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THE SOURCES OF THE ITALIAN MORTALITY
DECLINE, 1887-1955

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SOCIAL SCIENCE WORKING PAPER 88

July 1975

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INTRODUCTION

A recent spate of demographic literature on the environmental changes underlying twentieth century mortality declines in the West has challenged the traditional conclusion among demographers, stated by Stolnitz¹ in 1955-1956 that the accelerated gains in life expectancy after 1870 were due more to specific control measures than to further improvements in living standards. Writing in 1962, McKeown and Record² suggested that after 1870 increasing scientific knowledge about disease and a strong governmental commitment to public health improvement outweighed the importance of further increases in per capita income in explaining the late nineteenth and early twentieth century declines in mortality in Great Britain. In 1967 Somogyi³ stated that chemotherapy was responsible for most of the decline in infant and child mortality in Italy. However, Meeker⁴ and Higgs,⁵ writing on Great Britain; and Preston and Nelson,⁷ writing on international mortality changes, all now come to the conclusion that

specific medical improvements had little impact on mortality before the 1930's or even 1940's.

This intellectual turnabout is so striking that it bears further investigation for a country with better data than the U.S. and Great Britain. Further, research needs to be coordinated methodologically so that results from different countries can be adequately compared.

This paper is a somewhat limited attempt to fulfill both needs. Italy is chosen as the country for study because of the relative reliability of its cause-of-death data and because of the length of its standardized series. Deaths by sex, age, and disease are available nationally and provincially back to 1887. The United Nations Department of Economic and Social Affairs⁸ classifies the post-World War II Italian series as reasonably accurate up to age 64, after which an increasing proportion of deaths are attributed to senility. While the earlier data are undoubtedly not as good as the post-World War II data, senility never accounted for as much as 10% of the total deaths and by 1911-1913, unknown or unspecified causes constituted only 0.43% of the total deaths. Further, the Italian government has tried to make earlier cause-of-death data comparable with the more recent data in a comprehensive retrospective volume.⁹ It appears to be the case, therefore, that the entire Italian cause-of-death series can be used for mortality analysis if it is approached with caution and any unusual trends are thoroughly investigated. As a possible contribution to the second research need, a

methodology is developed throughout the paper and summarized in the appendix.

ITALIAN MORTALITY CHANGES, 1887-1955

Figure 1 shows the gains in life expectancy in Italy between 1880 and 1960 compared with the gains in life expectancy for Denmark, Norway, and Sweden between 1750 and 1970. In 1876-1887 the life expectancy in Italy was about 35 years. This was approximately equal to the life expectancy in Scandinavia in the late eighteenth century and was twelve years behind Scandinavia in the 1880's, suggesting that the mortality transition was just beginning in Italy at that time. The graph then shows Italy rapidly catching-up with Scandinavia in the twentieth century.

The mortality transition summarized in figure 1 can also be expressed as a decline in aggregate sex-age-standardized mortality rates as in figure 2. The mortality rates summarized in figure 2 were calculated from equation (1) (appendix).

The sex-age-standardized mortality rates shown in their aggregate form in figure 2 can be broken down into sex-age-disease-specific mortality rates. Changes in these sex-age-disease-specific mortality rates, in turn, can be expressed as percentage contributions to declines in the aggregate standardized mortality

FIGURE 1

LIFE EXPECTANCY AT BIRTH FOR ITALY (1880-1960)
AND DENMARK, NORWAY, AND SWEDEN (1750 - 1950)

Source: John D. Durand, unpublished figures

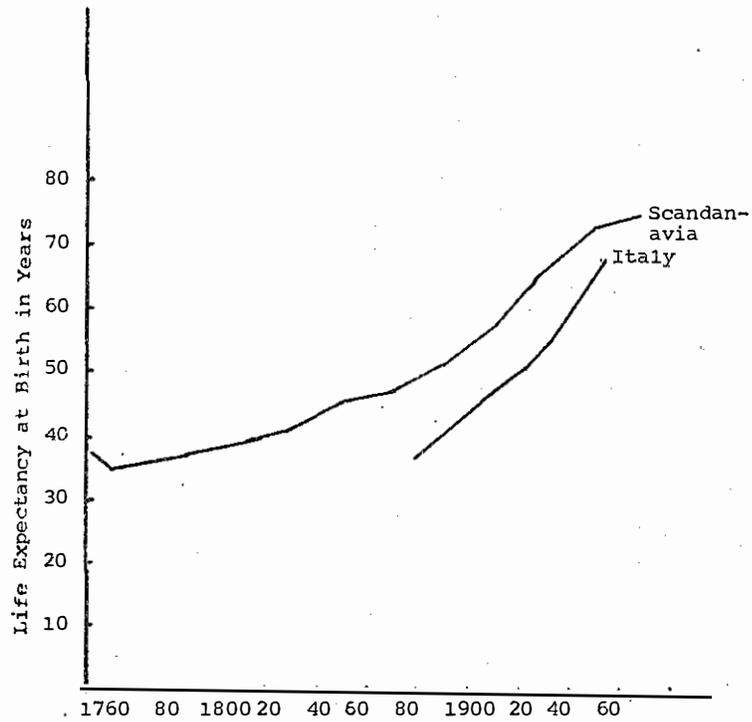
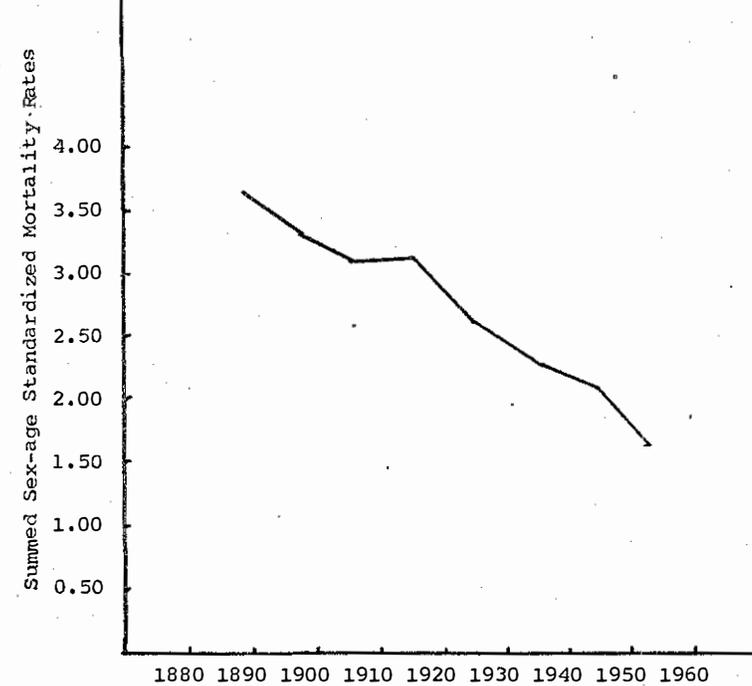


FIGURE 2

ITALY, AGGREGATE SEX-AGE-STANDARDIZED MORTALITY RATES
(1887-1889 to 1951-1955)

Source: Italy, ISTAT, Cause di Morte, 1887-1955, (Rome: ISTAT, 1958); Italy, ISTAT, Sommario di Statistiche Storiche Italiane, 1861-1955, (Rome: Istituto Poligrafico dello Stato, 1958)



decline. Table 1 outlines the percentage contributions of decadal declines in sex-age-disease-specific mortality rates to the aggregate mortality decline for those diseases whose standardized declines contributed as much as 1% each to the aggregate mortality decline between 1887-1889 and 1951-1955. The values summarized in Table 1 were calculated from equation (2) (appendix). 1911-1920 is omitted from the analysis from this point on because the slight increase in the aggregate mortality rate associated with World War I and the influenza epidemic makes the interpretation of percentage contributions of sex-age-disease-specific mortality declines problematical.

