the confusion among the Arikara was resolved only by interethnic contacts with other groups, namely the Mandan, Hidatsa, Sioux, and Euroamericans who through opposition gave definition to the group.

Sociocultural confusion during the after high-mortality epidemics was manifested in large numbers of refugees who established their own co-residence units. In many cases, the absence of authorities to order the distribution of the survivors resulted in the loss of additional personnel through starvation and exposure (Tomison and Walker 1952:265; Russell 1972:89). A process essential to survival was the linkage of women and children with men for the division of labor necessary for subsistence. Mechanisms for small-scale incorporation of widows and children through marriage and adoption existed before Euroamerican epidemic diseases were introduced to the Northern Plains (Cocking 1908:112), but the catastrophic and differential mortality overwhelmed these sociocultural systems. Culbertson (Bradley 1900:226, 233) found the fragmentation of Blackfeet families and co-residence units so extreme that many individuals sought refuge with other ethnic groups rather than face reincorporation into their own group.

The higher mortality rate of males in polygynous families and the needs of the survivors of these large families, coupled with the loss of material goods through sacrifice and abandonment
during epidemics, created a need among Native Americans for the superior Euroamerican technology. Possibly related to this need, several traders noted an increase in Native American trade following a major epidemic (Bradley 1900:232); however, no study of the Hudson's Bay Company or American Fur Company records has been made to investigate this phenomenon. Euroamerican technology such as weaponry has been shown to have effected sociocultural change on the Northern Plains (Secoy 1953), but the role of an increasing dependence on domestic Euroamerican trade goods in promoting sociocultural change is unknown.

Another means of accumulating resources necessary for survival was by raiding other ethnic groups for subsistence goods and for goods for trade with Euroamericans. Abandoned or unguarded camps were scavenged by epidemic survivors (Bradley 1900:227; Tomison and Walker 1952:290), but the organization necessary for a raid on other ethnic groups usually was not possible during the immediate post-epidemic period. Le Gauche (Denig 1961:72, 77-78), a major Assiniboine chief, lost approximately 220 of the 250 lodges under his leadership during the 1837-38 smallpox epidemic and planned an attack on the Mandan to draw Assiniboine refugees under his control. The attack failed because the Arikara co-residing with the Mandan ambushed the Assiniboine war party, killing 68 men and capturing 8 women (Chardon 1932:167). Le Gauche committed suicide soon afterwards (Denig 1930:401). The sociocultural disruption immediately following an epidemic required the conservation of males to act as hunters instead of raiders. An expression of this acute need is illustrated by the
apparent promotion of Blackfeet male adolescents to heads of households following the 1837-38 smallpox epidemic (Bradley 1900: 232). Once a degree of sociocultural reorganization had occurred, effective interethnic raiding was more feasible.

**Demographic and Territorial Change**

Major demographic and territorial change on the Northern Plains before 1850 was, in part, directly related to the combined factors of epidemic disease mortality and warfare. Gradual population movements of groups westward was a part of the domino effect caused by Euroamerican activity ease of the Northern Plains, but sudden and dramatic changes were produced by the effects of epidemic disease mortality. Population loss and associated territorial loss could be direct results of major mortality from an epidemic of smallpox, measles, or cholera, or, less directly, be caused through the loss of children from less virulent epidemics and the subsequent disruption of the population curve. The effect is more demonstrable for the major epidemics, and so I will restrict my discussion to them.

