LAKE EFFECT SNOWS IN WESTERN LOWER MICHIGAN: AN ANALYSIS OF THE DISTANCE INLAND OF GREATEST SNOWFALL ACCUMULATION

AND

THE DIFFUSION OF TUBERCULOSIS IN AFRICA, WITH PARTICULAR REFERENCE TO SOUTHERN AFRICA

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ABSTRACT

LAKE EFFECT SNOWS IN WESTERN LOWER MICHIGAN:
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SNOWFALL ACCUMULATION

By

Eugene Keith Dayton, Jr.

This study investigated two hypothesized causes of the variation of the distance inland of maximum snowfall for a given lake snow event in western lower Michigan. The first hypothesis stated that as windspeed (during the lake snow) increases, the distance inland of greatest snowfall receipts would increase. The second hypothesis stated that as the difference between the temperature of the airstream passing over Lake Michigan (during the lake snow) and the temperature of the water increased, the distance inland of greatest snowfall would decrease.

"Lake snow days" were selected on the basis of synoptic weather conditions from available data for a ten-year period, 1960-1970. Lake snow events occurring in western lower Michigan from the months of November to February were chosen, excepting those which occurred under northwesterly or northerly surface wind conditions and those which were accompanied by more than trace amounts

of snowfall in Wisconsin. An index of windspeed, based on the barometric pressure difference between Chicago and Traverse City, and a computed air-water temperature contrast were correlated with the distance inland of greatest snowfall for each selected lake snow day as recorded at weather stations located on four west-east transects in the study area. Both graphic and computer analyses were employed. The analyses suggest that the windspeed hypothesis is not supported by the evidence presented in this study, but that the air-water temperature contrast may account for 9% of the variance of the distance inland of greatest snowfall during lake snows in the western Lower Peninsula. This study concludes that intensive studies of lake snow events, using on-site instruments, might be a more appropriate way of dealing with this complex, mesoscale climatic phenomenon, especially since appropriate data are not readily available from documentary sources.

LAKE EFFECT SNOWS IN WESTERN LOWER MICHIGAN: AN ANALYSIS OF THE DISTANCE INLAND OF GREATEST SNOWFALL ACCUMULATION

By

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Introduction

One of the most striking weather features of the Great Lakes area is the phenomenon known as the lake snow. Lake snows are a type of snow storm, generally localized, which occur during certain synoptic weather conditions in areas located downwind from major bodies of water, such as the Great Lakes. In lower Michigan lake snows commonly occur from November to March within a broad belt paralleling Lake Michigan in the western part of the peninsula.

Despite being the focus of considerable climatological research, lake snows remain somewhat of an enigma. Typically, lake snows vary greatly in the frequency of their occurrence, in their intensity, and in the extent and locations of their occurrence. Usually, within the area affected by a given lake snow, the accumulation of snowfall is unequally distributed. Thus in Michigan the heaviest snowfall accumulation for a given lake snow may occur at the lakeshore, or elsewhere in an area up to 50 miles (or more) away from the lake. This paper will report on research which attempted to explain part of the variance of the distance inland of greatest snowfall accumulation during lake snows.

It was hypothesized that two variables, windspeed and the difference in temperature between the water of Lake Michigan and the air passing over it, could be correlated to the distance inland of greatest snowfall. Investigation of the effects of these variables was based on two hypotheses. The first was that as windspeed increased, the distance inland of heaviest snowfall accumulation would increase. The second was that as the difference between air and water temperatures increased, the distance inland of greatest snowfall accumulation would decrease. The research involved selection and analysis of available climatological data. The results seem to uphold the hypothesized inverse relationship between the air-water temperature contrasts and the distance inland of heaviest snowfall, but indicate there is little relationship between windspeed and the distance inland of greatest snowfall.

Lake Snows

Lake snow phenomena have been observed and analyzed for many years. In 1942 Remick noted the general synoptic and local conditions which characterize the lake snow (1). He cited a strong flow of polar air, a large air-water temperature contrast, a long over-lake wind trajectory, and orographic effects as being responsible for the lake-effect snowfall which he termed "snow flurries." Subsequent research has refined and added to these observations.

The lake snow is essentially snowfall which occurs due to the presence of a large body of water located upwind of the area where the snowfall occurs. The snow may occur in the absence of a low pressure system, and does not fall in areas upwind of the body of water.

In the Great Lakes region lake snows occur during strong polar outbreaks characterized by "the southeastward penetration of cold continental-polar air or arctic air into the central and eastern United States behind a low northeast of the Great Lakes" (2). From October to May this condition may produce localized snowfall on the lee shores of the Great Lakes. Locating the high to northwest of the lakes and the low to the southeast causes lake snows on the southern shores.

The mechanisms of the lake snow are partly understood. During the winter, the lakes act as heat and moisture sources for air passing over them. This occurs even in late winter and early spring when ice may cover substantial portions of the lakes (3). F. K. Hare has observed, "It is characteristic of airstreams undergoing such modification that they become highly unstable, vigorous convection being set up in their lower layers, accompanied by much cumuliform cloud" (4). As the water surface presents less frictional resistance to the air, windspeeds increase causing greater airstream vorticity. On reaching the lee shore, greater land resistance causes low-level

speed convergence. The warming of the lower layers steepens the lapse rate so that saturation is reached more easily (1). These effects, and possibly orographic ones also, trigger the lake snows.

Peace and Sykes, however, have expressed the view that the primary controls for lake snow occurrences are in conditions aloft, rather than at the surface. Although the authors hesitate to generalize from their detailed study of a Lake Erie snow storm, they cite "the lack of any indication of surface causes in these analyses except the tendency for the isallobaric minimum to lead band movement," concluding that "further understanding must await better upper-air observations" on the mesoscale (5). Namias has indicated that the gradual increase in snowfall from the 1930's to the 1950's in northeastern Ohio, northwestern Pennsylvania, and western New York is due to changes in long wave patterns so that more Alberta-type cyclones are brought to the mid-west (6). As lake snows may be considered "post-cold front lake squalls" (7) whose frequency would increase with the more frequent passage of frontal systems, the view that upper level controls are predominant may be correct. On the other hand, Schaefer and Changnon have separately indicated that increasing air pollution may be the major factor in the increase of snowfall in the Erie-Ontario area and in the Chicago-La Porte and southerwestern lower Michigan areas in recent years (8,9).

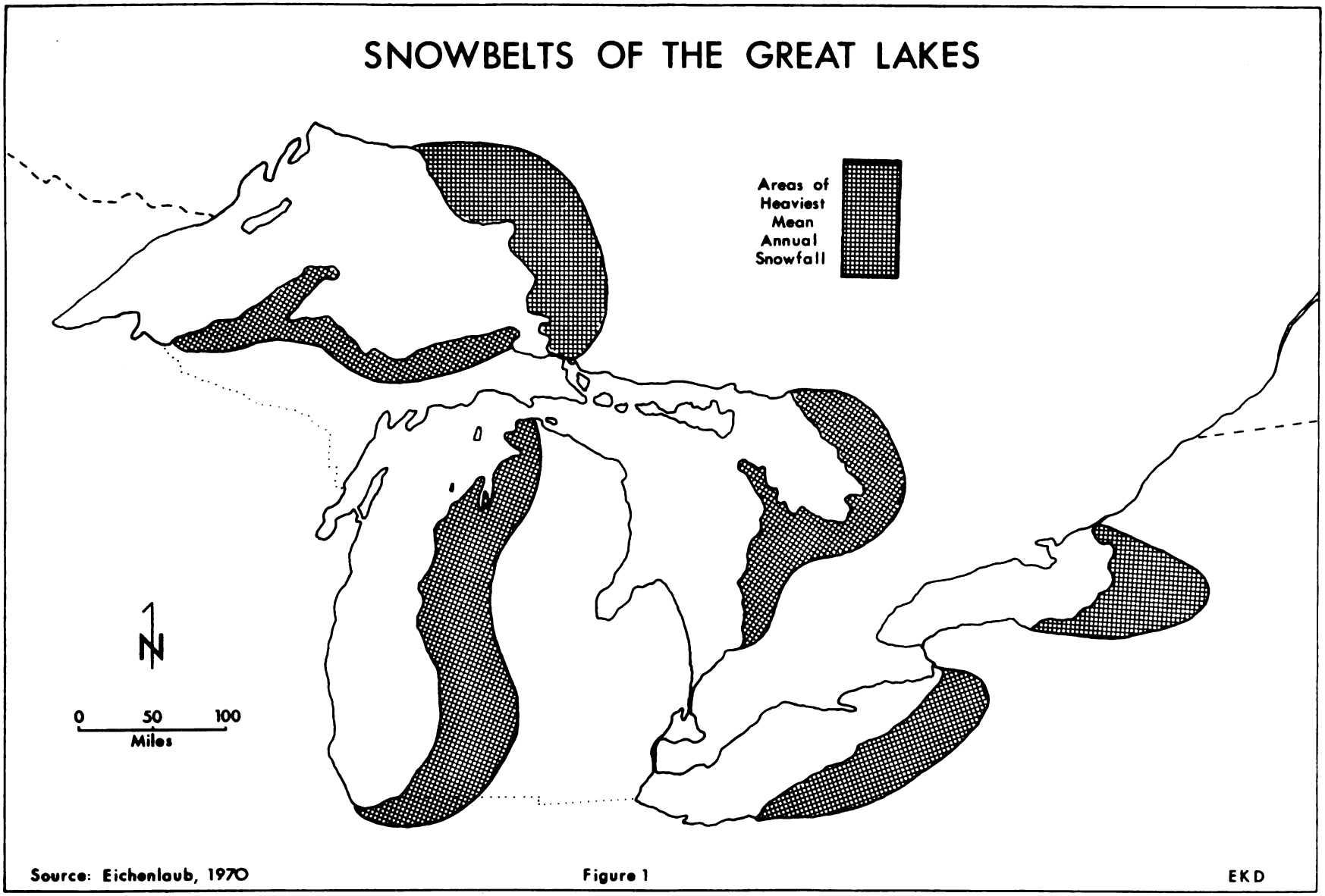
The intensity of lake snows may vary considerably. Frequently, however, they are spectacular, in some cases bringing 3 to 4 feet of snow in a two day period (10). On the other hand, lake snows may contribute relatively less precipitation to the seasonal totals than do frontal snows as the water content of lake snows is often much less than that of frontal snows, with an average snow-to-water ratio of 18:1 vs. 10:1 for frontal snow (11,12,1). Nevertheless, the intensity of the storms may be great enough to reduce visibility to zero on occasions, thus constituting a traffic hazard (11). The intensity of these storms has been related to the length of the airstream trajectory over open water, the air-water temperature difference, the atmospheric lapse rate, the vorticity of the airstream, and local orographic and site conditions (5,10).

The areal extent of the snowfall also may be related to the above factors. For example, relatively high landforms located near the lakeshore may restrict the areas of greatest accumulation of snow to the windward slopes--as in the Keweenaw Peninsula in Upper Michigan. Petterssen and Calabrese have suggested that the maximum ascent of air would occur where the lapse rate varies most rapidly (13). Thus, the location and extent of the area of greatest lapse rate variation might affect the location and extent of snowfall. Additionally, the areal extent is dependent on the alignment of the lake with the direction of flow of the

airstream. As the prevailing winter winds in the Great Lakes region are westerly, the areas which most frequently receive lake snows are the eastern shores. Figure 1 on page 7 (adopted from V. E. Eichenlaub (14)) illustrates this pattern.

However, Figure 1 fails to portray a significant difference between the areal extent of individual lake snows which occur on the lee shores of west-east aligned lakes (assuming a westerly wind) such as Lakes Superior, Erie and Ontario and of those snows occurring to the lee of north-south aligned lakes, such as Michigan and Huron. Lake snows to the lee of the former lakes, especially Erie and Ontario, tend to occur in narrow, elongated "corridors" having a west-east alignment. Lake snows from the latter, Lake Michigan in particular, tend to occur in broad belts paralleling the shoreline.

The Erie-Ontario type lake snows are generated by cloud trains which "generally consist of only a single band of cumulus congestus cells, with two bands occasionally coexisting over a single lake" with a general west-east alignment. As the clouds move overland, local winds shift to converge on the snow bands, thus intensifying their activity (5). These bands are generally narrow--only 15-20 miles wide, or less--but due to their long fetch over water, are heavily moisture laden, and may cause over three feet of snow to fall overnight in the


SNOWBELTS OF THE GREAT LAKES
Areas of Heaviest Mean Annual Snowfall
N
0 50 100
Miles
Source: Eichenlaub, 1970
Figure 1
EKD

affected area, while nearby areas may receive little or no snow at all (11). However, the snowfall boundaries may be gradual on other occasions (1).

In contrast, the Lake Michigan type lake snows (again assuming westerly winds) commonly occur over broad areas, usually within 20 miles of the eastern shore of the lake (15). These areas may be of great extent, affecting virtually all of the western part of the lower peninsula, or they may be discontinuous, or even localized. Occasionally, these snows occur as "bands of snowfall from 1 to 50 miles wide and from 25 to 100 miles long," being more like the Erie-Ontario type (14). It is interesting to note that when strong winds from the north or northwest occur these lake snow patterns are modified, so that southwestern Michigan and the Chicago-LaPorte areas receive the Erie-Ontario type of long, narrow "corridor" snowfall, greatly intensified by the long fetch of air over Lake Michigan. Elsewhere in western lower Michigan, under those conditions, no snowfall may occur, while the northern shore of the upper peninsula may receive the broad belts of snow which are generally typical of the western lower peninsula under westerly wind conditions.

One of the most puzzling aspects of lake shows is that the distance inland of greatest snowfall may vary greatly from one snowfall to the next. Eichenlaub has

averaged lake snow accumulations for western lower Michigan and reported that the heaviest mean annual accumulation from lake snows is found from 20 to 40 miles inland from the lake (14). But the heaviest snowfall for any given lake snow may occur anywhere from the lakeshore to 50 miles or more inland. This study was undertaken to discover in part why this variation in the distance inland of greatest snowfall accumulation takes place.

The causes of this variability are unknown. Petterssen and Calabrese have noted that this variability occurs both in highland and lowland areas (13). Remick noted that "considerable wind" usually accompanies lake snows (1). Leighly proposed that the air-water temperature contrast is related to the intensity of the lake's effect and subsequent snowfall (3). This idea has recently been reiterated by L. A. Hughes, who states: "The larger the air-water temperature difference, the greater the tendency toward heavy snow" (16). Remick, and McVehil and Peace suggest that at least a 5°F air-water temperature difference is necessary for lake snows to occur (1,2), but Johnson and Mook cite a 10°F difference as more likely to be a sufficient contrast (11). This paper will focus on the roles of windspeed and the air-water temperature contrast as factors in the variation of the distance inland of greatest snowfall for a given lake snow.