Over the whole period declines in tuberculosis, among all ages but particularly among males and females 15-39; pneumonia, particularly among males and females 5 years and older; and gastroenteritis and bronchitis, particularly among children under 5 and adults over 60 each contributed more than 10% to the aggregate mortality decline. Childhood infectious and parasitic diseases (measles, whooping cough, diphtheria, scarlet fever) all together contributed about 8% and declines in the other diseases each contributed 5% or less. There was little sex difference in the contributions of sex-age-disease-specific mortality declines to the aggregate mortality decline.

Breaking the mortality transition down by decades indicates that there were significant differences in the relative importance of the contributions of different diseases and age groups. Between 1887-1889 and 1895-1900 declines

TABLE 1

PERCENTAGE CONTRIBUTIONS OF SEX-AGE-DISEASE-SPECIFIC MORTALITY DECLINES TO THE SEX-AGE-STANDARDIZED MORTALITY DECLINE IN ITALY

| 1887-1889 to 1895-1900 | meas-les | whoop- ing cough | diph- theria | scarlet fever | tuber- culosis | menin- gitis | gastro- enter- itis | mal- ty- aria | pneu- monia | bron- chitis |
|--|----------|------------------------|-----------------|------------------|-------------------|-----------------|---------------------------|---------------------|----------------|-----------------|
| males 0-1 | 1.1 | .5 | .8 | .4 | 1.2 | -.2 | -2.4 | .4 | .8 | .2 |
| females 0-1 | .9 | .5 | .7 | .3 | 1.1 | -.2 | -2.2 | .3 | .7 | .3 |
| males 1-4 | 4.2 | .9 | 4.3 | 2.3 | 2.4 | -.1 | 1.1 | .8 | 2.0 | .6 |
| females 1-4 | 4.0 | 1.0 | 4.0 | 2.0 | 2.2 | -.2 | -1.8 | .6 | 2.0 | -.4 |
| males 5-9 | .6 | .1 | 1.8 | .9 | 0.0 | -.1 | -.2 | .3 | .4 | .1 |
| females 5-9 | .7 | .1 | 2.2 | .8 | .1 | -.2 | -.3 | .2 | .6 | .0 |
| males 10-14 | .1 | 0.0 | .4 | .2 | 0.0 | -.1 | -.1 | .1 | .1 | 0.0 |
| females 10-14 | .1 | 0.0 | .4 | .2 | .2 | 0.0 | -.1 | .1 | .2 | 0.0 |
| males 15-39 | | | | | -1.0 | .2 | -.2 | .6 | .4 | .2 |
| females 15-39 | | | | | .7 | 0.0 | -.2 | .4 | .2 | -.6 |
| males 40-49 | | | | | -.4 | .2 | -.1 | .4 | .7 | -.2 |
| females 40-49 | | | | | .4 | .1 | 0.0 | .2 | .5 | -.2 |
| males 50-59 | | | | | -.4 | .3 | -.4 | .5 | 1.0 | -.2 |
| females 50-59 | | | | | .1 | .1 | -.3 | .4 | .8 | 0.0 |
| males 60+ | | | | | .2 | .6 | -.2 | 1.2 | 2.3 | 2.2 |
| females 60+ | | | | | .7 | .4 | -.2 | 1.3 | 2.6 | 3.0 |
| total | 11.7 | 3.1 | 14.6 | 7.1 | 7.5 | .8 | -13.2 | 7.6 | 15.3 | 4.2 |
| percentage of sex-age-standardized death rate decline explained: | 51.9 | | | | | | | | | |

1895-1900

to

1901-1910

males 0-1
females 0-1
males 1-4

.2
.2
-.3

.4
.4
.3

.2
.1
1.1

.5
.5
1.3

.1
.1
.4

.5
.4
.6

.2
.6
1.1

.6
.2
.8

.7
2.0
2.0

TABLE 1 (continued)

| 1895-1900 to 1901-1910 | meas-les | whoop-ing cough | diph-theria | scarlet fever | tuber- culosis | menin- gitis | gastro- enter- itis | mal- aria | ty- phoid | pneu- monia | bron- chitis |
|---|----------|--------------------|-------------|------------------|-------------------|-----------------|---------------------------|--------------|--------------|----------------|-----------------|
| females 1-4 | 0.0 | .7 | 1.3 | .5 | 1.6 | .7 | 3.7 | 1.4 | 1.0 | -.6 | 2.9 |
| males 5-9 | 0.0 | 0.0 | .6 | .3 | .3 | .2 | .1 | .4 | .5 | .1 | .2 |
| females 5-9 | 0.0 | .1 | .7 | .2 | .4 | .2 | .1 | .4 | .6 | 0.0 | .2 |
| males 10-14 | 0.0 | 0.0 | .1 | 0.0 | .1 | .1 | 0.0 | .2 | .4 | 0.0 | .1 |
| females 10-14 | 0.0 | 0.0 | .2 | 0.0 | .2 | .1 | 0.0 | .2 | .4 | 0.0 | 0.0 |
| males 15-39 | | | | | -.1 | .3 | 0.0 | 1.0 | 1.4 | 1.6 | .3 |
| females 15-39 | | | | | .8 | .3 | -.2 | .7 | 1.8 | .9 | .3 |
| males 40-49 | | | | | -.1 | .1 | .2 | .6 | .2 | 1.8 | .2 |
| females 40-49 | | | | | .6 | .1 | .2 | .4 | .4 | .8 | .2 |
| males 50-59 | | | | | .2 | .1 | .5 | .8 | .4 | 2.0 | .4 |
| females 50-59 | | | | | .4 | .1 | .5 | .5 | .4 | 1.1 | .7 |
| males 60+ | | | | | .5 | .4 | 4.4 | 1.7 | .6 | 4.4 | 3.7 |
| females 60+ | | | | | .6 | .3 | 4.4 | 1.4 | .6 | 3.2 | 4.9 |
| total | .1 | 1.9 | 4.3 | 1.6 | 7.8 | 4.5 | 15.3 | 12.0 | 9.9 | 13.2 | 20.8 |
| percentage of sex-age-standardized death rate decline explained: 91.4 | | | | | | | | | | | |