Secoy (1953), in his analysis of the effect of the horse and gun on Plains warfare, found that a military fluorescence occurred when the horse and gun were incorporated into the sociocultural repertoire of an ethnic group. Dominance in military technology certainly enabled the group to expand its territory; however, in many cases this military dominance was enhanced by a large number of epidemic fatalities among the enemy.
Major territorial losses were suffered by epidemic-stricken groups at the western margin of the Northern Plains and by the horticultural village groups on the Upper Missouri River. In both areas, a situation existed in which the ethnic groups with unrestricted access to Euroamerican armaments happened to suffer a higher epidemic mortality than the neighboring groups with easy access to trade arms. To the west, the Salish and Kutenai habitually and continuously exploited the Northern Plains prior to the 1980-82 smallpox epidemic (Teit 1980); the Shoshoni, through their participation in the Spanish slave trade (Secoy 1953), were the enemy of practically every other ethnic group in the Northern Plains area. By the early 1800s, these three groups had been driven from the Northern Plains except for seasonal hunts. Both the Shoshoni (Lewis and Clark 1893:554) and the Blackfeet (Thompson 1915:336) explained the withdrawal of the western groups as resulting from the Blackfeet's possession of Euroamerican arms, but epidemic mortality was also a contributing factor (Teit 1930:315). Evidence suggests the western ethnic groups—the Crow, Kutenai, Nez Perce, Salish, and Shoshoni—wintered in large camps along the Rocky Mountain slopes (Bradley 1899:166; Spalding and Smith 1958:137; Teit 1930:315-316) and mortality in these camps from the 1780-82 smallpox epidemic was exceptionally high. The Blackfeet wintered in small groups, many of which escaped the disease (Tomison and Walker 1952:243, 268, 271, 288). The Blackfeet's lower mortality from this smallpox epidemic and their ability to acquire Euroamerican arms enabled them to drive their enemies westward across the Rocky Mountains. There was also, apparently,
a concerted effort by the Blackfeet to exterminate the Shoshoni by killing males and capturing women and children, both because they perceived an association of the Shoshoni with the epidemic and to recruit personnel into the Blackfeet ethnic group (Thompson 1915:399). A later smallpox epidemic struck the western ethnic groups in the 1801-02 period and increased the discrepancy of power still further. Several previously autonomous Salishan groups were forced to consolidate to form the historic Flathead ethnic group (Teit 1930:303-311).

Bowers (1950:1965) has discussed the magnitude of demographic and territorial change on the Upper Missouri River among the Mandan and Hidatsa, and Deetz (1965) discussed change among the Arikara. Fortified Arikara villages appeared in the mid-1700s (Deetz 1965:18), an indication that hostile contacts with other groups were increasing. Major change resulted from a series of epidemics that began about 1734-35. Bowers (1965:486) estimated the pre-epidemic populations of the horticultural village groups to have been 24,000 Arikara in villages between the Great Bend of the Missouri and the Grand River and 9,000 Mandan and Hidatsa in villages between the Heart and Knife Rivers. In 1804, Lewis and Clark (Lewis 1832:710-711) found about 2,000 Arikara, 1,250 Mandan, and 2,700 Hidatsa and attributed their decline to the effects of smallpox epidemics and attacks of the Sioux. At this time, Arikara villages were found clustered near the Grand River, and the Mandan and the Hidatsa both lived near the Knife River. This concentration of populations consolidated the remnants of about 18 Arikara villages (Tabeau 1939:124) into one cluster of
three villages, three distinct ethnic subgroups of Mandan into two villages (Bowers 1965:486), and three distinct Hidatsa ethnic subgroups into one village each (486). By 1847, the populations of these ethnic groups numbered 1,880 Ariakara, 1,350 Hidatsa, and 360 Mandan (Malock 1847:885). The reductions in numbers and concentrations in populations of these horticultural village peoples made them vulnerable to both epidemic diseases and Sioux attack; by 1862, the Arikara, Mandan, and Hidatsa were all co-residing in one village.