Methods

An attempt has already been made to verify the first hypothesis of this study (that the distance inland of maximum snowfall and windspeed during the snowfall are positively related in western lower Michigan). F. L. Charton in a Michigan State University Department of Geography Field Problem (17) attempted to correlate these phenomena by using actual wind and snowfall data from Michigan weather stations. He found no correlation between windspeed and the distance inland of greatest snowfall. However, a major problem of his approach was that he used actual wind data, which are available from only two stations in the area--Grand Rapids and Traverse City. Thus, the influences of those particular station sites on the wind data could not be controlled, nor could generalizations be made about wind conditions between the two stations, which are nearly 130 miles apart. Moreover, Charton's approach used only 8 samples from each of 3 latitudinal zones to obtain snowfall data. Additionally, his snowfall data were restricted to the 7 stations in his study area which record actual snowfall: Eau Claire, Grand Rapids, Ludington, Muskegon, Newaygo, Manistee, and Traverse City. The limitations of this sampling technique might have been severe enough to obscure any correlations. Thus, it was felt that a substantially different approach might show a

positive correlation between windspeed and distance inland of heaviest snowfall.

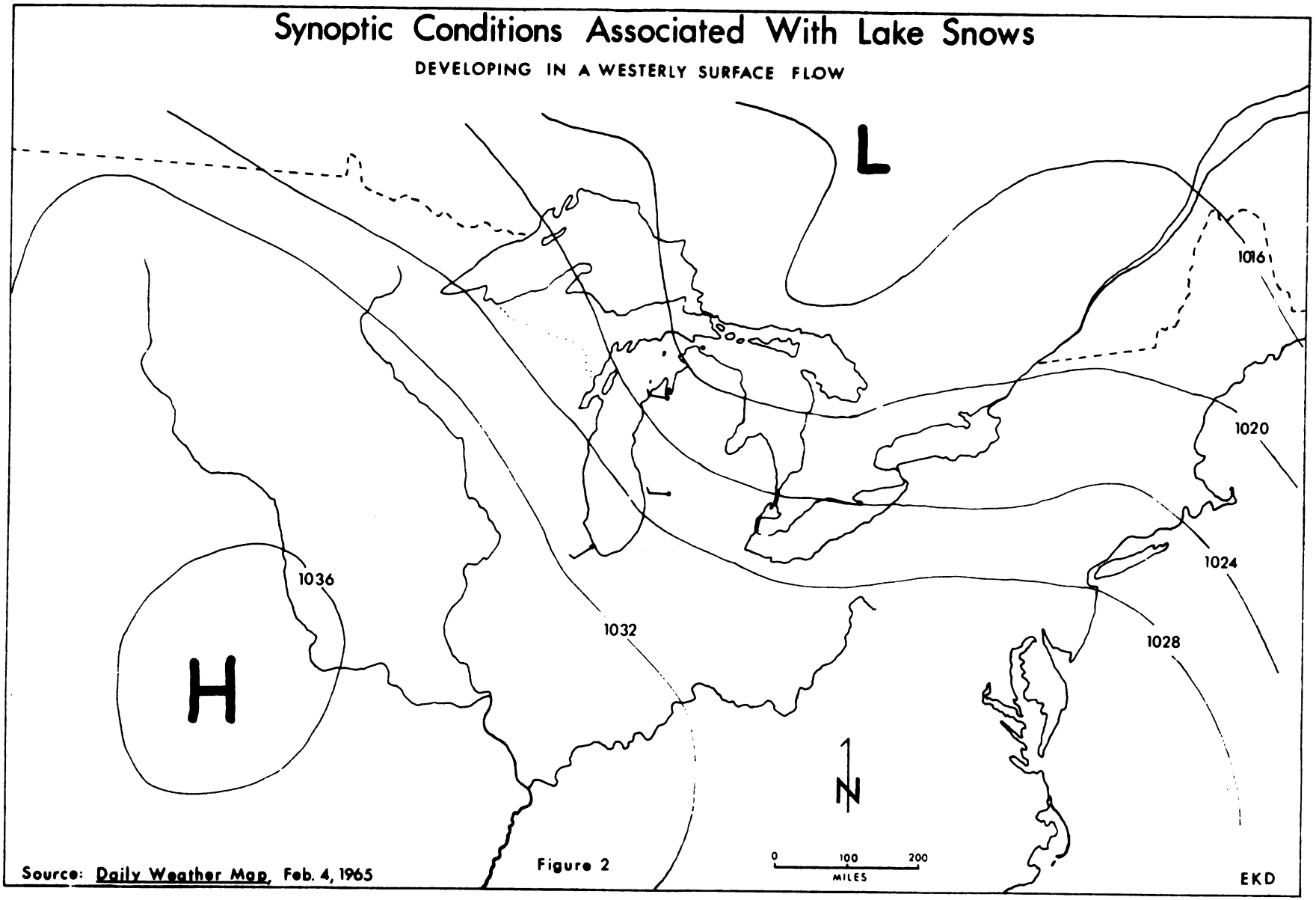
Selection of Lake Snow Days

The first step in the attempt to verify the hypothesis of this study involved the selection of appropriate lake snow events for further investigation. The initial procedure followed was not unlike that employed by Remick in his early study of lake-effect snows (1). A ten-year time period from November 1960 to February 1970 was chosen to allow selection of an adequate number of lake snow days to permit quantitative analysis of the data. Although lake snows may occur in Michigan from October to May, the most frequent occurrences under westerly wind conditions tend to fall between November and February (15).

Likely lake snow days were selected on the basis of synoptic weather patterns as interpreted from the U.S. Weather Bureau's Daily Weather Map (18). Days were chosen according to the existence of conditions described by Eichenlaub as likely to produce lake-effect snows: a low to the east of the Great Lakes and a high to the west, causing a flow of arctic air across Lake Michigan (14). Lake snows occurring under northerly or northwesterly wind conditions were excluded so that the distances inland from the lakeshore to recording stations along air trajectories could be kept reasonably constant. Thus, only likely lake snow days with westerly winds were selected. This selection

was accomplished by using isolines of barometric pressure as a guide. Under westerly wind conditions, surface barometric pressure declines northward between Chicago and Traverse City so that isobars on the weather map are plotted roughly normal to an imaginary line connecting the two cities. Winds across the lake flow at approximately a 45° right deflection from the isobars. Therefore, in order to eliminate northwesterly or northly wind conditions from the selection of lake snow days, only those days when the isobars crossed Lake Michigan at a 45° angle or less to the southern boundary of Michigan (which follows an east-west line) were chosen. Figure 2 on page 13 illustrates the synoptic weather conditions under which lake snows occur, while Figure 3 on page 14 illustrates the alignment of isobars across Lake Michigan under westerly wind conditions. In addition, only those days when temperatures of 32°F or lower were listed at Grand Rapids were chosen in order to help insure that snow rather than another form of precipitation would occur.

Using these criteria, 199 "likely" lake snow days were found in the ten-year study period. 23 days in November of 1960 and 16 days in January of 1961 were excluded from consideration when the Traverse City station did not report on the Daily Weather Map. As the barometric pressure readings from Chicago and Traverse City were used to infer windspeed, the omission of these data from the


Synoptic Conditions Associated With Lake Snows
DEVELOPING IN A WESTERLY SURFACE FLOW
L
H
1036
1032
1028
1024
1020
1016
N
0 100 200
MILES
Source: Daily Weather Map, Feb. 4, 1965
Figure 2
EKD

Isobaric Pattern Over Lake Michigan

ASSOCIATED WITH A WESTERLY SURFACE WIND

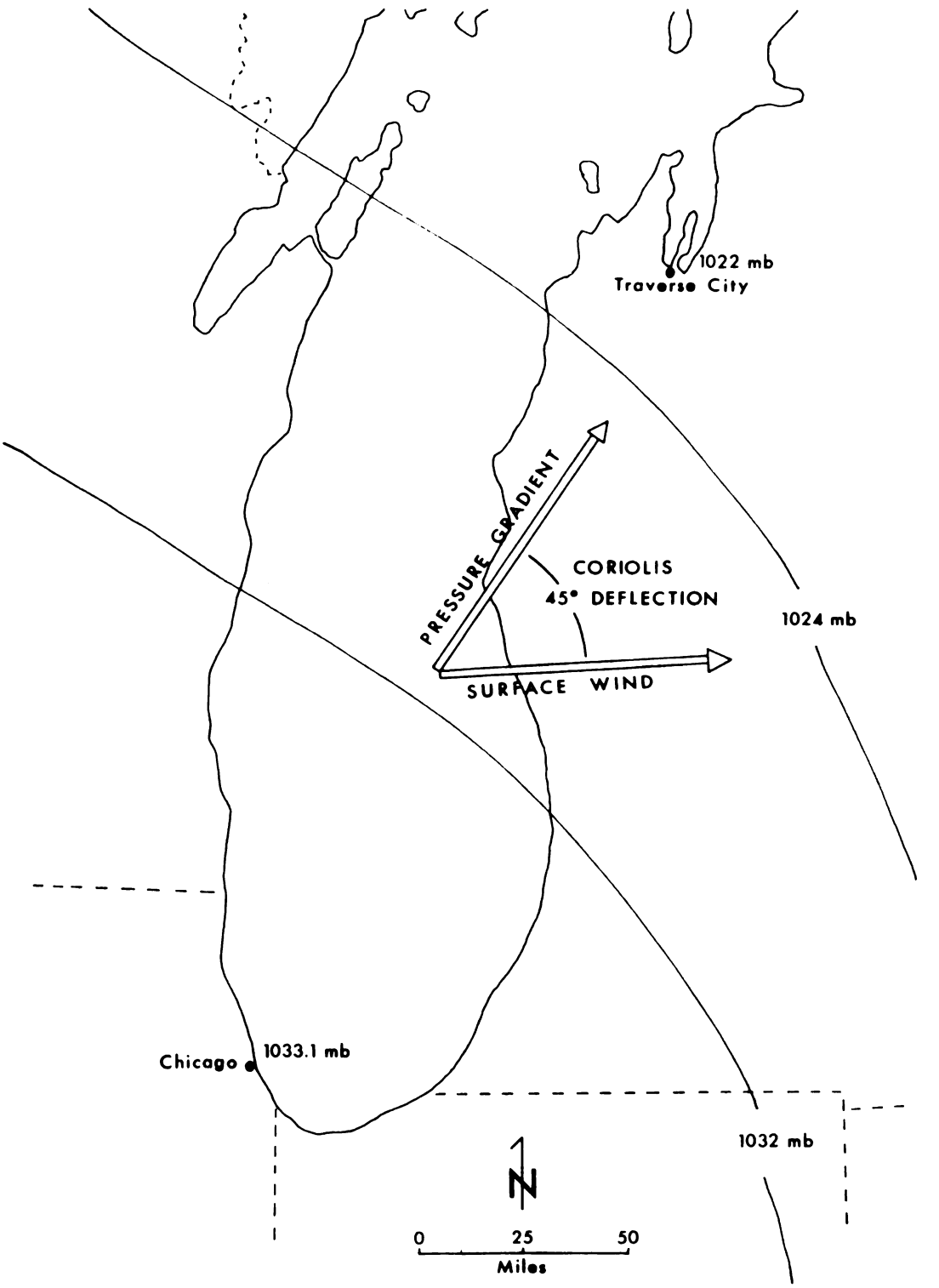


Figure 3

EKD

weather maps caused those days to be excluded from selection. However, synoptic conditions indicated that only 4 of the 39 omitted days were likely lake snow days.

The 199 "likely" days originally selected were then verified as actual lake snow days. This verification was accomplished by checking the recorded snowfall at 9 Michigan and 3 Wisconsin stations: Eau Claire, South Haven, Grand Rapids, Ludington, Muskegon, Newaygo, Manistee, Pellston, and Traverse City, Michigan; Manitowac, Milwaukee, and Green Bay, Wisconsin. These snowfall records were taken from Climatological Data (19). In order to eliminate snowfall caused by frontal conditions, any day on which .1" or more snow fell at any of the 3 Wisconsin stations was excluded. Moreover, at least .1" of snow must have been recorded at at least one of the 9 Michigan stations in order for a "likely" lake snow day to have been accepted as verified. These criteria resulted in rejecting 77 of the "likely" days, so that 122 lake snow days were accepted for the ten-year study period. It is interesting to note that the frequency of lake snows--as defined by these severely restrictive criteria--varied greatly, from as many as 8 in January, 1966 to none in November of 1960, 1961, 1962, 1963, and December of 1962. Seasonal (November-February) frequencies varied from 18 in 1967-68 to 6 in 1960-61, with about 12 per season on the average. The 122 lake snow days used in this study are listed in Table 2 on page 38.

Windspeed Data

Due to the lack of a suitable number of windspeed and direction recording stations in western lower Michigan, an indirect attempt was made to measure surface windspeed. Under the synoptic weather pattern characterized by an anticyclone to the west of the Great Lakes and a cyclone to the east, a barometric pressure difference exists between Chicago and Traverse City--as illustrated in Figure 3 on page 14. Under these conditions the barometric pressure is higher at Chicago than at Traverse City, although the differences between the pressure readings may vary greatly from day to day. Since the actual wind is deflected about 45° clockwise from the pressure gradient, these conditions cause westerly winds over western lower Michigan. As the pressure gradient steepens (i.e., the pressure difference between Chicago and Traverse City increases), the westerly winds generated by the pressure difference would increase in velocity. Thus, the pressure difference between Chicago and Traverse City can be used as an index of windspeed in the western lower part of the state.

While this method admittedly produces only a rough measure of windspeed, one that may be subject to local variation, it seems a preferable alternative to using the daily wind data from only Grand Rapids and Traverse City to compute an average windspeed. Although no attempt was

made to correlate actual windspeeds with the magnitude of recorded pressure differences, it seems likely that as the pressure difference increases the windspeed would increase. Thus, large pressure differences could be assumed to correlate with higher windspeeds, while smaller pressure differences would produce lower windspeeds.

Accordingly, barometric pressure readings for Chicago and Traverse City were taken from the daily weather maps. The pressure differences between Chicago and Traverse City were computed for the 122 verified lake snow days. The range of differences (in millibars) varied from 1.3 to 15.4, with the mean difference being 7.0 (millibars).

Snowfall Data

In order to record the distances inland of greatest snowfall a series of west-east transects were selected, following Changnon's reporting of Eichenlaub's analysis of annual lake snow accumulations in Michigan (15). The four transects and their stations are shown in Figure 4 on page 18.* Each transect consists of a series of weather stations, located on a general west-east line, located at distances of from less than 1 mile up to 83 miles away from Lake Michigan. The transects and distances (in miles) of each station from Lake Michigan (20) are as follows:

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*Specific station names are listed in Table 3 on page 39.