1901-1910
to
1921-1930

| | | | | | | | | | | | |
|---------------|-----|-----|-----|-----|----|----|-----|-----|-----|------|-----|
| males 0-1 | .1 | .2 | 0.0 | 0.0 | .3 | .4 | .7 | .1 | 0.0 | -1.2 | 3.0 |
| females 0-1 | .1 | .3 | 0.0 | 0.0 | .2 | .3 | .6 | .1 | 0.0 | -.9 | 2.3 |
| males 1-4 | .6 | .3 | .3 | 0.0 | .6 | .6 | 3.0 | .2 | .2 | -.9 | 2.2 |
| females 1-4 | .6 | .4 | .3 | 0.0 | .7 | .7 | 3.7 | .3 | .2 | -.8 | 2.5 |
| males 5-9 | .1 | 0.0 | .1 | 0.0 | .2 | .3 | .4 | .1 | .1 | .1 | .2 |
| females 5-9 | .1 | 0.0 | .1 | 0.0 | .4 | .4 | .4 | .1 | .1 | .1 | .3 |
| males 10-14 | 0.0 | 0.0 | 0.0 | 0.0 | .1 | .1 | .1 | 0.0 | 0.0 | 0.0 | 0.0 |
| females 10-14 | 0.0 | 0.0 | 0.0 | 0.0 | .3 | .2 | .1 | 0.0 | .1 | 0.0 | .1 |

TABLE 1 (continued)

| 1901-1910 to 1921-1930 | meas-les | whoop-ing cough | diph-theria | scarlet fever | tuber- culosis | menin- gitis | gastro- enter- itis | mal- aria | ty- phoid | pneu- monia | bron- chitis |
|---|----------|--------------------|-------------|------------------|-------------------|-----------------|---------------------------|--------------|--------------|----------------|-----------------|
| males 15-39 | | | | | 1.2 | .4 | .6 | .2 | .7 | 1.3 | .5 |
| females 15-39 | | | | | 2.0 | .5 | .8 | .2 | .5 | .7 | .7 |
| males 40-49 | | | | | .1 | .2 | .4 | .2 | .1 | 1.5 | .3 |
| females 40-49 | | | | | .5 | .1 | .5 | .1 | .1 | .6 | .4 |
| males 50-59 | | | | | .1 | .2 | .9 | .2 | .1 | 2.3 | .7 |
| females 50-59 | | | | | .2 | .1 | .8 | .1 | .1 | 1.2 | .7 |
| males 60+ | | | | | .1 | .2 | 3.9 | .3 | .1 | 2.7 | 2.8 |
| females 60+ | | | | | .1 | .2 | 4.0 | .3 | .1 | 2.4 | 3.7 |
| total | 1.6 | 1.2 | .8 | 0.0 | 7.1 | 4.9 | 20.9 | 2.5 | 2.5 | 9.1 | 20.4 |
| percentage of the sex-age-standardized death rate decline explained: 71.0 | | | | | | | | | | | |

1921-1930
to
1931-1940

| | | | | | | | | | | | |
|---|-----|-----|-----|-----|------|-----|------|-----|-----|------|-----|
| males 0-1 | .2 | .2 | 0.0 | 0.0 | .4 | .3 | 6.0 | .1 | 0.0 | -1.2 | 2.2 |
| females 0-1 | .2 | .2 | 0.0 | 0.0 | .3 | .2 | 5.1 | .1 | 0.0 | -1.1 | 1.8 |
| males 1-4 | 1.0 | .2 | .2 | .4 | .8 | .4 | 3.6 | .3 | .2 | 1.2 | 1.5 |
| females 1-4 | 1.0 | .3 | .1 | .4 | .8 | .4 | 3.9 | .3 | .2 | 1.2 | 1.6 |
| males 5-9 | .1 | 0.0 | 0.0 | .1 | .3 | .1 | .2 | .1 | .1 | .1 | .1 |
| females 5-9 | .1 | 0.0 | 0.0 | .1 | .3 | .1 | .3 | .1 | .1 | .1 | .1 |
| males 10-14 | 0.0 | 0.0 | 0.0 | 0.0 | .3 | .1 | .2 | 0.0 | .2 | .1 | 0.0 |
| females 10-14 | 0.0 | 0.0 | 0.0 | 0.0 | .7 | .1 | .2 | 0.0 | .2 | .1 | 0.0 |
| males 15-39 | | | | | 5.8 | .2 | .4 | .2 | .6 | .6 | .1 |
| females 15-39 | | | | | 6.5 | .2 | .6 | .2 | 1.0 | 1.1 | .2 |
| males 40-49 | | | | | 1.0 | .1 | .2 | .1 | .2 | .6 | 0.0 |
| females 40-49 | | | | | 1.6 | 0.0 | .4 | .1 | .2 | .5 | .1 |
| males 50-59 | | | | | .7 | 0.0 | .5 | .2 | .3 | 1.2 | .2 |
| females 50-59 | | | | | 1.1 | 0.0 | .6 | .1 | .2 | .9 | .3 |
| males 60+ | | | | | .8 | .1 | 2.3 | .3 | .3 | 1.1 | .7 |
| females 60+ | | | | | .8 | 0.0 | 2.2 | .2 | .3 | 1.1 | 1.0 |
| total | 2.6 | .9 | .3 | 1.0 | 22.2 | 2.3 | 26.8 | 2.4 | 4.1 | 7.6 | 9.9 |
| percentage of the sex-age-standardized death rate decline explained: 80.1 | | | | | | | | | | | |

TABLE 1 (continued)

| | whoop- ing meas- les | diph- theria | scarlet fever | tuber- culosis | menin- gitis | gastro- enter- itis | mal- aria | ty- phoid | pneu- monia | bron- chitis |
|--|-------------------------------|-----------------|------------------|-------------------|-----------------|---------------------------|--------------|--------------|----------------|-----------------|
| 1887-1889 to 1951-1955 | | | | .3 | .1 | .4 | .3 | .3 | 1.8 | .2 |
| males 50-59 | | | | .5 | .1 | .4 | .2 | .2 | 1.1 | .4 |
| females 60+ | | | | .3 | .2 | 1.8 | .5 | .5 | 3.6 | 2.0 |
| females 60+ | 2.7 | 1.2 | 3.1 | 11.6 | 2.2 | 2.0 | .5 | .5 | 3.2 | 2.7 |
| total | | | | 11.6 | 2.6 | 14.1 | 3.9 | 5.1 | 14.8 | 11.9 |
| percentage of the sex-age-standardized death rate decline explained: | | | | | | | | | | 72.4 |

Source: Italy, ISTAT, Cause di Morte, 1887-1955, (Rome: ISTAT), 1958.
Italy, ISTAT, Sommario.

in childhood infectious diseases and tuberculosis among children under 5 (primarily non-pulmonary tuberculosis) accounted for more than 40% of the aggregate mortality decline. Their combined contribution dropped to less than 10% in 1895-1900 to 1901-1910 and less than 5% thereafter. On the other hand, declines in adult tuberculosis (generally pulmonary tuberculosis) only showed a moderate contribution before 1921-1930 and then, with the exception of males during World War II, a consistently high contribution thereafter. Tuberculosis was not broken down into pulmonary and non-pulmonary tuberculosis because sex-age-specific tuberculosis deaths by type were not available before 1925. Other diseases whose declines contributed the most in the early decades were malaria and typhoid. In contrast, declines in meningitis, gastro-enteritis, and bronchitis mortality contributed the most between 1895-1900 to 1901-1910 and 1921-1940. The contributions of declines in pneumonia mortality were 5-15% between 1895-1900 and 1931-1940, rose to a peak of 47% from 1931-1940 to 1941-1950, and then fell to 24% from 1941-1950 to 1951-1955.