The operation of epidemic disease and warfare in effecting the consolidation of ethnic subgroups can also be suggested for the Arapaho (Kroeber 1902:5-7) and the Cheyenne (Grinnell 1905:141-142). The polyethnic co-residency and fused ethnicity of the Assiniboine and Cree resulted from numerous factors (Sharrock 1972), including those of warfare and epidemic mortality. The latter groups were engaged in active warfare with both the Blackfeet and Sioux. The combination of warfare and severe mortality of the 1837-38 smallpox epidemic led Denig (1961:63) to believe that the Assiniboine would not have survived if they hadn't merged with the Cree. Some 60 lodges of Assiniboine who had escaped the epidemic moved south from Canada to join the stricken bands in the United States following the epidemic. Mandelbaum (1940:183) suggested that the loss of territory by the Assiniboine from warfare and epidemic mortality was balanced by the Cree expansion into the Northern Plains, this expansion increasing co-residence between ethnic groups and the growth of fused ethnicity through polyethnic families (cf. Sharrock 1974:199).
The association of the Atsina with the Blackfeet subgroups before 1850 may have been the same sort of phenomenon, although ethnic boundaries were maintained (Kroeber 1908:145-146). A possible merger of the Arapaho and the Atsina was interrupted by the 1831-32 smallpox epidemic. The Atsina fled north to Blackfeet territory to escape the disease (Bradley 1900:226). The preservation of distinct ethnic subgroups among the Blackfeet and Sioux might have been due to their large population and nonsedentary lifeway; the Sioux, especially, avoided many later epidemics and were thus able to maintain a large population. Losses from warfare and epidemics offset the Sioux birthrate, resulting in a fairly constant Sioux population size. The Blackfeet, for much the same reasons, declined in population at a slower rate than neighboring ethnic groups. Because of their access to Euroamerican armaments and a large population from which to draw warriors, these ethnic groups were more subject than neighboring groups to sociocultural change engendered by territorial expansion and by the incorporation of extra-ethnic group prisoners.

Permanent Sociocultural Change

The processes of temporary sociocultural disruption and demographic and territorial change effected by epidemic diseases produced long-lasting and significant sociocultural change among the ethnic groups on the Northern Plains. The means by which the catastrophic epidemics were contracted and spread was beyond the comprehension of the Native Americans. Massive mortality occurred directly from disease infections, complications, and medical
treatments and indirectly from suicides resulting from the trauma of the loss of societal and personal identity. The high mortality necessitated a reorganization of sociocultural systems to incorporate epidemic survivors, both for the physiological and social needs of survivors and for the sake of the defense capabilities of the incorporating group. Often, the incorporation of survivors involved the amalgamation of members of one ethnic group within another.

This pattern of ethnic incorporation created extensive sociocultural change in the Northern Plains. Such change in major social institutions is indicated in the historical data from this region, but sociocultural change must have extended even to basic cultural symbols. In the case of the Arikara after the 1780-82 smallpox epidemic, Tabeau (1939:126) found that the linguistic differences between the Arikara medicine bundle groups, or ethnic subgroups, were reinforced by the attempts of each subgroup to maintain its variant of Arikara ethnicity:

Each of the tribes (Arikara medicine bundle groups) has its own particular one (dialect) so that no one can say that he knows the Ricara language....The alliances among the tribes (ibid), in which each party is jealous of its idiom, causes every child to adopt its language. The grandparents, the aunts, the uncles, and the nephews, who are brothers among the Savages, have all their different patois and, as all live ordinarily in the same lodge as one family, it seems like the tower of Babel and as if all speak without understanding.

In general, a sociocultural "patois" of shared understandings and behavior had to be developed for successful ethnic consolidation after an epidemic. This patois included the whole range of sociocultural phenomena and sometimes led to fused ethnicity.
The Arikara represent an extreme example of post-epidemic sociocultural confusion because there were no social avenues for ethnic subgroup consolidation. Even with ethnic consolidation around dominant sociocultural structures, such as a clan and moiety system embracing the ethnic group as a whole, change entered the social network. Among the Mandan (Bowers 1950), the number of clans was reduced from 13 to 7 to 4 after successive smallpox epidemics, and the ceremonial sanctions of these clans became devalued. Merging of the three Mandan subethnic groups created situations in which some ceremonies had several leaders of equal status and others no surviving leaders at all. Ascribed status and role performance in these ceremonies was also altered by epidemic mortality; because of the absence of individuals with inherited rights to take part in Mandan ceremonies, these ceremonies became non-exclusive. By 1850, consanguinal and affinal ties between the Mandan and the Assiniboine and Sioux were diffusing new elements into Mandan ceremonial life; these, in turn, were diffused to the Hidatsa and Crow through similar interethnic relations. Generally, the importance of ascribed statuses declined among the Missouri horticulturalist groups and were replaced by networks of achieved status gained through coup-counting and membership in fraternal societies.