WEATHER STATION TRANSECTS

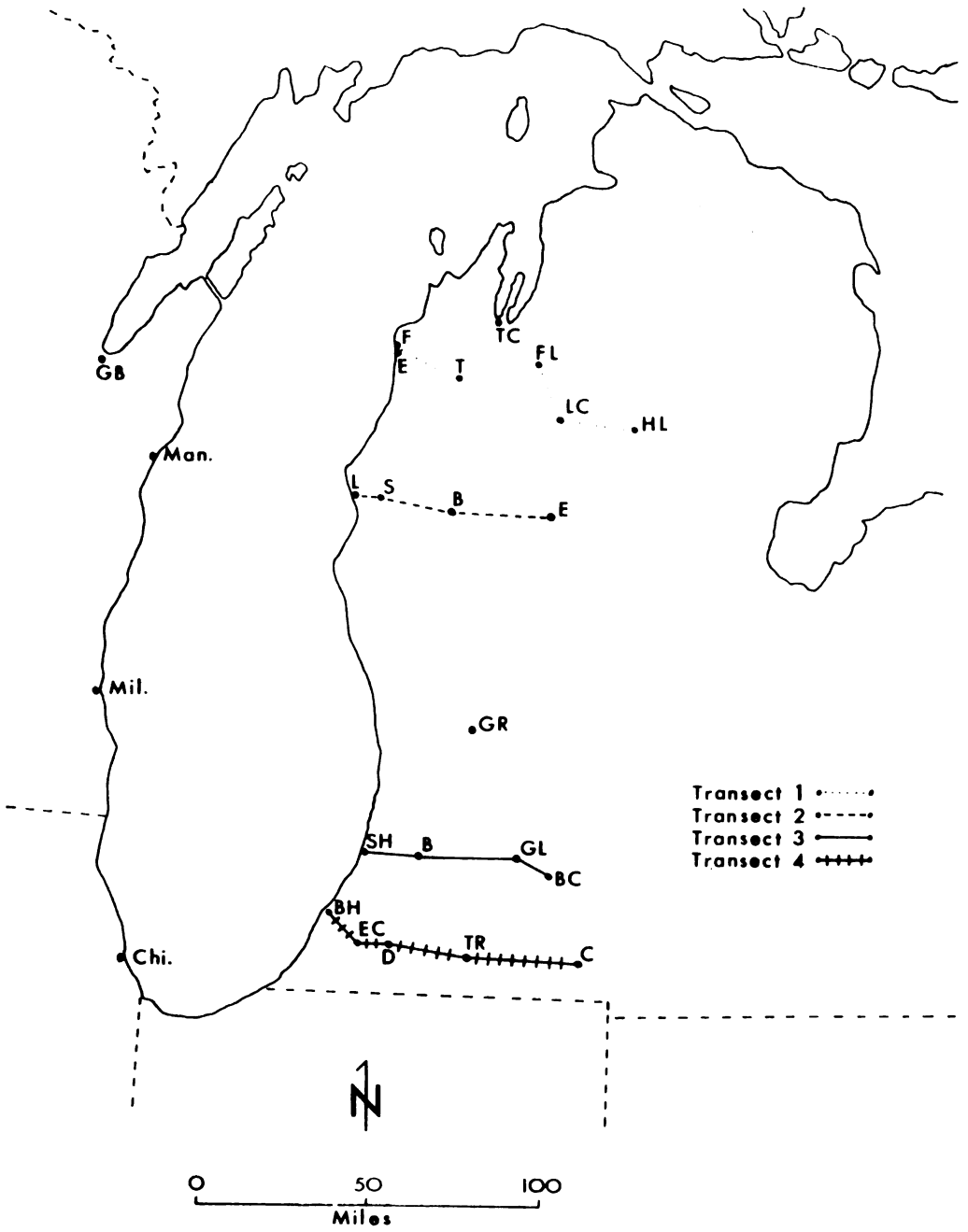

Figure 4

EKD

Transect #1: Frankfort (station located at Elberta prior to November, 1964)--1, Thompsonville--19, Fife Lake--42, Lake City--54, Houghton Lake--72.

Transect #2: Ludington--2, Scottville--9, Baldwin--36, Evart--75.

Transect #3: South Haven--1, Bloomingdale--18, Gull Lake--45, Battle Creek--58.

Transect #4: Benton Harbor--2, Eau Claire--14, Dowagiac--24, Three Rivers--49, Coldwater--83.

Because not all of these stations record actual snowfall, the daily precipitation figures were used for each station as listed in Climatological Data (19). This method followed Changnon's precedent in using precipitation (melted water) to compute snowfall amounts for western Michigan and eastern Wisconsin due to the lack of a sufficient number of snowfall-recording stations and to the variable snow-to-water content of lake snows (12). The use of these figures and the general west-east alignment of the station transects permitted an approximation of the distances inland of maximum snowfall (within each transect's area) for each lake snow day. However, during all or part of the study period, several stations (Fife Lake, Lake City, Ludington, Scottville, Baldwin, Bloomingdale, and Three Rivers) recorded their precipitation in the forenoon (as opposed to the more common afternoon recording). Accordingly, their readings were taken from the following day's listings in order to compensate for differences in recording times.

In addition, various stations (Fife Lake, Houghton Lake, Ludington, Baldwin, Bloomingdale, and Dowagiac) did not always report, occasionally missing an entire season's readings (as did Bloomingdale from November 1966 to February 1967). For those cases the appropriate lake snow days were omitted from those transects as it was not possible to determine which station in the transect had the greatest snowfall. Nevertheless, a sufficient number of lake snow events was recorded for each transect to permit statistical analysis (21). Precipitation accounts for 101 lake snow days were recorded for Transect #1, 80 for Transect #2, 83 for Transect #3, and 81 for Transect #4--345 in all.

Temperature Data

The second hypothesis (that the air-water temperature contrast is inversely related to the distance inland of maximum snowfall) required additional data gathering. In order to determine the approximate temperature of air passing over Lake Michigan, temperature data from Wisconsin stations were taken from Climatological Data for the appropriate dates. An average temperature was computed by first averaging the daily maximum and minimum temperatures for Green Bay and Milwaukee, and then averaging these two figures together. Thus, a "Wisconsin average" was achieved which represented the air temperature during the lake snow days. These ranged from a high of 34.25°F to a low of -15.25°F during the days under consideration.

The water temperature of Lake Michigan was computed by taking the average of the monthly average surface water temperatures at the center of the lake during the months of November to February (16). This procedure produced a surprisingly high 40.75°F for the average water temperature--a figure which, however, corresponds to Hughes' estimated 39°F average winter water temperature in Lake Michigan (16).

The air-water temperature contrast was then computed by subtracting the average air temperatures from the average water temperature for each lake snow day. The air-water temperature differences for various lake snow days varied from a minimum of 6.5°F difference to a maximum of 54.75°F difference, with 32°F the mean air-water temperature contrast.

Preliminary Test of the Windspeed Hypothesis

At this point a preliminary test of the windspeed hypothesis was attempted. It was felt that if this hypothesis were correct, then a greater barometric pressure difference (the measure of windspeed) would be positively related to maximum snowfall at a greater distance inland from the lake. The windspeed index was divided into quarters, with ranges in the pressure differences of 1.3-4.8, 4.9-8.4, 8.5-11.8, and 11.9-15.4 millibars. The lowest group (1.3-4.8 millibars pressure difference) contained 31 of the verified lake snow days. This group was used to represent the "low" windspeed days. The highest quarter contained

only 3 lake snow days; therefore, the second highest group (8.5-11.8 millibars pressure difference, containing 26 days) was added, forming the "high" windspeed group.

The Michigan stations used for verifying lake snows were divided into "lakeshore" and "inland" groups. The former comprised the stations at South Haven, Ludington, Muskegon, Manistee, and Traverse City. The latter set contained Eau Claire, Grand Rapids, Newaygo, and Pellston. The recorded snowfalls for each of those stations on the designated days were compared to determine if the heaviest snowfalls generally occurred inland or at the lakeshore. Some measure of individual judgement was used as not all cases were clearly inland or lakeshore maxima.

For the low windspeed group (31 days total), 14 days were clearly days when the maximum snowfall (as determined by this limited station network) fell at the lakeshore. 5 other days were "probably" lakeshore maxima. Thus, this analysis seemed to uphold the hypothesis as with low windspeeds lakeshore maxima were more common (19: 12). For the high windspeed groups (29 days total), 9 days were lakeshore maxima with 2 days as "probables." 10 days were inland maxima with 8 days as "probables." For higher windspeeds inland maxima occurred 18 times vs. 11 times for lakeshore maxima. These results also seemed to confirm the hypothesis.

Graphic Analysis

An analysis of the data was carried out using scattergrams to graphically portray the relationships of windspeed and the air-water temperature contrast to the distance inland of greatest snowfall. Scattergrams plotting the barometric pressure differences between Chicago and Traverse City (the index of windspeed) vs. the distance inland of maximum snowfall were constructed for each transect and for the transects combined. Figure 5 on page 24 illustrates the results for Transect #3. The mean windspeed index at each station location was computed. If the windspeed hypothesis were true, then the mean of the windspeed index might be expected to successively increase at each inland station. This does appear to occur for the data for Transect #3; however, the patterns were not consistent for the other transects or for the combined data.

A similar analysis was made for the second hypothesis. The air-water temperature difference was plotted against the distance inland of greatest snowfall for Transects #1 and #3. The mean temperature contrasts were computed for each station. Figure 6 on page 25 illustrates the results for Transect #1. The pattern here actually suggests that the inverse of the hypothesis would be true; however, the relationship is not clearly identifiable. The pattern for Transect #3 was similarly inconclusive.

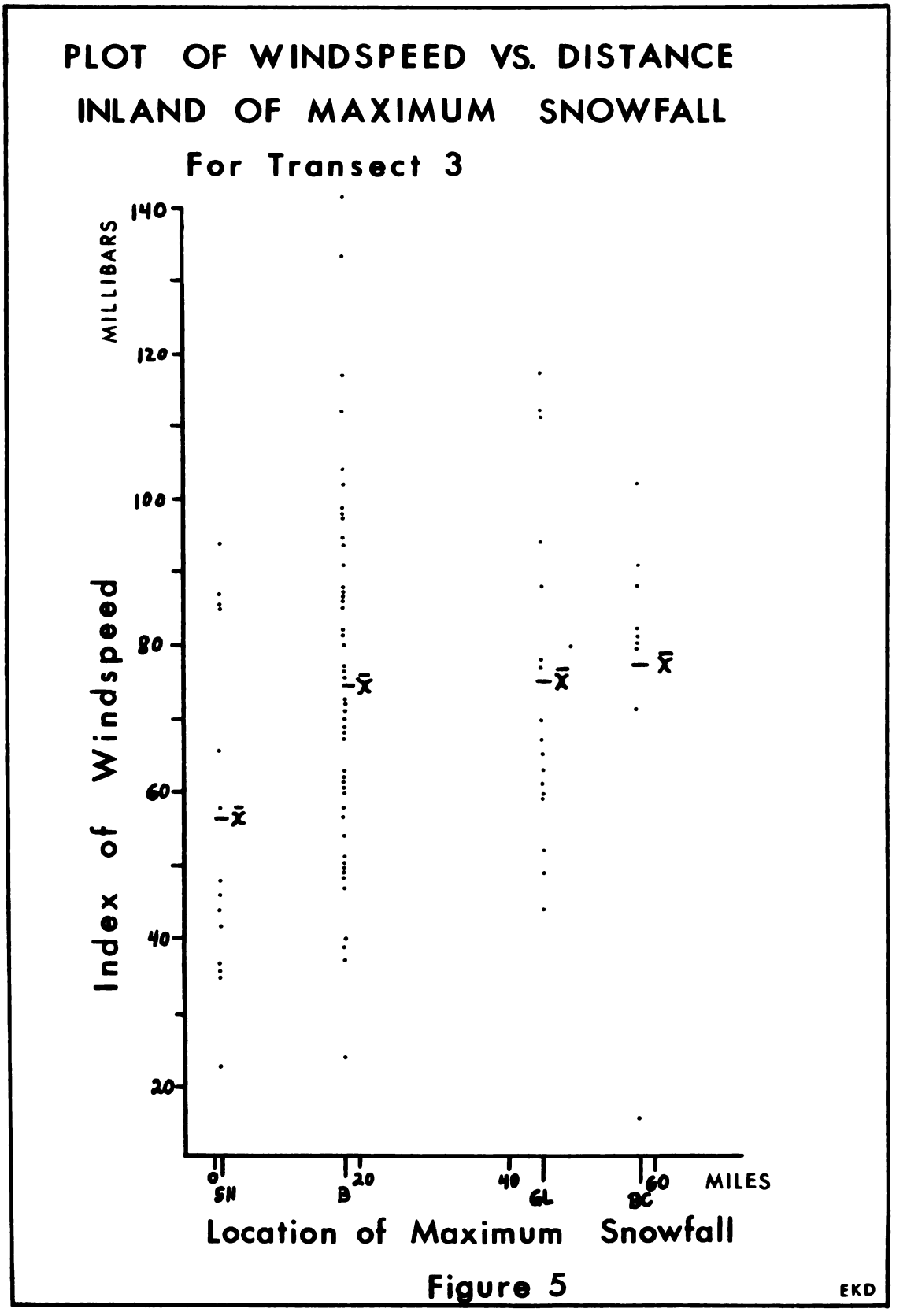
PLOT OF WINDSPEED VS. DISTANCE
INLAND OF MAXIMUM SNOWFALL
For Transect 3
MILLIBARS
140
120
100
80
60
40
20
Index of Windspeed
0
SH
20
B
40
GL
60
BC
MILES
Location of Maximum Snowfall
EKD