The next section develops hypotheses as to the socio-economic, environmental, and technological changes underlying these observed sex-age-disease-specific mortality changes on the basis of medical literature, and public health and economic literature dealing with specific changes in Italy.

TABLE 1 (continued)

| 1931-1940 to 1941-1950 | meas-les | whoop-ing cough | diph-theria | scarlet-fever | tuber-culosis | menin-gitis | gastro-enter-itis | mal-aria | ty-phoid | pneu-monia | bron-chitis |
|------------------------------|----------|-----------------|-------------|---------------|---------------|-------------|-------------------|----------|----------|------------|-------------|
| males 0-1 | .1 | .2 | 0.0 | 0.0 | .1 | 0.0 | 4.6 | 0.0 | 0.0 | 1.6 | .8 |
| females 0-1 | .1 | .2 | 0.0 | 0.0 | .1 | 0.0 | 4.2 | 0.0 | 0.0 | .8 | .6 |
| males 1-4 | .6 | .2 | .3 | .2 | .2 | .1 | 2.3 | .1 | 0.0 | 3.4 | .4 |
| females 1-4 | .6 | .3 | .2 | .2 | .1 | .1 | 2.7 | .1 | 0.0 | 3.1 | .4 |
| males 5-9 | .1 | 0.0 | .2 | .1 | .1 | 0.0 | .1 | 0.0 | .1 | .3 | 0.0 |
| females 5-9 | .1 | 0.0 | .2 | .1 | .1 | 0.0 | .1 | 0.0 | .1 | .3 | 0.0 |
| males 10-14 | 0.0 | 0.0 | 0.0 | 0.0 | .2 | 0.0 | 0.0 | 0.0 | 0.0 | .1 | 0.0 |
| females 10-14 | 0.0 | 0.0 | 0.0 | 0.0 | .3 | 0.0 | 0.0 | 0.0 | 0.0 | .1 | 0.0 |
| males 15-39 | | | | | 0.0 | .1 | 0.0 | 0.0 | .2 | 3.3 | 0.0 |
| females 15-39 | | | | | 4.0 | 0.0 | .1 | .1 | -.1 | 2.0 | .1 |
| males 40-49 | | | | | -.1 | 0.0 | 0.0 | .1 | -.1 | 2.7 | .1 |
| females 40-49 | | | | | 1.0 | 0.0 | .1 | 0.0 | 0.0 | 1.6 | .1 |
| males 50-59 | | | | | -.4 | 0.0 | .1 | .2 | 0.0 | 4.2 | .4 |
| females 50-59 | | | | | .7 | 0.0 | .2 | .1 | 0.0 | 3.0 | .5 |
| males 60+ | | | | | -.1 | 0.0 | 1.6 | .2 | 0.0 | 10.3 | 4.6 |
| females 60+ | | | | | .3 | 0.0 | 1.7 | .1 | 0.0 | 10.1 | 5.1 |
| total | 1.6 | .9 | .9 | .6 | 6.6 | .3 | 17.8 | 1.0 | .2 | 46.9 | 13.1 |

percentage of the sex-age-standardized death rate decline explained: 89.9

1941-1950

to

1951-1955

| | | | | | | | | | | | |
|-------------|-----|-----|-----|-----|----|-----|-----|-----|-----|-----|-----|
| males 0-1 | 0.0 | .1 | .1 | 0.0 | .1 | .1 | 3.1 | 0.0 | 0.0 | 2.2 | .4 |
| females 0-1 | 0.0 | .2 | .1 | 0.0 | .1 | .1 | 2.7 | 0.0 | 0.0 | 1.7 | .3 |
| males 1-4 | .1 | .1 | .2 | 0.0 | .3 | .1 | 1.8 | 0.0 | 0.0 | 1.7 | .2 |
| females 1-4 | .1 | .2 | .2 | 0.0 | .3 | .1 | 2.0 | 0.0 | 0.0 | 1.8 | .2 |
| males 5-9 | 0.0 | 0.0 | 0.0 | 0.0 | .2 | 0.0 | .1 | 0.0 | .1 | .2 | 0.0 |
| females 5-9 | 0.0 | 0.0 | 0.0 | 0.0 | .2 | 0.0 | .1 | 0.0 | .1 | .2 | 0.0 |

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TABLE 1 (continued)

| 1941-1950 to 1951-1955 | meas-les | whoop-ing cough | diph-theria | scarlet-fever | tuber-culosis | menin-gitis | gastro-enter-itis | mal-aria | ty-phoid | pneu-monia | bron-chitis |
|------------------------------|----------|-----------------|-------------|---------------|---------------|-------------|-------------------|----------|----------|------------|-------------|
| males 10-14 | 0.0 | 0.0 | 0.0 | 0.0 | .2 | 0.0 | 0.0 | 0.0 | .1 | .1 | 0.0 |
| females 10-14 | 0.0 | 0.0 | 0.0 | 0.0 | .3 | 0.0 | 0.0 | 0.0 | .2 | .1 | 0.0 |
| males 15-39 | | | | | 5.5 | .1 | .2 | .1 | .7 | 1.0 | .1 |
| females 15-39 | | | | | 4.9 | .1 | .1 | 0.0 | 1.0 | 1.0 | .1 |
| males 40-49 | | | | | 1.7 | 0.0 | .1 | 0.0 | .1 | .8 | 0.0 |
| females 40-49 | | | | | 1.0 | 0.0 | .1 | 0.0 | .2 | .5 | 0.0 |
| males 50-59 | | | | | 1.1 | 0.0 | .2 | 0.0 | .1 | 1.5 | -.2 |
| females 50-59 | | | | | .9 | 0.0 | .2 | 0.0 | .2 | 1.0 | 0.0 |
| males 60+ | | | | | .5 | 0.0 | .7 | .1 | .1 | 5.2 | -.6 |
| females 60+ | | | | | .7 | 0.0 | .7 | 0.0 | .1 | 4.6 | .1 |
| total | .2 | .6 | .6 | 0.0 | 18.0 | .6 | 12.1 | .2 | 3.0 | 23.6 | .6 |