The trend toward cognatic kinship and band structure on the Northern Plains is exemplified in the base substitution of the Mahsihkuta by the Hotamitaniu band among the Cheyenne as a result of fatalities from the 1849 cholera epidemic (Grinnell 1905:143-144). Grinnell, working with data from elderly informants, relates
that Cheyenne social organization followed about the same matri-local band pattern of the Crow until the mid-1800s. The Mahsihk-kuta, or Grey-Hair band, apparently carried the same genetic trait for grey hair as was found among Mandan and Hidatsa populations. During the 1849 cholera epidemic the band was exterminated and replaced within the Cheyenne ceremonial camp circle by the Hotamitaniu, or Dog Soldier band, whose membership was based on the fraternal social relations of a warrior society. Initially, the band was composed of men with non-Cheyenne wives, but its patrilocal and fraternal emphasis eventually made it the largest and most popular band, and thus a center of sociocultural innovation and change among the Cheyenne.

The trend of sociocultural changes promoted by the mortality and sociocultural trauma of epidemic diseases was toward poly-ethnic co-residency, interethnic marriage, and fused ethnicity, which led to sociocultural systems cross-cutting ethnic groups. Failure of traditional belief systems to prevent or explain epidemic mortality led to the incorporation of new symbol sets obtained from other groups and subgroups. Extreme stress on traditional systems of personnel recruitment, which were designed to deal with variables in resource distribution, warfare losses, and trade (Oliver 1962), resulted in confusion of personal and ethnic identity structures. High, recurrent mortality of ethnic group members from major and minor epidemics, and the subsequent proportional increase in warfare and other casualties, developed trends toward cognatic kinship systems and fraternal social institutions. Interethnic contacts between groups from trade and alliance
relations were strengthened by movement of epidemic survivors. Post-epidemic marriage and alliance networks served to transmit new cultural symbols and symbol sets throughout the Northern plains.
CHAPTER IV
DISCUSSION AND CONCLUSIONS

Modern ethnographic data from the Amazon Basin of South America are an appropriate testing field for this analysis of sociocultural change on the Northern Plains. Recent studies of the Kalopalo (Basso 1973), the Sharanahua (Siskind 1973), and the Yanamamo (Neel, Centerwall, Chagnon and Casey 1970) provide a social laboratory in which to test ethnohistorical data. As late as the 1940s and 1950s, a "virgin-soil" disease environment was preserved in the Amazon: among the Kalopalo of central Brazil, a 1946 influenza epidemic killed 13.88 percent of the ethnic group and a 1954 measles epidemic was fatal to 26.66 percent of the remaining population (da Silva 1972:267). In 1968, anthropologists (Neel, et al. 1970) observed the spread of a measles epidemic through the non-immune Yanomamo population. This epidemic had only limited mortality because of prompt Euroamerican medical care. The means by which the epidemics diffused were very similar to those in the Northern Plains: the diffusion of disease radiated from Euroamerican trading posts and missions, the infection being carried by native traders, people fleeing infected villages, and women and children captured in raids. Also, as on the Northern Plains, there was a peak season for epidemic disease spread in the Amazon Basin—the dry season, when intraethnic and interethnic contacts occurred most often.