Figure 5

PLOT OF AIR-WATER TEMPERATURE CONTRAST VS. DISTANCE INLAND OF MAXIMUM SNOW

For Transect 1

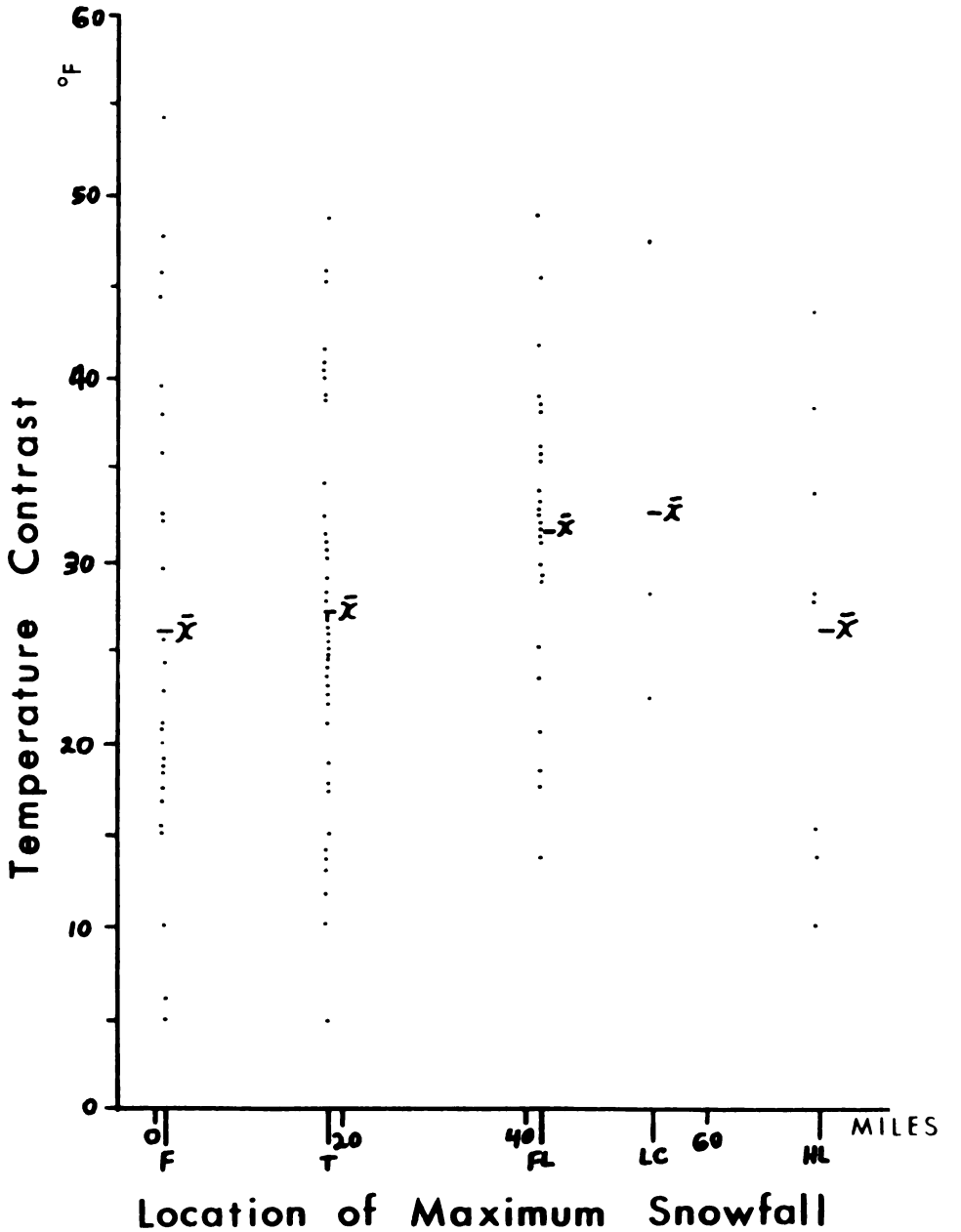

Figure 6

EKD

Computer Analysis

The uncertain results of the preliminary test of the windspeed hypothesis and of the graphic analyses indicated that a more rigorous quantitative analysis was appropriate. Accordingly, the data was analyzed using the package program LSDEL on Michigan State University's IBM 3600 computer. This program computes the simple correlations (r) of the input variables and performs an analysis of variance with regression, computing multiple correlation coefficients (R^2) and residuals from regression. The program was run for the data for each transect separately and for the combined data as well. The results are shown in Table 1 on page 27. The results of the simple correlations were then tested for their significance at the .05 level, using the F test ($F = \frac{r^2}{1-r^2} (N-2)$) (21).

The simple correlations (r) indicate that the windspeed hypothesis does not appear to be true. Correlation (r) values of only -.03 to .18 were computed, none of which were significant to the .05 level (i.e., they have a greater than 5% probability of being chance correlations). On the other hand, the temperature contrast hypothesis seems to be supported by the results. Correlations from -.22 to -.45 were computed, all of which are significant to the .05 level (i.e., are not likely to be chance correlations). However, the correlations between the windspeed and

TABLE 1

RESULTS OF COMPUTER ANALYSIS

Simple Correlations (r)

	Dist	Wndspd
Transect #1 (N=101)		
Wndspd	.07	
Temp	-.22*	.15
Transect #2 (N=80)		
Wndspd	.03	
Temp	-.45*	.25*
Transect #3 (N=83)		
Wndspd	.18	
Temp	-.28*	.12
Transect #4 (N=81)		
Wndspd	-.03	
Temp	-.26*	.17
Combined Data (N=345)		
Wndspd	.06	
Temp	-.30*	.17*

Dist: the distance inland of maximum snowfall
Temp: the air-water temperature contrast
Wndspd: the index of windspeed
*: significant to .05 level

Multiple Correlations (R^2)

	R^2	$R^{2\#}$
Transect #1	.06	.05
Transect #2	.22	.20
Transect #3	.13	¢
Transect #4	.07	.07
Combined Data	.10	¢
Combined Data**	.10	.09

**: significant to .01 level
#: after deletions
¢: no deletions

temperature variables ranged from .12 to .25, being significant to the .05 level in two instances. This finding raises the possibility that there is some interaction between the variables so that as windspeed increases the air-water temperature contrast increases (or vice versa). This seems likely as strong winds in the study area are often accompanied by arctic outbursts of frigid air.

The multiple correlations were computed at the .05 level of significance. R^2 values for the individual transects (before deletion) varied from .06 to .22; after deleting the windspeed variable, the range of values dropped only slightly to .05 and .20. Similarly, the combined data (before deletion) had an R^2 value of .10; after deletion of the windspeed variable (accomplished only at the .01 significance level), the R^2 value was .09. Thus, the minor decrease of the R^2 values after deleting the windspeed variable confirms the conclusion that the windspeed hypothesis is not supported by this analysis. The combined data run (.01 level) indicates that the air-water temperature contrast hypothesis explains about 9% of the variance of the distance inland of maximum snowfall. The deletion of the windspeed variable (combined data run) at the .01 significance level indicates that windspeed may explain 1% of the variance of the distance inland of maximum snowfall (with a 5% probability of error).

Conclusions

The analysis of the computer results indicates that the first hypothesis is probably false, that is, increasing windspeed does not cause the distance inland of greatest snowfall to increase. However, the results from Transect #3, where the r value was .18 (higher than for the other transects) for the simple correlation of windspeed and distance inland of greatest snowfall, are puzzling. The graphic analysis of this transect (Figure 5, page 24) also differed from those of the other transects. That orographic effects would be the cause of a positive correlation between windspeed and the distance inland of maximum snowfall seems doubtful. Although there is a group of hills in the Bloomingdale area with a 100 foot rise in elevation between South Haven and Bloomingdale, an even greater change of elevation occurs between Benton Harbor and Eau Claire on Transect #4, where neither the graphic nor the computer analyses are similar to those of Transect #3. The profile of annual snowfall accumulation along Transect #3 (as computed by Changnon (15)) is similar to the profiles of the other transects.

One factor which distinguishes Transect #3 from the others is that Lake Michigan is wider at that point than at the others. The lake's width at South Haven is about 79 miles, as opposed to 68 miles at Benton Harbor (Transect #4) and 62 miles each at Transects #1 and #2.

Whether this additional width at Transect #3 (which would increase the fetch of the air stream's trajectory over water at that point) is sufficient to meaningfully alter the lake snow patterns in that area needs further study. This writer can offer no other clues as to why the windspeed correlation for Transect #3 appears to be different from those of the other transects. It may be, that as graphic interpretaion is relatively more subjective than statistical analysis (22) and as the .18 r value of the windspeed variable is not significant to the .05 level, the anomalous correlation for Transect #3 is more apparent than real (in a statistical sense).

By contrast, the second hypothesis appears to be true, though with limited powers of explanation. Thus, as the air-water temperature contrast increases, the distance inland of greatest snowfall accumulation decreases. A greater air-water temperature contrast would result in the lake's giving relatively more heat and moisture to the passing airstream. Greater air mass instability would develop which possibly could be "triggered" into producing snowfall more quickly by the orographic effects of the western Michigan lakeshore.

The failure of this study to achieve a high degree of explanation seems to demonstrate that the areal variation in the maximum snowfall accumulation for any given lake snow is a response to multiple factors. Windspeed and

air-water temperature contrasts are only two of these factors, which accounted for only 10% (at the .05 significance level) of the variance in this study. It is fully recognized that other factors may be operative as well. Among these might be: local relief, specific site exposure, the length of the fetch of the airstream over water, condensation nuclei sizes and concentrations, the presence of snow already on the ground, degree of ice cover on the lake, and conditions aloft (long and short wave patterns, air temperature, windspeeds, etc.) (23,24,5).

The paucity of readily available data for many of these variables prohibits their investigation by use of data listed in standard documents, e.g., Climatiological Data. This lack of data is particularly true of mesoscale studies, as most weather stations record only temperature and precipitation. Thus, an "index" of windspeed had to be constructed for this investigation as suitable windspeed data are not available for the study area. The frequent failure of stations to record their findings additionally limited the data analysis. Moreover, the numerous possible errors in observing and recording data (to which snowfall observations are especially susceptible) cast further doubt on the reliability of these findings (1). Additional problems are created by the variation from station to station in the times of recording the data.

It is possible that extending the time period of the study (e.g., by including March in the "lake snow season" or by using data from a 20 year period) might have altered the findings. However, there are no strong arguments to support this suggestion. On the other hand, some other approaches in methodology might prove fruitful, were the study to be repeated. For example, the criteria used for designating lake snow days in this study are quite restrictive. A listing of .1" or more snowfall at any of the 3 Wisconsin "control" stations was sufficient to reject that date from the "likely" lake snow days list. Fifty-six of the 77 "likely" days that were rejected were omitted for this reason. Under the synoptic conditions characteristic of important lake snow events there is widespread air instability; thus, snowfall of .1" or more in Wisconsin might not be due to frontal conditions (the elimination of which possibility was the purpose of the .1" limit) but rather do general instability inherent in the airmass. Accordingly, some (or all) of the days rejected due to excessive snowfall in Wisconsin might have been added to the lake snow days list, increasing the "N" for each transect by 50% or more. Whether this would have made a difference in the conclusions of this study is problematical and only a second attempt at this analysis using new criteria would answer this question.

A similar question arises from the inclusion in this study of all lake snow events occurring from November to February in the same groups for statistical inference. There may be differences in the lake snows which occur early in the season (e.g., November to December) when both the lake and airstreams are warmer from those which occur later in winter. An analysis of 15 residuals (observations whose regression values were greater than one standard deviation from the mean) for Transect #3 indicated that 12 of these observations occurred in November or December. But a sample of 50 similar residuals taken from the combined data computer run (.05 level) had only 27 of the 50 observations occurring in November or December. Perhaps a different sort of analysis--one which distinguished between early and late season lake snows--would have generated different results. However, this sort of approach to understanding, i.e., an empirical-inductive approach via statistical inference, can easily degenerate into a numbers game, with numerous possibilities in statistical tests, groupings of data, operational definitions, etc. It may be that a better model (however informal) of the processes of lake snows is needed before empirical proofs of hypotheses about those processes should be attempted.

The results of this study seem to indicate that research of this type, involving complex mesoscale phenomena, cannot be adequately completed using readily available

climatological data alone. A possible alternative would be to pursue specific "case studies" of a number of lake snow events, using on-site recording instruments to attempt to determine the essential factors in the areal variation of the event. Ideally, such instruments ought to be capable of measuring the various factors (indicated above) which may affect the snowfall occurrence. In such a study, a broader scope of investigation would be appropriate, focusing on causes of variation in water content, intensity, and duration of the snowfall as well as its spatial locus. Perhaps the selection of a study area in western New York, where well-defined "corridors" subject to frequent lake snows are known to exist, would be desirable. On the other hand, locations in western Michigan, where orographic effects are less pronounced and a different type of lake snow usually occurs, might be equally suitable.

Studies such as the one reported in this paper are properly classified as synoptic climatology. Their purpose is to further the understanding of local and regional climates with reference to large scale circulation. The investigation of these mesoscale phenomena is crucial to the development of "basic mechanisms of climatic differentiation" (25). The difficulties of such research, with its need to identify and analyze the multivariate factors of what are essentially <u>systems</u> of phenomena, should serve as a challenge for further study.

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APPENDIX

TABLE 2

VERIFIED LAKE SNOW DAYS

Season	Dates	
1969-70	Nov. 1969:	12, 20, 30
	Dec. 1969:	19, 20
	Jan. 1970:	6, 10, 19
	Feb. 1970:	5, 13, 20, 21
1968-69	Nov. 1968:	19, 20
	Dec. 1968:	6, 20, 24, 31
	Jan. 1969:	1, 4, 7, 10, 11, 12, 26
	Feb. 1969:	4, 18, 24
1967-68	Nov. 1967:	4, 5, 6, 14, 27, 28
	Dec. 1967:	15, 22, 23, 26, 27
	Jan. 1968:	4
	Feb. 1968:	9, 12, 15, 17, 19, 22
1966-67	Nov. 1966:	4
	Dec. 1966:	2, 3, 26, 27, 31
	Jan. 1967:	8, 17, 18
	Feb. 1967:	9, 12, 15, 17, 19, 22
1965-66	Nov. 1965:	14, 17, 28, 29
	Dec. 1965:	15, 17
	Jan. 1966:	3, 11, 17, 18, 19, 24, 28, 31
	Feb. 1966:	3, 4
1964-65	Nov. 1964:	21, 22, 29
	Dec. 1964:	8, 15, 22
	Jan. 1965:	20, 29, 30
	Feb. 1965:	2, 3, 4, 13, 26
1963-64	Dec. 1963:	14, 15, 16, 19, 22, 28, 30
	Jan. 1964:	10
	Feb. 1964:	8
1962-63	Jan. 1963:	13, 14, 15, 24, 28
	Feb. 1963:	14, 20, 22
1961-62	Dec. 1961:	7, 8, 13, 27, 30
	Jan. 1962:	8, 10, 16, 18, 23
	Feb. 1962:	7, 20
1960-61	Dec. 1960:	22, 23
	Jan. 1961:	22, 27, 29
	Feb. 1961:	19

TABLE 3

WEATHER STATION LIST

Michigan	Frankfort (Elberta), Thompsonville, Fife Lake, Lake City Exp. Farm, Houghton Lake
	Ludington, Scottville, Baldwin State Forest, Evart
	South Haven Exp. Farm, Bloomingdale, Gull Lake Biol. Sta., Battle Creek WBCK
	Benton Harbor Airport, Eau Claire, Dowagiac, Three Rivers, Coldwater State School
	Traverse City FAA Airport, Grand Rapids WB Airport
Wisconsin	Green Bay WB Airport, Milwaukee WB Airport, Manitowac
Illinois	Chicago WB Airport

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ABSTRACT

THE DIFFUSION OF TUBERCULOSIS IN AFRICA,
WITH PARTICULAR REFERENCE TO SOUTHERN AFRICA

By

Eugene Keith Dayton, Jr.

Using reports from medical journals and other library sources, a study was made of the diffusion of tuberculosis in Africa. Special emphasis was given to Southern Africa (all the continental states south of Zaire and Tanzania) as the processes of diffusion in that region could serve as a model for the processes elsewhere on the continent.