percentage of the sex-age-standardized death rate decline explained: 59.5

1887-1889

to

1951-1955

| | | | | | | | | | | | |
|---------------|-----|-----|----|-----|-----|----|-----|----|----|-----|-----|
| males 0-1 | .2 | .2 | .2 | .1 | .4 | .2 | 1.9 | .2 | .2 | 0.0 | 2.0 |
| females 0-1 | .2 | .3 | .1 | .1 | .3 | .2 | 1.6 | .2 | .1 | -.1 | 1.3 |
| males 1-4 | 1.0 | .3 | .9 | .4 | .8 | .3 | 2.2 | .4 | .4 | .5 | 1.2 |
| females 1-4 | 1.0 | .4 | .9 | .4 | .9 | .3 | 2.5 | .4 | .5 | .5 | 1.3 |
| males 5-9 | .1 | 0.0 | .4 | .2 | .2 | .1 | .1 | .1 | .2 | .1 | .1 |
| females 5-9 | .2 | 0.0 | .4 | .2 | .3 | .1 | .2 | .1 | .2 | .1 | .1 |
| males 10-14 | 0.0 | 0.0 | .1 | 0.0 | .1 | .1 | 0.0 | .1 | .1 | 0.0 | 0.0 |
| females 10-14 | 0.0 | 0.0 | .1 | 0.0 | .3 | .1 | .1 | .1 | .2 | .1 | 0.0 |
| males 15-39 | | | | | 2.2 | .2 | .2 | .3 | .6 | 1.3 | .2 |
| females 15-39 | | | | | 3.2 | .2 | .3 | .2 | .7 | .8 | .2 |
| males 40-49 | | | | | .5 | .1 | .2 | .2 | .2 | 1.2 | .1 |
| females 40-49 | | | | | .8 | .1 | .2 | .1 | .2 | .6 | .1 |

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SURVEY OF MEDICAL, AND PUBLIC HEALTH AND ECONOMIC
LITERATURE AND DEVELOPMENT OF HYPOTHESES

Medical Literature

Gastro-enteritis and Childhood Infections and Parasitic
Diseases:¹⁰

Surveying the medical literature indicates that several of the isolated diseases have interrelated underlying causes. In particular, gastro-enteritis, a term describing a group of diarrheal digestive disturbances, which is symptomatic of poor nutrition and poor environmental sanitation, tends to work its worst havoc in a synergistic relationship with other diseases. In both children and adults diarrhea leads to general weakness which leads to more virulent cases of diseases which might be mild in healthy people. A person surviving an attack of infectious disease is further weakened, perhaps already suffering from malnutrition, and may easily succumb to the next attack of diarrhea. Among young children this syndrome is most often precipitated by weaning to unsanitary and low protein content food. Among adults development of the syndrome may be related either to poor sanitation and lack of protein in the diet or to the prevalence of some other nutritional deficiency disease, such as pellagra, which used to be common in Northern Italy.

Doctors attribute declines in gastro-enteritis among young children to the following factors: economic improvement;

control of communicable diseases, including environmental sanitation; and the development of modern pediatrics. Economic improvement combined with better knowledge of infant care and feeding to make better nutrition and better home nursing of sick children possible. Control of communicable diseases reduced exposure to diseases which exacerbated the effects of gastro-enteritis; and modern pediatrics made it possible to save children who would have died from gastro-enteritis, malnutrition, or infectious diseases in the past. Analogously, controlling gastro-enteritis through improving nutrition and sanitation reduced the risk of death from infectious diseases.

In addition to the beneficial effects of better nutrition and environmental sanitation on childhood infectious and parasitic diseases, particular medical changes and other environmental changes could have contributed to declines in mortality due to these diseases in certain time periods. In the first decade (1887-1889 to 1895-1900) there seems to have been a significant decline in the virulence of the diphtheria and perhaps the scarlet fever strains prevalent in Europe. Some studies indicate that these disease mutations were permanent while others suggest that the disease strains had become more virulent again by the first or second decade of the twentieth century. A diphtheria anti-toxin was developed in 1890, but there is considerable debate in the literature as to its usefulness and its potential contribution to the decline in diphtheria

mortality. However, in 1913 a diphtheria vaccination was developed, which does seem to have been successful. In the mid-1920's a scarlet fever anti-toxin was developed and serum treatment began to be used to reduce measles mortality during major epidemics. Penicillin, which greatly reduces the risk of complications from childhood infectious diseases was developed in 1928, although the evidence suggests it was not widely used before World War II. In 1931 a whooping cough vaccination was developed and in 1935 the first successful control of disease with sulfanimides was demonstrated. Since then, chemotherapy has revolutionized the control of infectious disease.

Tuberculosis:¹¹

Before 1921, when BCG vaccine was developed, the only means of preventing the contraction of tuberculosis were to maintain bodily resistance through good nutrition and fresh air, to pasteurize milk to prevent bovine infection, and to isolate the sick from the well. In 1943 it was discovered that streptomycin kills the tuberculosis bacillus, making chemotherapy of tuberculosis possible. Before that discovery the tuberculosis cure depended upon a lengthy period of rest and a high protein diet.

Additional aids to diagnosis and cure of tuberculosis were developed during the first decade (1887-1889 to 1895-1900) or shortly before. Forlanini made artificial pneumothorax possible in 1882. Although this method did not gain international

acceptance until the International Medical Conference of 1912, it was accepted earlier in Italy. In 1895 Roentgen discovered X-rays, making it possible to diagnose pulmonary tuberculosis before the terminal stage of the disease had set in. The other major diagnostic aid, the tuberculin test, was developed in 1890. With the possible exception of artificial pneumothorax, these discoveries probably had little immediate impact on tuberculosis mortality, however.

Typhoid Fever:¹²

Doctors agree that declines in typhoid fever mortality are due almost entirely to improved water sanitation. This can be accomplished by households boiling water or making sure their drinking water comes from uncontaminated sources, or through government inspection and water sanitation.

Malaria:¹³

There were no major improvements in our knowledge of how to control malaria between the beginning of the period under study and World War II. Mosquitoes could be controlled by draining swamps and keeping irrigation water moving, and by screening the windows of homes. The primary malaria prophylaxis was quinine, which was first brought to Europe in the seventeenth century. After World War II the use of DDT to control mosquitoes spread widely and a variety of cheaper prophylaxis drugs were developed.

In addition to the virulent forms of the disease, there is a chronic, debilitating form of malaria. The case-fatality rate is relatively low, but it acts in synergisms with other diseases. Adults are more likely to succumb to bronchitis, while children are more likely to die from infectious and parasitic or respiratory diseases.

Bronchitis and Pneumonia:¹⁴

These diseases are more often complications of other diseases than initiating causes of illness. Bronchitis may be an acute upper respiratory infection, a complication of some infectious disease, or the terminal stage of chronic malaria or heart disease. Pneumonia is often a complication of one of the major childhood infectious and parasitic diseases or of an acute respiratory infection. Because these diseases are complications or other diseases, interpretation of their decline is difficult. It is not possible to determine if their declines are the result of declines in their initiating causes or if they simply reflect better reporting of causes of death. For example, pneumonia among young children in Italy increases from 1887-1889 to 1941-1950 while childhood infectious and parasitic diseases decline, suggesting both better reporting of pneumonia deaths and an overstatement of the decline in childhood infectious and parasitic diseases. Similarly, in the case of bronchitis, it is not possible to tell to what extent declines reflect real improvements in mortality or better reporting of chronic heart disease,

which is increasing. Penicillin, developed in 1928, is effective against both acute bronchitis and pneumonia.