A problem of validity in the cross-cultural comparison of the two areas is that there are differences in the ecological
habitats and the human adaptations toward these habitats. Ethnic groups on the Northern Plains were much more mobile than those of the Amazon Basin because of their bison hunting subsistence. This mobility increased the probability of contacts between different ethnic groups. In the Amazon Basin, the horticultural subsistence and jungle environment served to isolate co-residence groups and probably contributed to intraethnic warfare, a relatively infrequent occurrence on the Northern Plains. Whereas most ethnic groups of the Northern Plains region interacted extensively with other groups, in the Amazon Basin, co-residence groups interacted almost entirely intraethnically with other co-residence groups. This suggests that the interethnic exchange of personnel and ideas seen on the Northern Plains would not be duplicated in the Amazon Basin. Instead, the Amazon Basin ethnic groups were forced to consolidate within the ethnic group as a whole. The cross-cultural comparison remains worthwhile nevertheless, as it illustrates the intraethnic sociocultural effects of catastrophic epidemic disease mortality.

A perhaps instructive insight into the feelings possibly experienced by Northern Plains Indians following the illness or death of friends or relatives can be gained from Siskind's (1973: 44) ethnography of the Sharanahua of eastern Peru, an ethnic group that lost one-half to two-thirds of its membership to epidemics between 1925 and 1950. Siskind related:

The world seems small and closing in and each regards his kin as a small island of strength, the loss of even one to be a personal danger. The wailing for a sick kinsman is highly stylized, the dirge like, "my child is dying, my child is dying",
and is fueled, perhaps, not by grief for a unique person, but for the danger of one more tree gone in a small grove, never to be replaced.

The overt prayers to avoid epidemics that Ewers (1973) found among the Comanche do not appear to have been duplicated on the Northern Plains, but the chaos caused by epidemics was known to all adults. As with the Sharanahua, the remembrance of what had once been was vocalized at the impending loss of yet another relative or friend.

The study of post-epidemic sociocultural effects in the Amazon Basin is severely limited by the lack of long-term historical information. The recent polyethnicity among the Upper Xingu of Brazil is probably an indirect product of epidemic disease mortality but is more directly a result of the purposeful consolidation of ethnic groups into this area by the Brazilian government (Basso 1973:vii-viii). Among the Sharanahua, however, Siskind (1973:52) did find ethnic fusion to be a result of epidemic mortality and the scarcity of permissible marriage partners. This mortality lowered the marriage distance within Sharanahua society, leading to the abandonment of distant classificatory sib terms (Siskind 1973:64). In general, as on the Northern Plains, there was an ongoing trend toward a bilateral cognatic kinship terminology and a merging of co-residence groups (1973:44-46, 199-200). Basso (1973:viii) did not attempt to measure the sociocultural impact of epidemic disease mortality on the Kalopalo but infers that it was great. In a discussion of the difference between the real and ideal practice of Kalopalo politics, however, one part of the impact is made explicit: Kalopalo individuals with hereditary
leadership rights were replaced in leadership roles by individuals with strong networks of kith and kin (Basso 1973:132-138). The development of social factions among this ethnic group was seen by Basso (1973:119-121) to be a product of the 1954 measles epidemic, which eliminated overwhelming support for any one leader. This internal conflict, often observed on the Northern Plains, may possibly lead to future co-residency of at least one Kalopalo faction with another ethnic group should the conflict not be resolved.

The sociocultural effects of epidemic disease mortality seems to be very much the same among ethnic groups of both the Northern Plains and the Amazon, in type if not in kind; and it appears that the effects engendered by catastrophic mortality constitute a distinct set of influences in producing sociocultural change. The distinctiveness of this set of influences relies on the fact that the mortality alters the basic demographic structure of an ethnic group without directly adding any new variables to its sociocultural repertoire. Because of the world-wide contacts of Europeans with non-immune populations beginning with the 15th century, it is very probable that all ethnic groups in the Americas, Africa, Australia, and the Pacific Islands were strongly affected by this type of sociocultural change as were the Europeans themselves during plagues of Black Death during the 7th and 14th centuries.

On the Northern Plains, the fluorescence of the horsetrans-human Plains culture prior to 1850 occurred as a result of the interplay of many factors. Physical movement of populations into
the region brought about increased contact between representatives of different sociocultural systems. Trade networks transmitted both ideas and goods among ethnic groups. Superior technology increased the efficiency of exploitation of the Plains environment, and the facility of transporting material goods and personnel was increased by the acquisition of the horse. Despite these innovations, by 1850 the Native American ethnic groups on the Northern Plains presented "...a gloomy prospect for the future" (Mitchell 1851:324).
Table 1 - Characteristics of Major Epidemic Diseases
(after Benenson, 1971)

I. Smallpox

Types: Alastrim (Variola minor): Less extreme symptoms with case fatality rate of 2% or less.