A brief discussion of the epidemiology of tuberculosis established the importance of environmental conditions in the spread of the disease. It was noted that crowding, malnutrition, stressful social and occupational conditions, and the presence of other disease complexes create conditions which may predispose tuberculosis infection and transmission. The course of the disease in susceptible populations was noted as acute manifestations of tuberculosis characteristically display a significantly different pattern of morbidity and mortality than do the chronic forms of the disease.

Eugene Keith Dayton, Jr.

That tuberculosis was relatively absent from the African interior prior to the European intrusions meant that most Africans were highly susceptible to tuberculosis infection. The role of development in Southern Africa was noted as a factor in the diffusion of the disease. The aggregation under stressful conditions of large numbers of Africans in close association with tuberculized Europeans and other non-Africans facilitated tuberculosis transmission. The South African mines were especially instrumental in this process. The migration of Africans between their homes and the mines and other foci of infection and the repatriation to their homes of tuberculous Africans helped spread the disease to rural areas deep in the interior.

The historical development of this diffusion was traced for Southern Africa, with concluding remarks noting that the processes of diffusion are continuing, that chronic forms are replacing the acute manifestations of the disease, and that the disease still represents a serious health problem for African nations. A brief appendix offers similar evidence for the introduction and diffusion of the disease elsewhere on the continent.

THE DIFFUSION OF TUBERCULOSIS IN AFRICA, WITH PARTICULAR REFERENCE TO SOUTHERN AFRICA

By

Eugene Keith Dayton, Jr.

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Introduction

Western (particularly European) inputs in Africa over the past century have had a profound impact on the peoples and landscape of that great continent. The development of transportation and communications facilities, the introduction of new governmental, social, educational, and medical services, and the creation of new industries and commercial enterprises have had varied effects, the pro's and con's of which are frequently argued. However, one of the Western inputs to the African scene which has not been as fully discussed is the introduction and diffusion of diseases as a consequence of the European intrusion into the continent.

This paper will discuss the spread of tuberculosis in southern Africa. Emphasis will be placed on the role of development in increasing and facilitating this spread. The purpose is to demonstrate how development, which includes urbanization, the establishment of commercial and industrial enterprises, and the creation of transportation-communication infrastructures, can also bring unexpected and unplanned results (e.g., the spread of tuberculosis) with serious and longlasting consequences for the peoples involved. It is intended to point out the need for a

more comprehensive awareness of the effects development can have on an area, so that further development planning can avoid some of the mistakes of the past.

Southern Africa (defined here as all the continental states south of Zaire and Tanzania) was chosen to illustrate this problem for three reasons. First, it will be shown that this area, especially South Africa, has been a major source for the diffusion of tuberculosis in Africa. Second, the processes of diffusion in southern Africa are typical of those which are operational elsewhere in the continent, and are therefore a representative example. Third, the spread of tuberculosis in southern Africa is somewhat better documented than in the rest of the continent and can accordingly be more accurately and reliably reported.

Clinical Manifestations and Treatment of Tuberculosis

Caused by an infection of the mycobacteria, M. tuberculosis, tuberculosis may be manifested in several forms. Pulmonary tuberculosis is most common, but bones and joints, the gastro-intestinal, lymphatic, cardiovascular, and nervous systems, and the skin and serous membranes may also be infected. The various types of infection have historically been called by different terms; thus, pulmonary forms were often known as consumption or phthisis, while other types were called scrofula, lupus,

Pott's disease (and others). In particularly severe cases, called miliary tuberculosis, the disease is characterized by a general body infection which is disseminated by the blood stream (1).

Among populations where the disease is endemic, chronic forms of tuberculosis predominate. Thus, individuals may suffer intermittent periods when the disease is active, deteriorating the affected organs and weakening their general condition, while at other times the disease may be completely arrested by the body's defense mechanisms. Many individuals with high resistance may be infected with out ever showing symptoms, or even knowing they have been infected. However, in populations with little inherited resistance to tuberculosis, or whose health conditions are weakened by poor diet, bad housing, overwork, and/or other disease complexes, acute forms of tuberculosis are more likely to occur. In these cases the disease causes rapid tissue degeneration and subsequent impairment of health, and death may occur within a few months after infection (2).

Various procedures, including rest cures and surgery, have been used to treat tuberculosis. At present chemotherapy is commonly used as it can effectively and cheaply destroy the infecting bacillus. Immunity can be acquired by innoculation with B.C.G. vaccine (with about 80% effectiveness) for those not already infected (3).

However, the millions who already are infected (and therefore unable to be immunized) plus the emergence of drug resistant strains of tuberculosis pose serious problems for control and treatment of the disease (4).

Epidemiology

Determining the epidemiology of tuberculosis in Africa is fraught with problems. To begin with, there is a question of the definition of "active cases"--what is the threshold between benign infection (very common) and active infection? Secondly, there is a problem of interpretation of various tests and measures of case rates. A whole range of procedures, including examination for manifest symptoms, X-rays, tuberculin tests (again, with various methods), and sputum smears, have been employed to diagnose the disease and to determine the presence of infection. Historically and spatially there has been little standardization of tuberculosis epidemiological procedures in Africa (3).

Data collection has been very scattered and sometimes unreliable on the continent. Surveys tend to be localized, and only intermittently carried out. Hospital case rates or notifications are unreliable indicators due to changing medical practices, numbers and distributions of hospitals and clinics, and rates of use by native populations (5). Continent-wide, data on rates of prevalence,

incidence, and mortality are very sketchy; nevertheless, compilations of available data have shown historically as well as presently, that the tuberculosis situation in Africa is very serious (3,6).

Despite problems with the data, various indicators will be used to substantiate these claims. The opinions of medical officers, hospital case rates, as well as more official countrywide rates will be used. The data from surveys will be employed as well, with tuberculin tests considered the most reliable of these indicators (5). Although some of these rates will be astonishingly high, overall they must be viewed as conservative. For example, tuberculin tests may be read as negative despite the presence of infection under several commonly occurring conditions: infection with certain diseases (including measles), presence of some drugs, severe tuberculosis or other mycobacteria infection, acute malnutrition, tuberculosis foci present but healed, and test given during the six weeks period between infection and sensitivity development (4). Similarly, hospital and other case rates often do not take into account those infected who do not seek medical help or who are located far from medical facilities (7). Thus, it is likely that the epidemiological data for Africa represents only a part of the actual situation.

Transmission Cycle

The major tuberculosis transmission cycle in Africa is person to person infection. This usually occurs via respiration of infected breath droplets. Coughing may "jettison M. tuberculosis-carrying droplets to a distance of five feet," and these droplets may remain suspended in the air for days (8,9). Subsequent respiration or ingestion of these droplets can lead to pulmonary, abdominal, or other tuberculosis forms. Infection can result from the respiration or ingestion of infected dust or food. Infection through skin abrasions may also occur (2).

Humans may acquire bovine or other animal forms of tuberculosis by drinking infected milk or milk products or eating infected meat. Close contact with diseased animals--as when sick cattle are kept in the same hut where people sleep--also creates conditions for animal to man transmission (10). But these types of tuberculosis are less common, probably accounting for less than 10% of all cases in Africa (9).

Ecology of Transmission

As with many other communicable and parasitic diseases, the overall environmental conditions in which a people live are of paramount importance in the development of tuberculosis in both the population and the individual. These environmental factors are typically interrelated. For example, genetically inherited resistance

to tuberculosis is associated with endemic tuberculosis conditions. Where tuberculosis is absent from a population, the people are unlikely to have the antibodies and defense mechanisms (e.g., fibrosis) which are crucial as a defense against the disease (4).

But there are several environmental factors which are independent of the presence or absence of tuberculosis which are also important. Diet is one of these; individuals (or populations) with inadequate diets (particularly important is an adequate protein intake) are more likely to develop active cases if infected. Housing conditions and population densities are another factor. As housing deteriorates (generally indicative of overall economic-social-public health deterioration) and crowding increases, the likelihood of being exposed to infection increases. Standards of sanitation and public health, and the presence of other disease complexes which can reduce resistance are also important.

Age and sex are relevant factors. Generally, adolescents are at greatest risk due to the physiological stresses experienced at that age and to their first becoming exposed to a wider environment at that time. Males are usually more affected than females, probably due to the male's greater exposure at work to possible infection, and due to the environmental conditions of some occupations (2,4). Psychological stress is a factor also.

Experiences which upset emotional well-being can be harmful to bodily defense mechanisms and thus contribute to the spread of the disease (11). Occupation has been correlated to tuberculosis rates as well; like housing, it can serve as an overall indicator of these environmental factors which influence the likelihood of infection and progress of the disease (9).

Introduction of Tuberculosis to Southern Africa

There is good evidence to show that tuberculosis was absent from the interior of Africa prior to the coming of the white man in the late 19th and early 20th centuries. In 1857 Livingstone noted that "tuberculosis did not exist" in the interior (4). Dr. William Budd wrote in 1867, "In the interior, where intercourse with Europeans has been limited to casual contact...there is reason to believe that phthisis does not exist" (12). In 1921 Dr. Mitchell in South Africa reported that "all records go to show that fifty or sixty years ago the disease was comparatively rare, both amongst the Europeans and the Native inhabitants of the subcontinent" (13). MacVicar called tuberculosis "almost unknown" in 1909 in the interior of South Africa (14).

Even in the early part of this century the disease was not known in parts of the interior. In Botswana medical officers did not diagnose tuberculosis cases until the 1920's (15). This absence of tuberculosis from

interior regions must have been due to the relative isolation of the various ethnic groups prior to the European invasion. This isolation kept tuberculosis from spreading from the Arab and Egyptian areas to peoples elsewhere in the continent (16). Similarly, this isolation kept the disease from spreading from the coastal forts, where Europeans had been established for many years, to the peoples of the interior (12). Isolation can be so effective a block to the transmission of the disease that as late as 1957 some areas of Namibia (South-West Africa) which have no contact with the sea have reported only rare cases of pulmonary tuberculosis (17).

However, this isolation has meant that when Africans have come into contact with tuberculized peoples they have suffered high rates of morbidity and mortality due in part to their lack of inherited resistance to the disease. Dr. Budd noted this in the mid-19th century (12). The clinical manifestations in susceptible populations was described in 1920 as an "absence of fibrotic reaction, tendency to rapid generalization, and a speedy course towards a fatal termination" (14). Although over time acute cases tend to be replaced by chronic ones, the cases common in Zambia and Rhodesia in the early 1960's were still "advanced and destructive" (7).

The following examples illustrate how serious tuberculosis can be in susceptible populations. In 1803

and again in 1810 the British transported three to four thousand black Africans from Mozambique to serve in the army in Ceylon. By 1820 only 440 of them were still alive; most of the others had died from tuberculosis. 9,000 blacks taken from South Africa by the Dutch had a similar tuberculosis mortality (18). In France in 1917-18 British and black African soldiers, living under similar conditions of food, housing, and discipline, had 5.7% and 56% tuberculosis death rates respectively. More tuberculosis deaths occurred in a few African companies than in the whole British army in France (14).

But what were the sources of tuberculosis infection in southern Africa? It seems that these were of two types: disseminated sources and localized foci of infection. Of the former type, returning black soldiers and auxiliaries would have been one source of infection to other Africans. One observer noted an increase of tuberculosis among the Basutos after men returned from service as auxiliaries in the Boer War (19). Certainly some of those who served in France must also have brought tuberculosis back with them. Another scattered source of infection would have been the Europeans sick with tuberculosis who went to southern Africa to seek a healthier climate. The Annual Health Report of the Cape Colony for 1896 warned against the dangers of tuberculosis spreading due to these sources of infection (13). Similarly, Rhodesia was used as a "haven of refuge

for the phthisical" until laws were enacted to prevent the continued immigration of tuberculous Europeans (20).

But undoubtedly the more important sources of infection were various places and institutions where the risks of infection were significantly great to warrant calling them foci of infection. These would include missions, schools, railways, prisons, seaports, and the mines--places where population densities and living conditions would be favorable to the transmission of tuberculosis (4). The mines in particular were the major foci of infection in southern Africa. There, Africans came into close contact under ideal conditions for disease transmission with British and Chinese miners who were highly infected with tuberculosis.

In the late 19th century thousands of Europeans immigrated to South Africa to work on the diamond, gold, and coal mines. Perhaps 30% of them were infected with "Miner's Phthisis" (21). A shortage of African labor forced the importation of 50,000 Chinese coolies in 1905 who were also highly tuberculized. By 1908, 19,000 Europeans, 12,000 Chinese, and 150,000 Africans were working on the Rand gold mines alone, developing there a large infector pool which served as the primary basis for the spread of tuberculosis to other parts of southern Africa (22).

Conditions Facilitating Transmission in the Mines

As the major foci of tuberculosis infection in southern Africa, the South African mines need to be discussed further to point out the conditions at the mines which put the miners working there at great risk of being infected with tuberculosis. Certainly one of these conditions worth considering was the sheer amount of work required of the miners. The normal work day began at 4:30 AM and lasted until 3:00 or 4:00 PM, with only Sundays off. It was very hard work, requiring strenuous efforts. The environment in the mines was demanding. Temperatures of 85-90°F were common, with water frequently sprayed to keep down the dust, thus maintaining high relative humidity conditions. This created ideal conditions for the transmission of tuberculosis (23).

The high amounts of silica dust in the mines tended to cause the development of lung lesions which are predispositional to tuberculosis (24). The dust can have malign influences even on quiescent or apparently healed lesions so that inactive infections can become active again (4). These dust conditions led mine medical officers to conclude in 1921: "The association of tuberculosis with silicosis is, of course, very common, especially amongst the natives, and may occur at any stage of the latter disease. At the present time it is practically true to say that those who die from the disease die from this complication" (25).

Another significant condition at the mines was the miners' diet. It has been estimated that miners need up to 4,700 calories per day (9). This amount of calories may have been met in the typical miner's diet as cited by Gelfand: "3 lbs. of maize, ropoka, or mabele meal per day, salt, a little canned meat occasionally, and sometimes a ration of beans, groundnuts and pumpkins" (26). However, no herbs, green vegetables or fresh meat were included; thus essential proteins, vitamins, and other nutrients were lacking. Even into the mid-1940's malnutrition was partly responsible for much of the tuberculosis occurring in the mines (27).

Housing conditions in the miners' compounds were another contributing factor to the transmission of the disease. Miners' housing usually consisted of an unpartitioned room sleeping 24 persons (28). But sometimes "little or no provision was made for housing Africans," and the housing problem was not really attacked with much success until the 1950's (22).

Certainly the psycho-social stress suffered by the miners, thrown together in unfamiliar surroundings, must have been a contributory factor as well. Tuberculosis has been correlated with "stressful life experiences, such as increased occupational and residential mobility..., increasing disintegration of social participation and interpersonal

relationships, and of heightened isolation" (11). Such were the psycho-social conditions commonly found in the South African mines.