Meningitis:

The U.N. Department of Social Affairs¹⁵ suggests that meningococcal meningitis may be the world's most underreported and poorly diagnosed diseases. Fatal attacks may be diagnosed as convulsions or meningitis tuberculosis if a careful autopsy does not follow. The only known means of preventing contraction of this disease is to maintain bodily resistance through good nutrition and avoidance of chills and to avoid contact with the sick. U.S. Public Health Officials¹⁶ suggest that overcrowding contributes to its spread. Penicillin now helps to reduce its mortality.

Summary:

The above discussion suggests that improvements in nutrition and environmental sanitation may well account for a high proportion of the decline in mortality before 1921-1930. From that time on, improvements in medical technology may become progressively more important.

Evidence of Socio-economic and Environmental Changes
in Italy in Each Decade

1887-1889 to 1895-1900:

Nutritional evidence presented in Figures 3 and 4 does not allow a definite conclusion as to whether average nutrition was improving in Italy during this time period. Applying the FAO protein and calorie standards for Southern Europe,¹⁷ Figures 3 and 4 indicate that Italians consumed, on the average, sufficient total and animal proteins, but insufficient calories, in the decades of the 1880's and 1890's. Further, there was no apparent improvement over these decades, although a previous increase in average consumption of animal proteins in the 1860's and 1870's might account for reductions in mortality in the 1880's and 1890's.

Figure 5 indicates that there was also no increase in per capita income during these decades. Thus, any mortality improvements which required increased expenditures must have come about through personal or government decisions to spend more money on measures to improve health and less money on some other goods and services. There is evidence of one expanding government program which was partially funded by the individuals who benefitted: an environmental sanitation program. During this period, the goal was to provide all cities with populations of 15,000 or more with sanitary drinking water.¹⁸ Some of the funds were in the form of low interest loans and others were

FIGURE 3

ITALY, PER CAPITA DAILY PROTEIN CONSUMPTION, DECADENAL AVERAGES, 1861-1870 to 1951-1955

Source: ISTAT, Sommario di statistiche storiche italiane, 1861-1955,
(Rome: Istituto poligrafico dello stato), 1958, p. 233

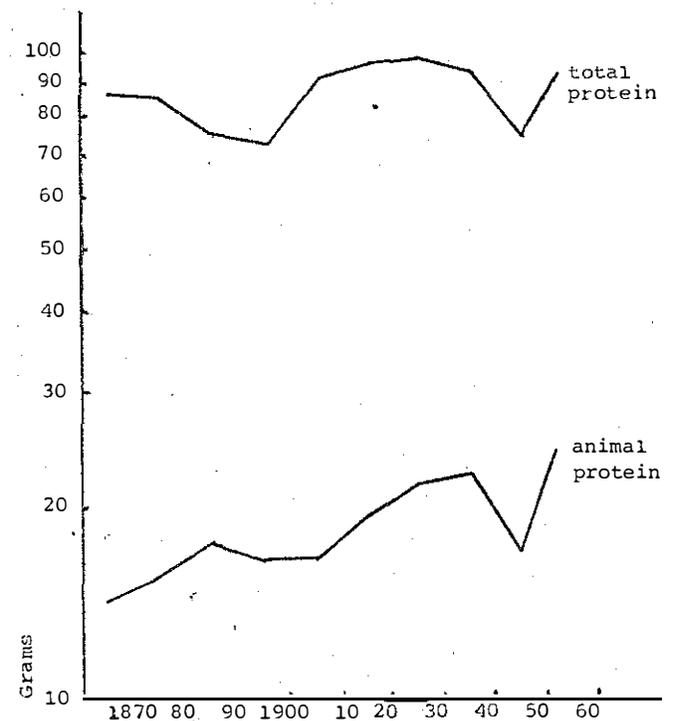


FIGURE 4

ITALY, PER CAPITA DAILY CALORIE CONSUMPTION, DECADENAL AVERAGES, 1861-1870 to 1951-1955

Source: ISTAT, Sommario, p. 233

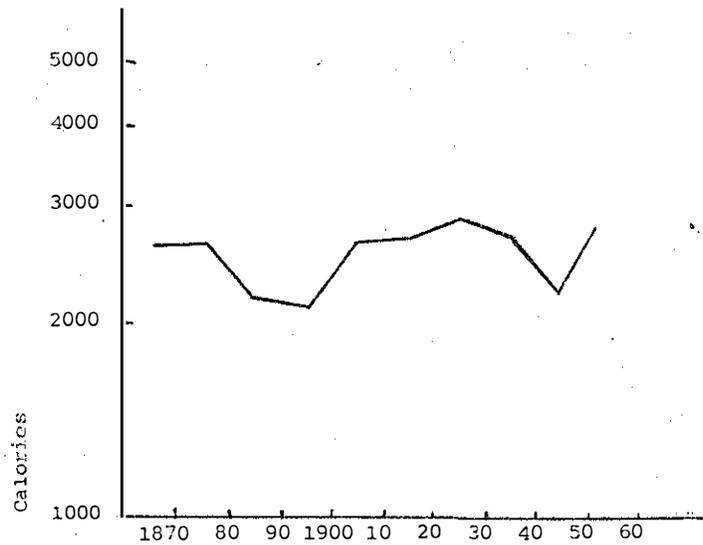
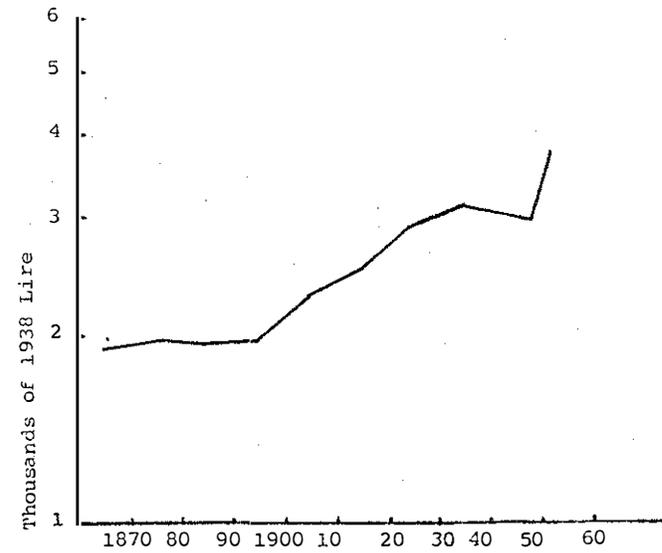


FIGURE 5

ITALY, PER CAPITA INCOME IN CONSTANT LIRE, DECADENAL AVERAGES, 1861-1870 to 1951-1955

Source: ISTAT, Sommario, p. 216



grants to the communes. This sanitation program was not extended to rural areas until the 1920's.

1895-1900 to 1901-1910:

The data presented in Figures 3 to 4 indicate marked improvement in average protein and calorie consumption and in per capita income from the 1890's to the 1900's. Average calorie consumption moved from insufficient to sufficient during this decade. This evidence combines with the fact that gastro-enteritis mortality began to decline during this second time period to suggest that there were significant nutritional improvements in this time period. Per capita income growth allowed greater expenditure on such things as education, diphtheria anti-toxin, pulmonary tuberculosis cure in sanatoriums, and malaria eradication, but evidence of changes in these expenditures is lacking.