Classic Smallpox (Variola major): Extreme symptoms with case fatality rate of unvaccinated at 40 to 50%.

Mode of Transmission: Droplet spread; contact with respiratory discharges of the skin and of the mucous membranes of the patient; contact with articles contaminated by the above.

Period of Communicability: From development of the earliest lesions to the disappearance of all scabs.

Incubation Period: Normally 10 to 12 days to onset of illness and 2 to 4 more to the onset of the rash.

Symptoms: Sudden onset of fever, malaise, headache, severe backache, prostration, and occasional abdominal pain.

Mortality: Deaths normally occur between the 5th and 7th day. Hemorrhagic cases, characterized by bleeding from the nose and ears, die on the 2nd day.

II. Measles

Mode of Transmission: Droplet spread; contact with respiratory or urine secretions of patient; contact with articles contaminated by the above.
Period of Communicability: Beginning of the prodromal period to four days after the appearance of the rash.

Incubation Period: About 10 days after exposure to initial rash.

Symptoms: Cough and fever. Dusky red blotchy rash appears on the 3rd to 4th day and lasts 4 to 6 days. Secondary pneumonia possible.

Mortality: High case fatality rate with long intervals between infections. Mortality during high fever period and to secondary complications.

Immunity: Conferred through infection.

III. Group A Hemolytic Streptococccic Infections

Types: Wide range of diseases differentiated clinically by the portal of entry and tissue of localization.

A. Streptococcal sore throat: Localized to throat.

B. Scarlet fever: Streptococcal sore throat with a rash. This rash does not involve the face except in Negroid populations, but there is some flushing.

C. Erysipelas: Locii on face and legs of a velvety rash with a raised border. Mortality to 10% in this form.


Mode of Transmission: Principally by intimate contact and with contact with nasal secretions of patient; also droplet spread and by contaminated articles and food.
Period of Communicability: Ten days during clinical illness, low order from 2 or 3 weeks, in carrier stage for months.

Incubation Period: One to 3 days, rarely longer.

Symptoms: Variable to locii of infection. High fever, nausea, and vomiting accompany severe infections. Secondary infections include acute rheumatic fever. Symptoms may last months.

Mortality: Variable to locii of infection and secondary complications.

Immunity: Conferred immunity to rash only.

IV. **Whooping Cough (Pertussis)**

Mode of Transmission: Droplet spread; contact with mucal secretions; contact with articles contaminated by above.

Period of Communicability: Highly communicable during the early catarrhal stage; communicable during the entire infection.

Incubation Period: About 7 days.


Mortality: Fatality to 15% of cases, 70% of deaths in patients under 1 year of age.


V. **Cholera**

Types:  
A. **Cholera infantum**: Mild case, usually in children.  
B. **Cholera morbus**: Asiatic cholera, extreme cases.
Mode of Transmission: Ingestion of water contaminated by feces and vomit of patients; contact with feces of patient; ingestion of food contaminated by water, soiled hands, and flies.

Incubation Period: One to 3 days.

Symptoms: An intestinal disease characterized by sudden onset, acute cramps, profuse watery stools, acidosis, and circulatory collapse.

Mortality: Case fatality in untreated cases may exceed 50%, death may occur within a few hours of onset.

Immunity: Conferred through infection.
REFERENCES CITED

Arese, Count Francisco

Audubon, John

Barrow, John E.

Barth, Fredrick

Basso, Ellen B.

Beckwith, Martha Warren

Benenson, Abram S.

Berthrong, Donald J.

Billington, Ray Allen

Bowers, Alred W.
Brackenridge, H. M.