As with most ecological relationships, these factors did not operate independently. It was their combined and interacting effects which made the mines so significant as a source of tuberculosis infection. This situation led one physician to observe, "A case of open tuberculosis in a phthisis-producing industry may be as dangerous as a typhoid carrier in a kitchen" (24). Surgeon-General Gorgas, on inspecting the South African mines in 1914, accurately predicted that tuberculosis would replace pneumonia (then the number one killer) as the most important disease among the miners. His recommendations for more housing, better food and sanitation practices recognized the importance of some of these factors (23). One of the tragedies of the South African mines is that these recommendations were so slowly and inadequately carried out.

Conditions Facilitating the Diffusion of Tuberculosis in Southern Africa

Three major developments can be distinguished as factors which enhanced the diffusion of tuberculosis in southern Africa. The first of these is the expansion of workers' migration between the mines and their home areas. The second is the deliberate policy of repatriating tuberculized Africans from the mines back to their home villages.

The third is the continuing economic development of the entire region so that additional foci of infection were established.

Prothero has pointed out that migration is not a recent development in Africa; Africa has traditionally experienced small and large scale migrations of various typs (29). But economic development in South Africa set off patterns of migration which may be unique in African history (although they are paralleled elsewhere on the continent). Tens of thousands of Africans, mostly young men, migrated from their villages to work on the mines and in other industries of South Africa (and later elsewhere in southern Africa), staying more or less temporarily on the job, and then returning to their home villages again. Often they would repeat this procedure several times. Although lured on by a "promise of work, money, more freedom, and a variety of attractions to which distance lends enchantment," (30) often all they took back with them was infectious tuberculosis.

This migration of Africans to the mines began in the 1870's. By 1900 some 100,000 Africans were working on the Rand gold mines alone; by 1908 over 150,000 (22). Labor agents went out to the villages to recruit workers and many areas experienced repeated increases in the numbers of men who migrated to the mines. For example, in Botswana by 1938-40 25% of all adult males were away on

the mines, although recruiting from north of 22° South latitude had been prohibited for many years due to the high mortality rates on the mines (15). In the Transkei up to 50% of the male workers were away at any one time (31).

Most of these workers stayed about a year and then returned home (15). Even those who worked elsewhere in the cities tended to migrate rather than stay in one place permanently (32). Thus, large numbers of Africans came into contact with tubercular environments and were able by their movements to spread the disease throughout southern Africa.

Interrelated with these migratory movements was the mining companies' policy of repatriating to their home villages any Africans who contracted tuberculosis at the mines. There was some opposition to this practice on medical grounds. One doctor observed, "If they repatriated people who were foci of infection, they would infect others in their kraals, so that one was simply spitting into the well from which they had to draw their water" (33). But counter arguments won out: "natives" would probably refuse treatment; it would be useless to retain those found likely to die within a month; and provincial and private hospitals and sanatoria were sufficient to treat the "natives" (34). Thus, a policy of

"all cases fit to travel are sent to their homes" was instituted, which undoubtedly was a major factor in the spread of the infection (23).

A number of migrants applying for work were rejected for having tuberculosis, and repatriated also. In 1920, 807 of 30,544 Africans (2.6%) applying were rejected, most for having tuberculosis. Another group of 36,243 had 2% similarly rejected. In 1921, 1.7% of 62,942 examined at Germiston were refused for "defective lungs" (35).

The mortality rates among those repatriated were very high. One study traced 112 sick miners who returned to the Transkei in 1924; a year later 65 were dead and only 28 of the survivors were recovered enough to work (23). A similar study in 1918 found 26 dead, 11 not fit for work, and 37 missing of 100 repatriates traced (36). A study in Malawi of returning miners found 3% were "phthisical" and 9.5% suspected of having tuberculosis (37). While in their home villages and kraals, these infected men could readily transmit the disease to any who came into contact with them. Given the crowded sleeping conditions of the villages, it is likely that much of the spread of tuberculosis resulted from this practice of repatriation.

The continual expansion of developed areas in southern Africa was the third factor in tuberculosis diffusion. Railroad construction spurted ahead after the

discovery of gold, increasing from 69 miles of track in 1870 to over 2,000 miles in 1890. By 1897 the lines reached Bulawayo in Rhodesia, and Lusaka by 1905. The growth of cities similarly increased during this period (22). By 1928-29, 990,000 adult male Africans were employed in mineral extraction industries in South Africa alone (23).

One aspect of this economic development was the special effects of the expansion during the Second World War. Reduced food rations and an increased housing shortage contributed to a rise in the tuberculosis death rate from 250 to 700 per 100,000 in some industrial areas of South Africa (38). A similar increase in tuberculosis morbidity among town people and mine workers especially occurred in South-West Africa (39). Thus, economic development, with no adequate provision for feeding or accommodating the influx of workers, was held responsible for these increases in tuberculosis rates (27).

Evidence of Tuberculosis Diffusion in Southern Africa

Although the evidence is somewhat sketchy, the diffusion of tuberculosis in southern Africa can be fairly well demonstrated. One kind of evidence to this effect is indirect. It assumes that people who have low positive reaction rates to tuberculin and who show little resistance to the disease have had little historical exposure to tuberculosis. Thus, when cases do occur among them, that is

evidence of the spread of the disease to their locality. The data on tuberculosis morbidity and mortality rates among black South Africans in the B.E.F. in France in 1918 illustrates this point. The South African Native Labour Corps (Bantus) had a morbidity rate of 290.7 cases per 10,000 and a mortality rate of 221.9 deaths per 10,000. The Cape Colony Labour Corps (Cape Colored) had corresponding rates of 444 per 10,000 and 103.6 per 10,000 (6). The higher prevalence but lower mortality rates of tuberculosis among the Cape Colored is evidence that over time they had gained a greater resistance to the disease due to their longer contact with whites (and subsequent risk of infection). The lower morbidity but higher mortality rates for the Bantu indicate the disease was of more recent introduction among them.

Similar conclusions can be drawn for Africans from Mozambique. In 1920 of 60,164 Africans from Portugese East Africa examined before working on the mines, only 1.86% were rejected for all causes. One group of 462 applicants who were refused had only 83 cases of tuberculosis among them. In 1905 only 14 cases of tuberculosis were found in one survey of southern Mozambique, of which 10 of them had worked on the mines (35). But in 1923 a League of Nations report described tuberculosis as frequently occurring throughout Mozambique, particularly in Lourenco Marques (40). By 1949 tuberculosis was the principal cause of hospital deaths in Mozambique (9).

A second form of evidence for the spread of tuberculosis is an increase of positive tuberculin reaction rates among a population over time. 93,979 miners tested in South African (Rand) mines were 65%+ in 1930 (23). Another large series of tests in the Rand mines in 1937 showed 72%+ to tuberculin (6). By 1945 the tuberculosis mortality rate in Bantus had doubled its 1938 value (9). In 1960 a survey of African schoolchildren (both sexes) in Rhodesia found reaction rates of 69.9%+ in 18 year-olds. A survey in 1950 in the same area had reported rates of 40.8% in 16-19 year-old schoolchildren (5).

The course of the development of tuberculosis in Rhodesia is probably typical of all southern Africa. In. 1906 the Native Labour Bureau's figures showed a steady increase of tuberculosis among the Africans. Gelfand reports that the problem was such that the public was "seriously concerned and even horribly frightened at the enormous mortality of Natives employed on the mines" (26). By 1951 tuberculosis was expected to become the main health problem of the country (41).

In 1962 Grave and Ililonga reported there were few cases in the Balovale and Kabompo districts when roads were nonexistent and villages widespread. However, "In recent years...migration to the Copperbelt and Rand mines has become appreciable, and among those returning we have found quite a few tuberculotics." A largely rural area, Balovale

had an incidence of 106.4 per 100,000 vs. 120.1 per 100,000 for Rhodesia as a whole. But in accordance with normal epidemiological patterns for tuberculosis, the more densely populated villages generally had the higher case rates (7).

Shennan has developed a matrix of tuberculosis rates vs. environmental conditions as follows:

		Infection Rates (Incidence)	
		Low	High
Disease Rate (Prevalence)	Low	High (alt.) rural Hilly rural European farms Newly tuberculized areas	
	High	Towns	Low (alt.) rural Rural industrial concerns

Shennan has also mapped the prevalence of tuberculosis in four districts of Rhodesia using an index of actual rates compared to the average expected rate for the area. The map distributions provided the basis for the above matrix (5). The findings here are probably not unlike those elsewhere in southern Africa.

In Angola by 1923 there were many tuberculosis victims, especially in Luanda (40). One physician wrote of the urgent need for sanatoria for the treatment of patients (42). 395 hospital cases of tuberculosis in 1938 doubled to 770 in 1940, and by 1951 tuberculosis accounted

for ten times as many deaths as any other disease (43). In 1947 pulmonary tuberculosis was the cause of 20% of all hospital deaths in Angola (9).

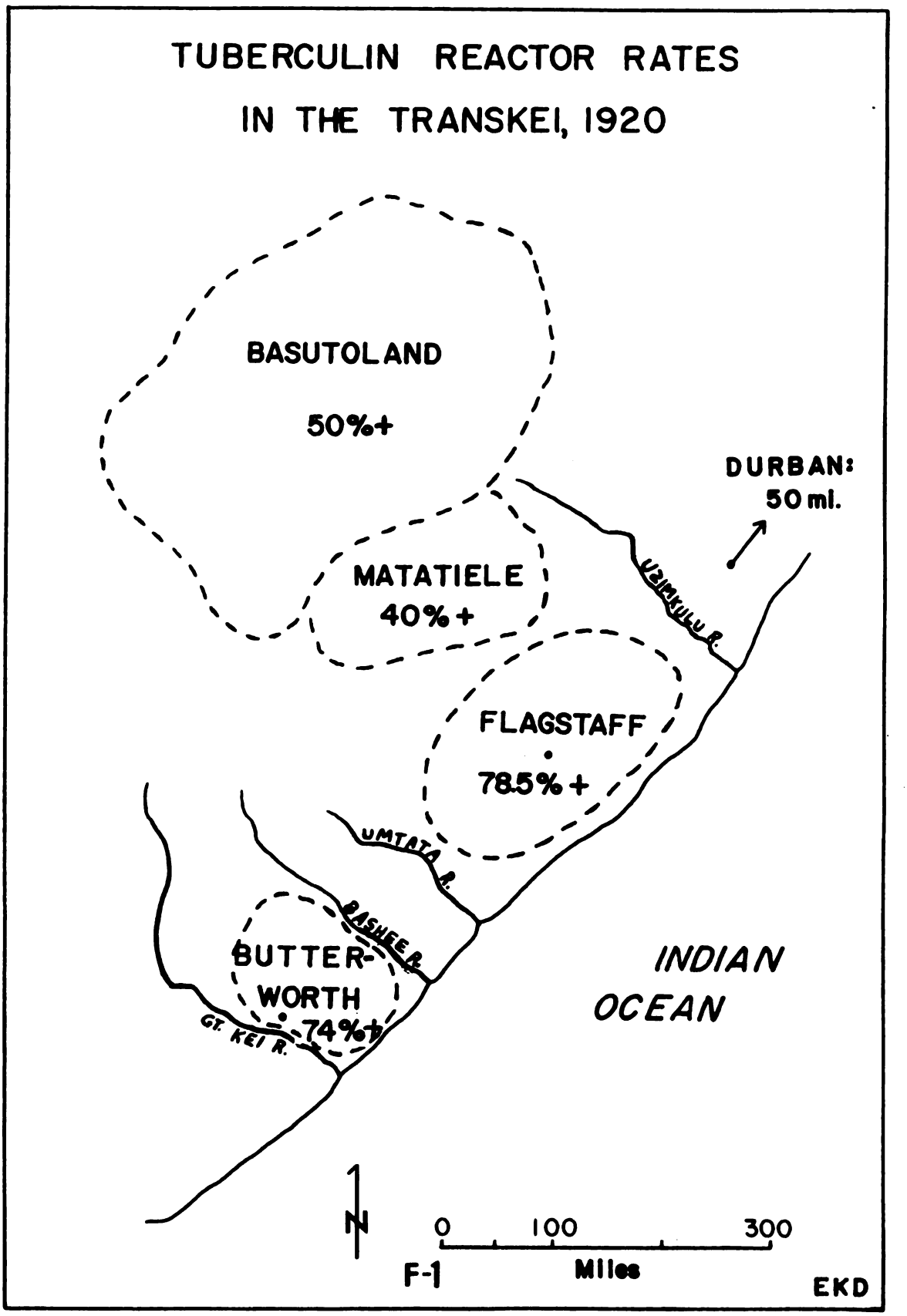
In Malawi by 1945 tuberculosis was the major cause of death, accounting for 10.4% of all hospital deaths (9). A survey in the Domasi Development Area in 1951 showed an overall tuberculin reaction rate of 65.4% indicating that infectivity rates were high in the area (44). Surveys carried out between 1952 and 1956 at the Rhokana and Mufulira mines in Zambia's Copperbelt showed active case rates of 4.3 per 1,000 for the African community as a whole, 5.6 per 1,000 for adult males vs. 2.8 per 1,000 for adult females. Reaction rates were 73.4%+ in adult males (45).

In Botswana Schapera reported that the prevalence of tuberculosis was "generally attributed to the influence of contact with Europeans, especially in the towns and other centres of work; it is held that migrants become infected there and after returning home spread the disease to others" (15). In 1933 the Principal Medical Officer reported that the distribution of tuberculosis was fairly even in southern districts, but lower rates were found in areas north of 22° South latitude where recruitment had been forbidden. The Annual Medical Report of 1936 cited many cases of tuberculosis in women, children, and young adults who had never been to the mines, but who had

friends or relatives who had worked on the mines. Other sources of infection were the railway and telegraph lines and service in the Boer War, WWI and WWII (15).

In the Transkei extensive tuberculin surveys were undertaken in 1921. The findings have been mapped on page 24. Extremely high rates (generally 75 to 90%) prevailed in adults whether living in their kraals or in locations (towns) or other institutions. Reaction rates at the Lovedale school increased from 62%+ in 1913 to 77%+ in 1928. It is notable that the lowest infection rates occurred in the areas of Matatiele where contact with Europeans had been less. But the highest incidence of active cases also was found in that region, illustrating again the general pattern of infection (less infection, high susceptibility) (23).

In 1943 physicians in the Transkei reported tuberculosis was steadily and rapidly increasing in the Butterworth area (46). One doctor felt "by far the greatest individual problem we have to deal with is that of pulmonary tuberculosis;" he stated he had seen members of families die off one by one, and that 143 new cases occurred in a three month period in one village (47). Thus, by the 1940's tuberculosis had been fairly well established in many areas of southern Africa.