The major change in government expenditure during this period was the establishment of the malaria eradication program. Between 1900 and 1904 the legislature enacted a series of laws making free quinine available to workers in malarial areas. In 1907 a workmen's compensation law was set up which allowed workers to claim compensation from employers if they contracted malaria. Along with the provision of quinine, the government allotted money to drain swamps to create more agricultural land so that the peasants could earn more money to build themselves better housing and purchase netting to keep out the mosquitoes.¹⁹

1901-1910 to 1921-1930:

Figures 3 and 4 indicate a slight rise in protein and calorie consumption as well as a resumption of growth in animal protein consumption during this period. Figure 5 shows continued growth in per capita income. The growth in protein and calorie consumption as well as further significant reductions in gastro-enteritis mortality suggest modest private nutritional improvements. Per capita income growth continued to allow further increases in other health care expenditures, although evidence of specific changes is still lacking.

Several changes in government expenditures may have had an impact on mortality during this period. In 1925 the government set up a national organization for mother and child welfare, one of the goals of which was to reduce infant and child mortality due to digestive diseases, primarily gastro-enteritis.²⁰

Among the mandates of the organization were to:

- provide for the protection and welfare:
 - of pregnant women and needy or abandoned children
 - of sucklings and weaned children, up to the fifth year of age belonging to families which cannot afford the necessary care for a rational bringing up
 - of children of all ages belonging to poor families....²¹

By 1949, however, there were still only 800 public health nurses in Italy, or one per 55,000 people, and these nurses were unevenly distributed in the rural areas. In general, the work of carrying out this mandate was entrusted to the government-supported midwives, of whom there was supposed to be one in each community. An organized fight to eradicate tuberculosis was also begun in the 1920's. Whenever possible, new-born infants of parents who

had tuberculosis were vaccinated with BCG. In 1927 a compulsory tuberculosis health insurance program was instituted. Workers had to contribute to the fund and received benefits for themselves and their families if they contracted tuberculosis and agreed to enter a sanatorium to be cured. Between 1926 and 1936, 40,000 sanatorium beds were provided by the government in various areas of the country and numerous tuberculosis outpatient clinics were built. In 1929 the government began to require that milk be inspected before sale.²² Between 1910 and 1920 the government set up rural bakeries to supply cheap wheat bread to maize eating communities in an effort to reduce the incidence of pellagra.²³ Finally, the water sanitation program discussed above was extended to rural areas during the 1920's.

1921-1930 to 1951-1955

Figures 3 and 4 indicate a slight decline in per capita protein and calorie consumption during this period, but the average level was still sufficient by FAO standards. Per capita animal protein consumption was stable. These data suggest that there may have been a slight decline in average nutrition during this time period. Figure 5 shows a further increase in per capita income, except during 1941-1950.

The new government programs which might have reduced mortality due those diseases whose declines contributed more than 1% to the aggregate mortality decline were a DDT malaria eradication program and compulsory diphtheria vaccination after World War II.²⁴

Summary:

The above evidence suggests that environmental sanitation was significant throughout the period under study and that nutritional improvements, both private and government supported were of major importance after 1900. Per capita income growth may have had an impact on mortality from 1900 on, and government programs to eradicate specific diseases seem to have made positive contributions in every time period.

Summary and Hypotheses

Table 2 combines the summaries of the medical, and the Italian public health and economic literature. This table and the above discussions suggest the following hypotheses regarding the socio-economic, environmental, and technological changes underlying the Italian mortality decline. From 1887-1889 to 1895-1900 environmental sanitation was the most important underlying environmental change. From 1895-1900 to 1901-1910 environmental sanitation and private purchases of nutrition and other available means of improving health share approximately equally as the most important underlying changes. From 1901-1910 to 1921-1930 public health improvements supported by the government come much more to the fore, perhaps overshadowing the importance of private purchases of the means to better health. It may be the case however, that the impact of these public health improvements

TABLE 2

SUMMARY OF SOCIO-ECONOMIC AND ENVIRONMENTAL CHANGES IN ITALY
THAT MIGHT HAVE REDUCED MORTALITY DUE TO MAJOR DISEASES:
1887-1889 to 1951-1955

| 1887- 1889 | 1895- 1900 | 1901- 1910 | 1921- 1930 | 1931- 1940 | 1941- 1950 | 1951- 1955 |
|---|---------------|---------------|---------------|---------------|---------------|---------------|
| environmental sanitation (urban) → | | | | | | |
| private nutritional improvement → | | | | | | |
| per capita income growth → | | | | | | |
| malaria eradication → | | | | | | |
| wheat bread for maize eating areas → | | | | | | |
| organization for mother and child welfare (nutrition and education) → | | | | | | |
| anti-tuberculosis society → | | | | | | |
| rural environmental sanitation → | | | | | | |
| compulsary diphtheria vaccination → | | | | | | |
| DDT → | | | | | | |
| per capita income growth → | | | | | | |

SUMMARY OF MEDICAL IMPROVEMENTS

| | |
|--|--|
| artificial pneumothorax → | |
| milk pasteurization → | |
| quinine → | |
| diphtheria anti-toxin → | |
| tuberculin test → | |
| X-ray → | |
| toxin-anti-toxin diphtheria vaccine (1913) → | |
| measles serum treatments → | |
| penicillin → | |
| toxoid diphtheria vaccine → | |
| scarlet fever anti-toxin → | |
| BCG vaccine → | |
| chemotherapy → | |
| streptomycin → | |
| rehydration → | |
| anti-malarial drugs → | |

was not really felt until the next decade 1921-1930 to 1931-1940. The same could be said of the many medical breakthroughs of the 1920's. From 1921-1930 to 1931-1940 on, medical improvements explain an increasing proportion of the mortality decline.

CONCEPTUAL FRAMEWORK AND TESTS OF HYPOTHESES

The above discussion of the medical, and public health and economic literature suggests the following conceptual framework for classifying the socio-economic, environmental, and technological changes for the purpose of testing the above hypotheses. These underlying changes might be classified according to their responsiveness to the following four kinds of changes: private decisions and expenditures; government decisions and expenditures; technological change in either medicine, public health, or home health care; and natural environmental change. Within the first two classifications, subcategories of expenditures on disease prevention and control might be:

- I. Private decisions and expenditures
 - a. private nutritional improvements
 - b. other expenditures on health care
- II. Government decisions and expenditures
 - a. government redistribution programs

- b. environmental sanitation
- c. other public health programs

Changes in expenditure on these items could come through per capita income change, changed preferences for health care or nutrition, or changes in relative prices. Technological change raises productivity at a given level of expenditure and would include all additions to knowledge about health care. Natural environmental changes are not amenable to control by man at this time.

Ideally, to test the above hypotheses we need to estimate the lives saved due to each type of socio-economic, environmental, or technological change. One possible way to estimate the impact of changes in categories one and two would be to estimate the coefficients of separate determination cross-sectionally from a series of additive functions, where independent variables would be changes in expenditure and dependent variables would be declines in sex-age-disease-specific mortality rates. Equation (3) (appendix) outlines a possible series of such functions. Technological change and natural environmental change might be treated as changing parameters. Since it is possible for mortality to decline even if one of the underlying factors does not change, the functions would be assumed to be additive. The coefficients of separate determination for the inputs and micro studies of the lives saved because of particular innovations in medical practice would provide a basis for estimating the percentage contribution of the change in each underlying factor to each sex-age-disease-specific

mortality decline. Natural environmental change might be treated as a residual.