Bradbury, John

Bradley, James H.
1900 Affairs at Fort Benton from 1831 to 1869. Contributions to the Historical Society of Montana 3: 201-287.

Brunner, Edward M.

Cabeza de Vaca, Alvar Nunez

Cadott, N.

Carver, John
1778 Travels Through the Interior Parts of North America in the Years 1766, 1767, and 1768. London: Printed for the Author and sold by J. Walter at Charing-Cross and S. Crowder in Paternoster Row.

Chardon, Pierre
Clark, W. P.

Cockburn, T. Adrian

Cocking, Matthew

Cohen, Lucy Kramer


Culbertson, T. A.

Curtis, Edward S.


da Silva, Pedro Agostinho

Deetz, James
Dempsey, Hugh A.  


Denig, Edwin Thompson  


De Smet, Pierre-Jean  

De Vaca, Cabeza  

Dobyns, Henry F.  

Dolassus  
1952 "Letter, Dolassus to Casa Calvo, April 3, 1801," in *Before Lewis and Clark*, A. P. Nasatir, ed., St. Louis, St. Louis Historical Documents Foundation.

Dougherty, John  
1831 *Letter to William Clark, October 29, 1831*. Bureau of Indian Affairs, Letters Received from the Upper Missouri Agencies, 1824-1881. United States Documents, Record Group 75, Serial Number 234, Microfilm Roll 883.

Evens, John  
Ewers, John C.


Fenner, Frank

Fidler, Peter

Franklin, John

Fulkerson
1838 Letter to William Clark, June 10, 1838. Bureau of Indian Affairs, Letters Received from the Upper Missouri Agencies, 1824-1881. United States Documents, Record Group, Serial Number 234, Microfilm Roll 883.

Glassner, Martin Ira

Gregg, J. B.

Grinnell, George Bird


1972 Blackfoot Lodge Tales. Lincoln: University of Nebraska Press.
Harmon, Daniel W.  

Henry, Alexander and David Thompson  

Houck, Louis  

Houghton, Douglas  
1832 *History of Smallpox in the Chippewas of Lake Superior.* 

Howard, James H.  
1960 *Dakota Winter Counts as a Source of Plains History.* 


Hunter, John Dunn  

Hyde, George E.  

Jablow, Joseph  

James, Edwin  

Kane, Paul  
Kehoe, Alice B.  

Kelly, Fanny  

Ketcham, H. T.  

Koch, Peter  

Kroeber, Alfred L.  


Kurz, Rudolph Friederich  

La Jonquire  

Larocque, Francois  
1911 Journey from the Assiniboine to the Yellowstone River 1805. Publications of the Canadian Archives 11 Ottawa.

Larpenteur, Charles  

Lavender, David  
La Verendrye, Pierre Gaultier

Lewis, Meriwether
1832 *Statistical Review...American State Papers, Indian Affairs.* Vol. 1, Doc. 113, pp. 706-721.

Lewis, Meriwether and William Clark

Libby, O. G.
1920 *The Arikara Narrative of the Campaign Against the Hostile Dakotas, June 1876.* North Dakota Historical Collections 6.

Liberty, Margot

Linderman, Frank B.
1962 *Plenty-Coups, Chief of the Crows.* Lincoln: University of Nebraska Press.

1972 *Pretty Shield, Medicine Woman of the Crows.* Lincoln: University of Nebraska Press.

Lovee, John

Luttig, John C.

MacDonnell, John

Mackenzie, Charles
Mallery, Garrick

Malock, G. C.

Mandelbaum, David G.

Marcy, John

Martin, Calvin

Matthews, Washington

Maximilian, Prince of Wied-Neuwied

McLeod, Archibald H.

Mengarini, F. R. Gregory

Mitchell, D. D.

Moodie, D. W. and Arthur J. Ray

Mooney, James


Moore, T. P.

Morgan, Lewis Henry

Nasatir, A. P.
1952 Before Lewis and Clark. St. Louis: St. Louis Historical Documents Foundation.

Neel, James V., Willard R. Centerwall, Napoleon A. Chagnon, and Helen L. Casey

Newman, Marshall T.