TUBERCULIN REACTOR RATES
IN THE TRANSKEI, 1920
BASUTOLAND
50%+
MATATIELE
40%+
FLAGSTAFF
78.5%+
BUTTER-
WORTH
74%+
DURBAN:
50 mi.
UMTATA R.
BASHEE R.
GT. KEI R.
INDIAN
OCEAN
N
0
100
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Present Conditions

The current tuberculosis situation in southern Africa is certainly very serious (48). Surveys in the 1950's and 1960's have revealed that the disease continues to spread at a high rate. The incidence rates for several areas (with date of survey) are as follows: Transkei (1955)-5%, Natal (1956)-3%, Botswana (1964)-4%, Swaziland (1964)-3%, Lesotho (1964)-2%, Rhodesia (1960)-4%. Roelsgaard found prevalence rates to be about 5 per 1,000 (with much variation) (5). Tuberculosis notifications have increased in South Africa from 66,761 in 1966, 69,612 in 1967, to 70,684 in 1968 (49).

A study of 55 cases in a Rhodesian hospital in 1965 showed that the average active case remained at large for 4.8 months prior to treatment. During this period the victim can readily transmit the disease to others. Thus, Shennan anticipates that infectivity rates will continue to increase until a saturation level is reached with over 90% of the population reacting positively to tuberculin testing (3).

A second aspect of the current situation is that although tuberculosis is still a serious disease for Africans, the cases are becoming more like the chronic forms common to Europeans instead of the acute forms typical in African cases at the beginning of the century. This was noted in the Public Health Report for Rhodesia in 1950;

acute non-fibrosing cases were being replaced by chronic and persistent types (41). An analysis of tuberculosis morbidity patterns at a Cape Town hospital in 1967 showed that tuberculosis in the non-white wards at that time was similar to that found in the white wards in the 1930's. Bantu figures were not given, but tuberculosis admissions fell from 12.6% of all Cape Colored male admissions in 1939-43 to 4.0% of all Cape Colored male admissions in 1965 (50). Rising socio-economic conditions and widespread chemotherapy were cited as the causes of the decline, but the development of inherited resistance may also be a relevant factor in this development.

A third significant aspect of the current tuberculosis situation is the increased rates of urbanization which have been common in Africa in the last two decades. Although southern Africa is still 84.6% rural (South Africa only 49.6% rural), cities in the region are growing rapidly. Luanda, for example, has increased its population from 142,000 in 1950 to 335,000 in 1970 (est.); Pretoria gained from 278,000 in 1950 to 750,000 in 1970 (est.) (51). The World Health Organization expects the urbanization of Africa "to continue at an increasing pace for many years" (52).

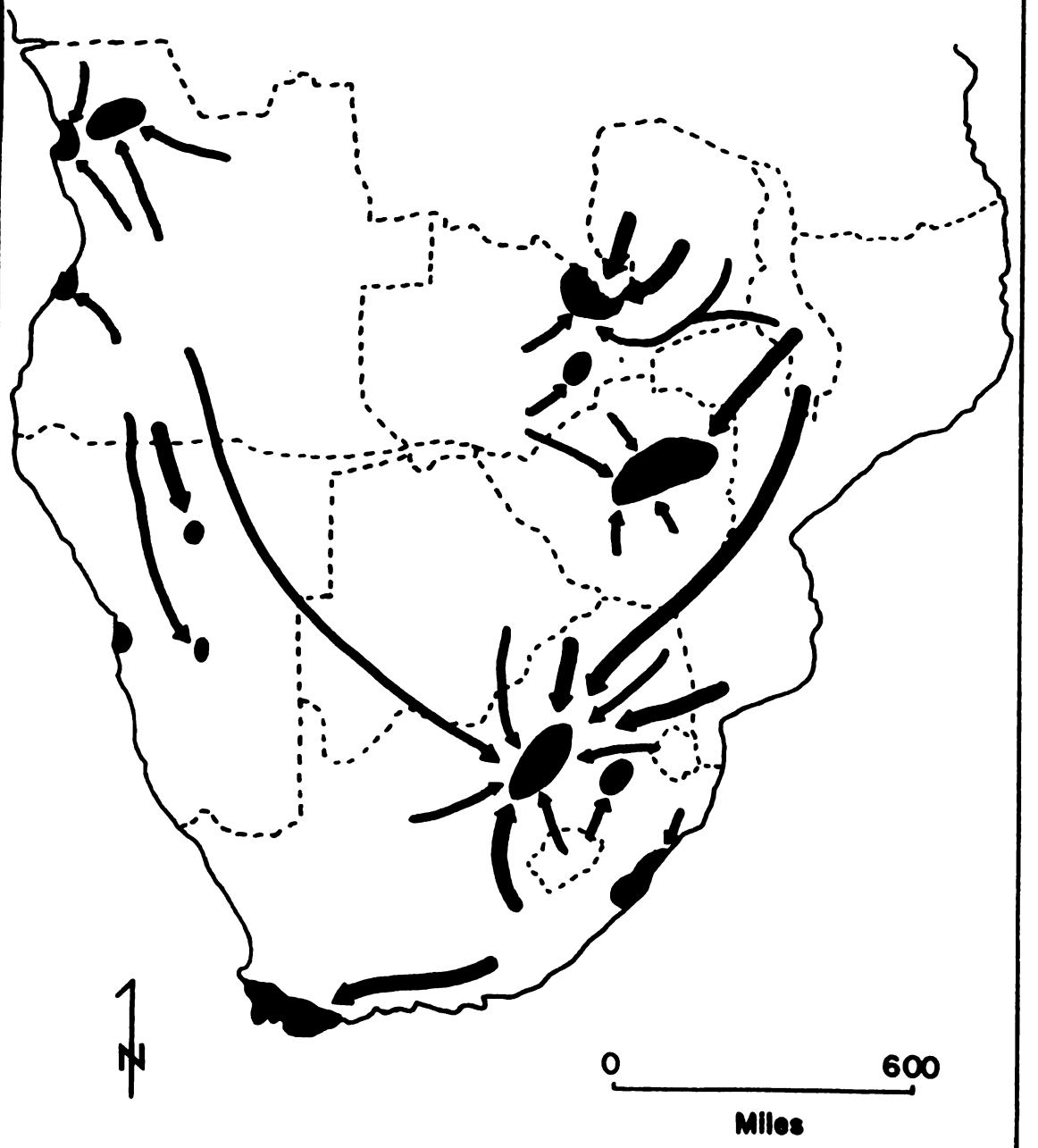
Concomitant to this urbanization has been increasing urban crowding. Single men in Lusaka averaged 1.6 men per room, with 3 to 5 men per room not uncommon (32).

Crowding increases the risk of tuberculosis infection as the density of infectious cases determines an individual's chances of encountering the infection (3). Tuberculosis can then cause the individual's ability to work to decrease and thereby touch off a downward spiral of poverty and ill health. Grave and Ililonga have described a similar process in rural environments as well: "Once tuberculosis is established in them the downward spiral of decreased vigour leading to less cultivation, to malnutrition and to spreading disease is soon begun, and the results are seen in crowded unkept villages with life at a subsistence level" (7).

Conclusions

Hance has characterized economic progress in Africa as the growth of "islands" of economic development (52). These major areas of economic development have been mapped (after Hance, (53)) for southern Africa on page 28. They have been, as indicated, the targets of population movements in this region. It would not be unreasonable to characterize the spread of tuberculosis in southern Africa as reverse movements along the same lines as the population movements. But a more precise delimitation of this development requires yet more investigation. Perhaps a more thorough survey of the medical literature might piece together better the outline developed here.

POPULATION MOVEMENTS IN SOUTHERN AFRICA



SOURCE: HANCE, 1970

F-2

But much work can be done about the current situation in order to deal more effectively with present and future tuberculosis problems in southern Africa. Shennan has indicated that an ecological approach should be used to better understand the epidemiological patterns of tuberculosis in this region. Demographic data and occupational distributions (including geographical mobility and occupations at risk to tuberculosis infection) need to be surveyed. Cultural factors--including family and community structures, natural lines of authority, domestic habits, religious beliefs, attitudes to disease and beliefs about the causation of tuberculosis, and normal routes of travel and meeting places--all these need to be understood as they affect the mode of transmission, readiness to accept treatment, the nature of and best target for propaganda, and the most suitable locations of clinics (3).

Knowledge gained about tuberculosis in southern Africa may well be applicable to the tuberculosis problems of the rest of the continent, and to other Third World areas. Patterns of tuberculosis diffusion may be similar to those of other diseases; thus successful methods for tuberculosis prevention and control might be used for other health problems as well. Since industrial and agricultural development may depend on health improvement (as well as vice versa (54)), a successful attack on tuberculosis may be a prerequisite for economic development

in Africa. Although drugs and B.C.G. vaccine can provide some measure of tuberculosis control, countervailing forces of population growth, stagnant economies (52), and a whole range of housing, nutritional, and other health problems seem to have the upper hand. Clearly, the battle has yet to be won.

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APPENDIX

EVIDENCE FROM ELSEWHERE IN AFRICA

APPENDIX

EVIDENCE FROM ELSEWHERE IN AFRICA

Liberia

Although this paper has focused on the diffusion of tuberculosis in southern Africa, it is important to realize that similar developments were occurring elsewhere on the continent. The situation in Liberia may serve as one example. At a mission established in the interior in 1926, tuberculosis was found to be rare among the Africans, occurring mainly in Mandingo traders (1). The Harvard African Expedition in 1926-27 found tuberculosis to be common in the coastal towns, especially among the Americo-Liberians, but "apparently not prevalent among the tribes inland," noting, in fact, only one case of "phthisis" (2). A survey of the Gola people in a relatively isolated area of eastern Liberia in 1952 found only a 4% tuberculin reaction rate among adolescents (3). Yet by 1964 the yearly tuberculosis incidence rate was 2% for the country as a whole (4).

Zaire

The case of Zaire is much like that of southern Africa. A survey in Leopoldville (Kinshasa) in 1913

showed only 7% of those tested were positive reactors (5). By 1915 observers concluded the disease was definitely established in Boma, Leopoldville, and Stanleyville (Kisangani) (6). In 1918 a law was passed requiring by 1921 all European immigrants to have a certificate indicating that they were free of tuberculosis (7). Nevertheless, the disease continued to spread:

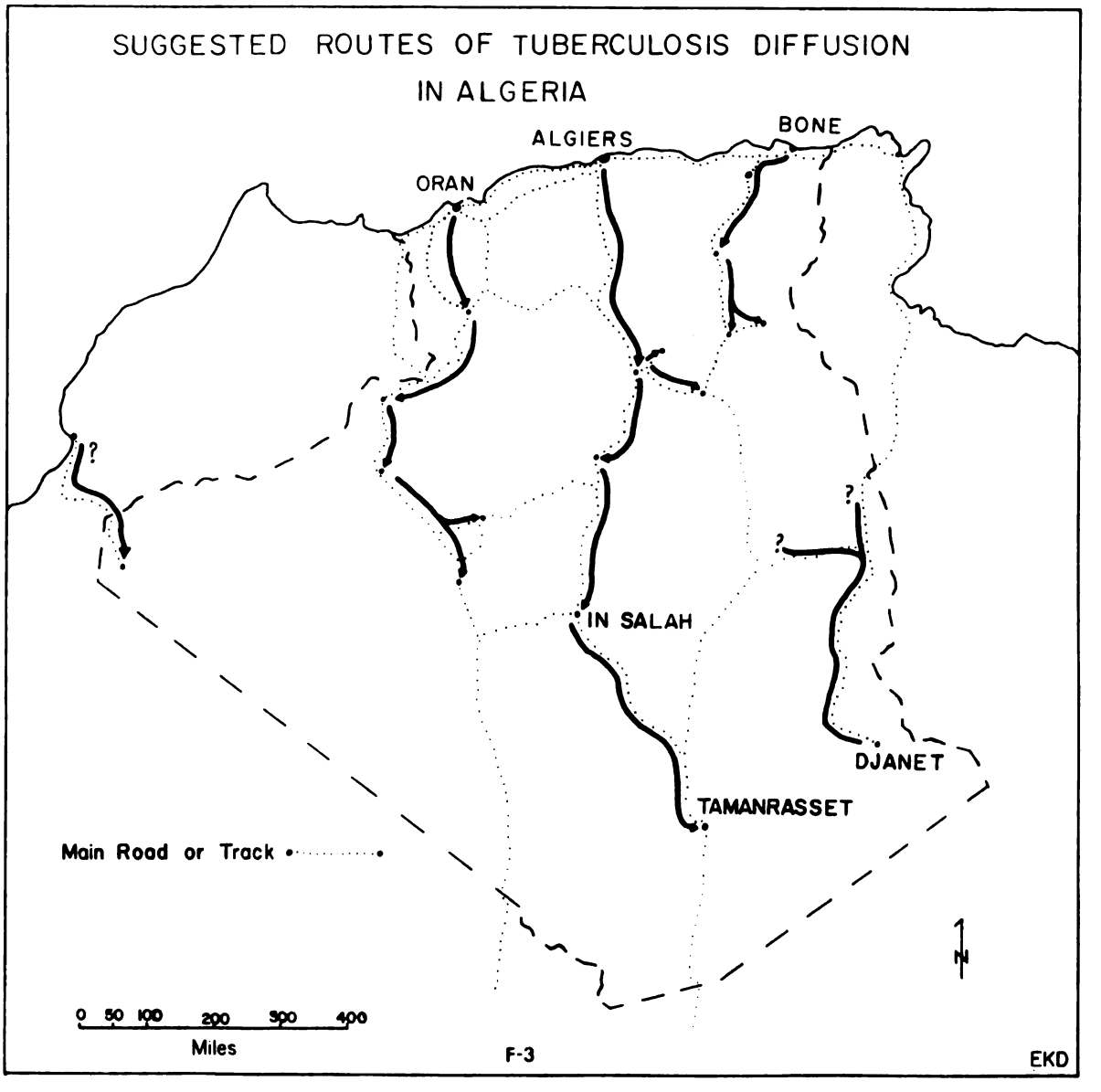
> The European carrier of Koch's bacillus has contaminated the native auxiliaries living in contact with him, soldiers, boatmen, labourers; and these, returning to their villages, have spread the infection in their turn. Little by little endemic foci of tuberculosis have become established around the more important European centres and, although still rare in the interior, the scourge is spreading (8).