Once the percentage contribution of each underlying change to each sex-age-disease-specific mortality decline has been estimated, the percentage contribution to the aggregate mortality decline of each underlying change operating through each sex-age-disease-specific mortality decline can be estimated by multiplying the contribution of each underlying change to each sex-age-disease-specific mortality decline by the contribution of that sex-age-disease-specific mortality decline to the aggregate mortality decline. Summing the percentage contribution to the aggregate mortality decline of each underlying change operating through each sex-age-disease-specific mortality decline over sex, age, and disease then would equal the percentage of the aggregate mortality decline explained by each underlying change (see appendix).

Unfortunately, however, sufficient data are not available at this time to test the hypotheses as described above. Assembling of data on expenditure changes by small geographical unit and micro studies of lives saved because of particular medical improvements are tasks for future research. All we can say at this point is whether or not there is a probability that a particular underlying change had an impact on a particular sex-age-disease-specific mortality decline. This knowledge, gained from the medical, and public health and economic literature, however, provides a basis for suggesting rather gross confidence intervals

can. Since it is undoubtedly the case that no one underlying factor can explain as much as 100% of the decline in any one sex-age-disease-specific mortality rate, we are left from Table 3 with the only possible conclusion being that technological change had negligible impact on mortality before 1901-1910 to 1921-1930 and played at best a secondary role in the Italian mortality transition before 1931-1940 to 1941-1950. Given the assumption about the value of penicillin, not even discovered until 1928, in explaining the mortality decline from 1901-1910 to 1921-1930, it is probably safe to conclude from Table 3 that the impact of technological change was negligible before 1921-1930.

While the above conclusions clearly cannot be construed as tests of the hypotheses developed earlier, they do lend considerable credence to those hypotheses. In the first period, 1887-1889 to 1895-1900, sanitation and private expenditures on health care have the greatest potential explanatory power. However, the absence of income growth during that decade suggests that sanitation was probably more important. From 1895-1900 to 1921-1930, private and government nutrition, sanitation, and other private expenditures on health care have the greatest potential explanatory power. From 1931-1940 to 1951-1955, technological change comes to have the same potential explanatory power as nutrition, sanitation, and other private expenditures on health care. Further credence is lent to these conclusions by the fact that they support the conclusions of the recent research on the

U.S., Great Britain, and international mortality declines.

CONCLUSION

This paper has developed hypotheses regarding the contributions of socio-economic, environmental, and technological changes to the Italian mortality decline between 1887-1889 and 1951-1955 and suggested a way that these hypotheses might be tested in the future. The hypotheses, that environmental sanitation and nutritional improvement account for most of the aggregate mortality decline in Italy before 1921-1930 and that medical improvements become progressively more important thereafter, substantially contradict earlier demographic work which stressed the role of medical improvements throughout the twentieth century, but are consistent with more recent demographic research. By extension, these hypotheses suggest that wherever diarrheal diseases have been widespread, as they were in Italy, more attention should be paid to the impact of nutritional changes on mortality.

APPENDIX

(1) sex-age-specific mortality rates and aggregate standardized mortality rates:

$$R_{dtis} = \frac{\sum_{s=1}^2 \sum_{i=1}^n \frac{(d_{tis})(Y_i)}{P_{tis}}}{P_{tis}}, \text{ where}$$

d = deaths

t = time

P = population

i = age group

s = sex

Y = number of years in each age group

n = number of age groups; and

R_{dtis} = aggregate sex-age-standardized mortality rate in time t.

(2) percentage contributions of declines in sex-age-disease-specific mortality rates to the aggregate mortality decline:

$$C_{d[t-(t+1)]isj} = \frac{[[\frac{(d_{tisj})(Y_i)}{P_{tis}}] - [\frac{(d_{(t+1)isj})(Y_i)}{P_{(t+1)is}}]]}{R_{dtis} - R_{d(t+1)is}} 100,$$

where new variables

j = disease group; and

$C_{d[t-(t+1)]isj}$ = the percentage of the aggregate mortality decline from t to t+1 explained by the decline in sex-age-disease group isj from t to t+1.

$\sum_{s=1}^2 \sum_{i=1}^n \sum_{j=1}^m C_{d[t-(t+1)]isj} = 1.0$, where new variable

m = number of disease groups.

$$\sum_{j=1}^m \frac{(d_{tisj})(Y_i)}{P_{tis}} = \frac{(d_{tis})(Y_i)}{P_{tis}}$$

(3) functions for estimating the percentage contributions of underlying changes to sex-age-disease-specific mortality declines:

$$\frac{(d_{tisj})(Y_i)}{P_{tis}} - \frac{(d_{(t+1)isj})(Y_i)}{P_{(t+1)is}} = f(\Delta PN, \Delta OP, \Delta GR, \Delta GS, \Delta PH; \text{tech}; NE),$$

where new variables

PN = private expenditures on nutrition;

OP = other private expenditures on health care;

GR = government expenditures on redistribution programs

to improve health and nutrition;

GS = government expenditures on environmental sanitation;

PH = government expenditures on other public health

programs;

tech = technological change from t to t+1; and

NE = natural environmental change from t to t+1.

Expansion of these functions might include the addition of second order interaction terms among types of expenditures as well.

(4) estimating the percentage contributions of underlying changes to the aggregate mortality decline:

$C_{[t-(t+1)]isjk} = (C_{d[t-(t+1)]isj}) (V_{[t-(t+1)]isjk})$, where new variables

k = socio-economic, environmental, or technological change;

$V_{[t-(t+1)]isjk}$ = the percentage of the decline in mortality of sex-age-disease group isj from t to $t+1$ explained by socio-economic, environmental, or technological change k from t to $t+1$ (estimated from the coefficients of separate determination for the inputs and micro studies of the lives saved because of particular innovations in medical practice. Natural environmental change might be treated as a residual);

$C_{[t-(t+1)]isjk}$ = the percentage of the aggregate mortality decline from t to $t+1$ explained by socio-economic, or technological environmental/change k from t to $t+1$, operating through the decline in mortality of sex-age-disease group isj .

$\sum_{s=1}^2 \sum_{i=1}^n \sum_{j=1}^m C_{[t-(t+1)]isjk}$ = the percentage of the aggregate mortality decline from t to $t+1$ explained by socio-economic, environmental, or technological change k from t to $t+1$.

FOOTNOTES

*I especially would like to thank William H. Newell for his untiring help at every step in the research and writing of this paper. Were it not for the obvious errors that are my own I should credit him with joint authorship. I would also like to thank John D. Durand and Richard A. Easterlin for their help and criticism during the dissertation stage; and Robert Higgs and T. Paul Schultz for their comments on earlier drafts. This paper was presented in an earlier draft at the Population Association of America Annual Meetings, April, 1975.

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