Nute, Grace Lee (ed.)
1942 Documents Relating to Northwest Missions 1815-1827, Minnesota Historical Society, St. Paul.

Oliver, S. C.
1962 Ecology and Cultural Continuity as Contributing Factors in the Social Organization of the Plains Indians.
University of California Publications in American Archaeology and Ethnology, Vol. 47.

Palliser, John

Pease, William B.

Pilcher, Josiah
1838a Letter to William Clark, February 5, 1838. Bureau of Indian Affairs, Letters Received from the Upper Missouri Agencies, 1824-1881. United States Documents, Record Group 75, Serial Number 234, Microfilm Roll 884.

1838b Letter to William Clark, February 27, 1838. Bureau of Indian Affairs, Letters Received from the Upper Missouri Agencies, United States Documents, Record Group 75, Serial Number 234, Microfilm Roll 884.

1838c Letter to William Clark, July 3, 1838. Bureau of Indian Affairs, Letters Received from the Upper Missouri Agencies, 1824-1881. United States Documents, Record Group 75, Serial Number 234, Microfilm Roll 884.

1838d Letter to William Clark, September 12, 1838. Bureau of Indian Affairs, Letters Received from the Upper Missouri Agencies, 1824-1881. United States Documents, Record Group 75, Serial Number 234, Microfilm Roll 884.

Provencher, Father

Ray, Arthur J.

Reed, A. S.

Reed, Henry H.

Redfield, A. H.

Roberts, Kenneth

Robertson, Colin

Robinson, Doane

Roe, F. G.

Russell, Osborne

Secoy, Frank R.

Sharrock, Susan R.

Sibley, John
1832 "Historical Sketches of Several Indian Tribes in Louisiana...," American State Papers, Indian Affairs, Vol. 1., Doc. 113, pp. 721-725.

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
Simpson, George
1847 Narrative of a Journey Round the World During the Years 1841 and 1842. London: Henry Colburn Publisher.

Siskind, Janet

Spalding, Henry H. and Asa Bowen Smith

Stearn, E. Wagner and Allen E. Stearn

Stuart, Granville

Sully, Alfred


Tabeau, Pierre Antoine

Tanner, John

Teit, James A.
Thompson, David  
1915 David Thompson's Narrative. J. B. Tyrrell, ed.,  
Publications of the Champlain Society 12, Toronto.

Thwaites, Reuben Gold  
1908 The French Regime in Wisconsin, 1743-1760. Wisconsin  
Historical Collections, Vols. 16, 17, and 18.

Tomison, William and William Walker  
1952 Cumberland House Journals and Inland Journals, 1775-82,  
Hudsons Bay Record Society.

Truteau, Jean Baptiste  
1952a Letter to Baron Carondelet, July 3, 1796. In: Before  
Louis: St. Louis Documents Foundation.

1952b Truteau's Journal, 1794-1795. In: Before Lewis and  
Louis Historical Documents Foundation.

Vaughn, Alfred D.  
of Indian Affairs. House Executive Documents 1. 33 Cong.,  
1 Sess., (Ser. 710), pp. 352-359. Washington, D.C.

of Indian Affairs. House Executive Documents 1. 34 Cong.,  

of Indian Affairs. House Executive Documents 1. 35 Cong.,  

Vestal, Stanley  
1934 New Sources of Indian History 1850-1891. Norman:  
University of Oklahoma Press.

Vogel, Virgil J.  

Warren, G. K.  
1875 Preliminary Report of Exploration in Nebraska and  
Dakota in the Years 1855-'56-'57. Department of the  
Wheat, Carl I.
1957 Mapping the Trans-Mississippi West. San Francisco: Institute of Historical Cartography.

Wheelock, T. B.
1834 Journal of Colonel Dodge's Expedition from Fort Gibson to the Pawnee Pict Village, Summer, 1834. American State Papers, Military Affairs 5: 373-382.

Wilson, Gilbert Livingstone

Wissler, Clark