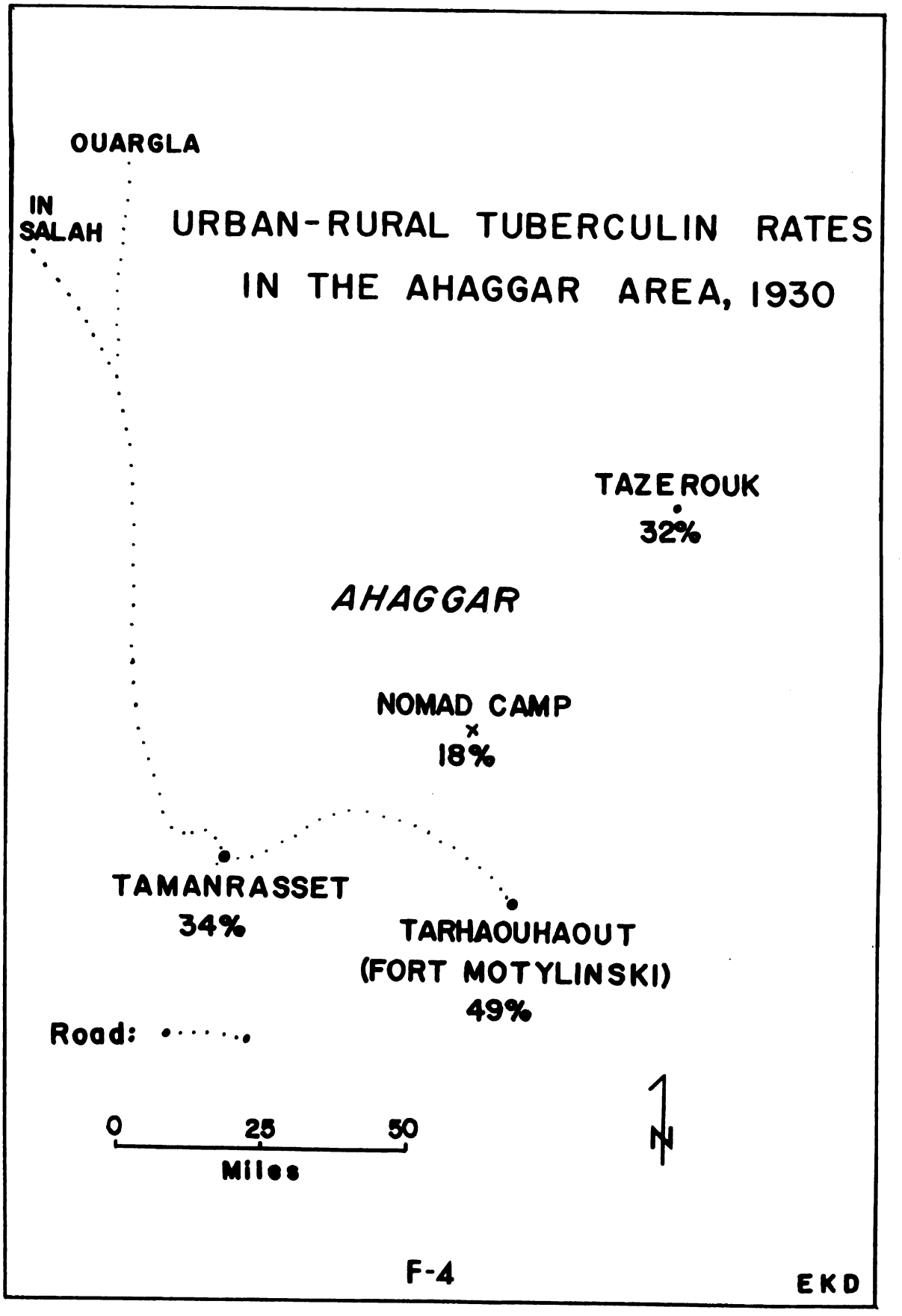
In 1925 a survey of schoolchildren in Stanleyville found 41.4% to be tuberculin positive (9). Hospital case rates increased from 652 new cases with 216 deaths in 1930 to 877 new cases with 452 deaths in 1940 (7). One Union Miniere camp reported 20 deaths out of 23 cases in 1930, yet the medical officer there rejected the use of B.C.G. vaccine as "our native population being already submitted to so much vaccination of every kind" (10). The rural-urban pattern developed typically, as a 1939 survey showed 49% positive in Leopoldville vs. 30-37% positive in interior villages (11). Even in the late 1950's tuberculosis mortality rates remained at 4-13% of the population (vs. 1% for western Europe) (12).

Algeria

In Algeria the French began a series of tuberculin testing surveys as early as 1911. By 1954 over 30 of these surveys had been completed, giving better coverage of that country than for any other African nation. However, the surveys were poorly standardized, particularly with regard to age and ethnic groups, so that comparison of the findings with each other is difficult. Moreover, the great time span between the initial surveys and the later ones and the restricted area each survey generally covered limit their use in determining the diffusion of the disease. Normally, an area once surveyed was not surveyed again, so that a temporal comparison for the same area was not possible either.

Nevertheless, the data and the opinions of the medical officers involved show that tuberculosis spread from the Mediterranean littoral (where it had long been endemic) to the interior as the French explored and established forts in the Sahara. The map on page 39 shows the probable lines along which this diffusion took place. The disease spread along the routes linking the major towns, with European settlements (e.g., Ft. Motylinski) serving as local foci of infection. The map on page 40 shows the typical pattern of higher rates in the towns as compared with nearby villages and nomads' camps, in this case for the Ahaggar area (13). Similar patterns


SUGGESTED ROUTES OF TUBERCULOSIS DIFFUSION
IN ALGERIA
ALGIERS
BONE
ORAN
IN SALAH
DJANET
TAMANRASSET
Main Road or Track
0 50 100 200 300 400
Miles
N
F-3
EKD


OUARGLA
IN SALAH
URBAN-RURAL TUBERCULIN RATES
IN THE AHAGGAR AREA, 1930
TAZEROUK
32%
AHAGGAR
NOMAD CAMP
18%
TAMANRASSET
34%
TARHAOUHAOUT
(FORT MOTYLINSKI)
49%
Road:
0
25
50
Miles
N
F-4
EKD

were found in the Tidikelt area (In Salah) and the Djanet area (14,15). A compilation of several surveys completed between 1911 and 1912 revealed average reaction rates of 49.2%+ in the coastal zone, 47.9%+ in the plateaux region, and 42%+ in the northern Sahara, illustrating how the prevalence of the infection declined away from the areas of greatest European contact and denser settlement (16,17).

Increasing rates in various localities were tied to the return of soldiers from military service in 1921 (18). A rise in the reactor rates in a relief camp near Geryville (south of Oran) from 48%+ in 1922 to 58%+ in 1923 was linked to both a shortage of food and the poor living conditions in the camp (19). Acute, rapidly developing cases in Saharan areas where infectivity rates were low were also noted, indicating that the disease was recently introduced to those localities (20). Despite B.C.G. vaccination campaigns, by the 1950's reactor rates of 20-40% or more were common even among nomadic groups which 20 to 30 years earlier had very low infectivity rates (21,22,23,24,25).

Additional Evidence

That tuberculosis is of fairly recent introduction to Africa was noted elsewhere on the continent. A German physician working in Fernando Po observed that tuberculosis was rare on that island until the 1930's when it began spreading (26)--a curious observation in view of the area's

long period of contact with Europeans. In Kenya a Dr. Philp of the Tuma Tuma Mission Station found no cases of tuberculosis among African school boys from 1909 until 1920; similarly, his examinations of hundreds of Africans for Carrier Corps service in 1916-1917 found no frank cases of the disease. But after a famine in 1918 and subsequent influenza epidemic, tuberculosis became more common (27). A survey among Nilotic peoples in the southern Sudan in 1931 discovered only 39 cases in 181,000 Africans, an infection rate of 20 per 100,000 (28).

In Tanzania tuberculosis deaths were seen to rise dramatically from 1903 to 1913 (29,30):

Number of Deaths from Tuberculosis

Year	Europeans	Africans
1903-04	3	18
1904-05	1	18
1905-06	2	9
1906-07	5	19
1907-08	6	20
1908-09	6	33
1909-10	21	49
1910-11	16	64
1911-12	17	90
1912-13	20	140

Average European tuberculin reaction rates were higher than the African rate (27%+ vs. 25%+), but both African and Indian children in Tanzania had rates higher than the adult rates, an ominous portent for future conditions (30).

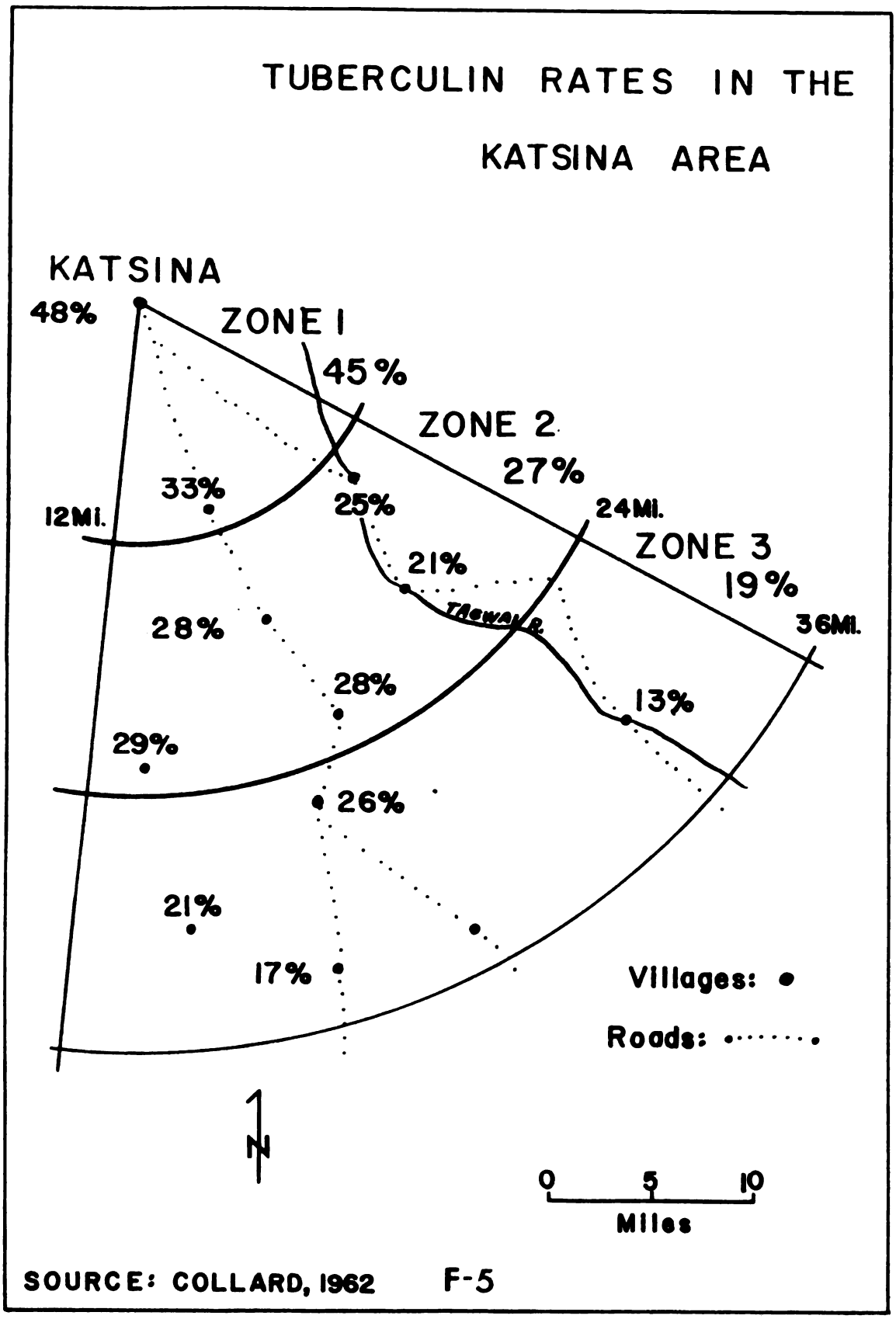
As in southern Africa, mines elsewhere on the continent formed major foci of infection. This was especially

true of the deep-shaft gold mines in the Tarkwa area of Ghana. Over 30,500 Africans were working there in the 1930's, most being migrants from elsewhere in West Africa (31). Journeying on foot from up to 500 miles away, many arrived in an exhausted physical condition (11). Silica dust in the mines further weakened the lungs, so that infection became more likely. As in South Africa, the seriously ill were encouraged to return home, while those who were not able to make the trip were entered in the Register of Death at Tarkwa as "beggars" (31).

Besides the mines and other "islands" of development, other sources of contact with Europeans provided means of introducing and spreading the disease. H. H. Scott reports that tuberculosis was rare in Sierra Leone until "Kru boys engaged themselves to serve on ships voyaging to England, infection was contracted, and on their return they constituted foci for the spread of the disease in their native haunts" (32). The movement of African troops from Eritrea (Ethiopia) and Somalia to Libya by the Italians fostered the diffusion of tuberculosis in Eritrea when the troops were returned home (33,34). Similarly, soldiers returning home from World War II are suspected to have introduced tuberculosis to the Teso District of Uganda, where only 3 cases had been recorded prior to that time (35).

Urban-rural patterns were generally maintained elsewhere in Africa, as well. The map on page 45 is a reproduction of Collard's mapping of the tuberculin reactor rates in a sector of Katsina Province in northwestern Nigeria (36). There is a trend towards lower rates moving away from the main town. Data from surveys in Senegal in 1911-12 show a similar pattern with higher rates in the larger towns (37). Surveys in 1964 showed higher incidence rates in urban than rural areas in Sierra Leone (urban-5%, rural-4%) and Zanzibar (urban-2%, rural-1%) (4). However, the prevalence of open cases was found to be 10 times greater in rural Kenya than in Nairobi as the people tend to return to their homes in the country when seriously ill (38).

Traditional African life also created conditions which may have facilitated the spread of tuberculosis. For example, a French physician attributed poor personal hygiene and eating from a common platter as conditions which facilitated the transmission of the disease among the Senegalese (39). Dr. S. Bell has observed that repeated pregnancies and prolonged lactation may predispose tuberculosis infection among Ameru (Kenya) women (40). Seasonal hunger among pastoral peoples may also predispose the infection (41). The cramped sleeping quarters common in traditional African homes also facilitate transmission of the disease (42).


TUBERCULIN RATES IN THE
KATSINA AREA
KATSINA
48%
ZONE 1
45%
ZONE 2
27%
ZONE 3
19%
12MI.
24MI.
36MI.
33%
25%
21%
28%
28%
13%
29%
26%
21%
17%
TRAIL
Villages: •
Roads: .....
N
0 5 10
Miles
SOURCE: COLLARD, 1962
F-5

However, urbanization and economic development have not only added some new conditions for tuberculosis transmission (e.g., the deep-shaft mines), but have aggravated some of the others. For example, urban housing, with "women and children sleeping in passages, under beds, or small children laid across the foot of beds already occupied by three adults," (43) is enormously inadequate continent-wide (44,45). Migration from rural areas to urban centers has significantly aided the spread of tuberculosis (46).

In sum, the causes of the diffusion of tuberculosis in Africa have been multi-variate, working in conjunction with each other as well as independently. This was noted in 1922 by an American physician visiting the Sudan; he cited stress, overcrowding, intestinal parasitism, malaria, veneral disease, alcoholism, and the "hasheeh habit" as being predispositional to tuberculosis infection (47).

Despite some massive B.C.G. vaccination campaigns (up to 1 million vaccinated each year (48)) and widespread availability of chemotherapy, the disease remains serious and continues to spread. Control measures which would attack the problem from several angles are needed--and indeed, were proposed as early as 1913 (49). The concept of a multipronged attack on disease problems is again emerging (50,51,52) and will hopefully be better implemented in the future.

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