Mortality from congenital anomalies

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Abstract

Research has been conducted on the relationship between postnatal mortality from dominant causes and age, using data from the US, Japan and former Czechoslovakia, during the period 1979–1991. The logarithm of a total mortality, total mortality excluding deaths caused by accidents, logarithm of mortality caused by congenital anomalies, logarithm of mortality caused by other respiratory diseases and logarithm of mortality caused by diseases of the nervous system fell linearly with the logarithm of age, during the interval of 0–10 years. Congenital anomalies showed a log–log mortality experience that was linear until the age of 45, in some populations. This linear log–log dependence corresponds to the two-parametrical Weibull distribution, if the slope is greater than \(-1\). However, the logarithm of mortality from congenital anomalies declined with the slope equal to \(-1\). In those congenital anomalies with the slope equal to \(-1\), the logarithm of number of deaths in 1 year declined with the logarithm of age (that quantity corresponds to the density of defects in the theory of reliability). Consequently, there was applied another distribution function described in this paper. The number of deaths due to congenital anomalies over the period of 1 year was inversely proportional to the age at death. A hypothesis that the individual has his/her span of life strictly determined by the level of malformation at the moment of birth might explain the fact. A natural consequence of this hypothesis together with the facts mentioned above is as follows: the more serious the defect was, the more frequent this defect was. © 1998 Elsevier Science Ireland Ltd. All rights reserved.

Keywords: Congenital anomalies; Mortality; Aging; Distribution of probability of death

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1. Introduction

There is no doubt that any research which deals with a description of the mortality rate curves is very important, as medicine assumed as its main stimulating task to defer the moment of death for as long as possible. The total mortality rate within the whole population is the decisive criterion of that effort. It is the age of individual what affects most the mortality rate. The main part of population dies at the age for which it applies the exponential relationship is equal to 1. That relationship was recognized for the first time by Gompertz (1825). The Gompertz law was verified to be valid for several other partial causes of death (Riggs, 1990, 1991, 1992, 1993). Having excluded the deaths resulting from accidents, the relation at least applies for the population over the age of 15 and until the age of 95 years (Dolejs, 1997). Consequently, the logarithm of mortality rates grows linearly with the age in accordance with the relationship (Eq. (1)):

\[ R(t) = R(O) \cdot e^{k \cdot t} \] (1)

\( R(O) \) and \( k \) are Gompertzian parameters. The populations for which the Gompertz law applies can be considered homogeneous. To put it differently, it means that the probability of death of every individual rises along with the mortality rate rises within the total population (Strehler and Mildvan, 1960). The causes of death until the age of 10 years decidedly differ from the dominant causes of death in the period when the main part of population dies. The relation between the former causes and the age represents the subject of this paper. The total mortality of the period until 10 years falls in the course of age. A linear decrease of the logarithms of both of total mortality and some partial mortalities with the logarithm of age have been verified. The Weibull distribution function describes the linear dependence of the logarithm of mortality upon the logarithm of age for the cases with the slope greater than \( -1 \). The Weibull distribution is defined for density function in the relationship (Eq. (2)):

\[-dS(t)/dt = (m/a) \cdot t^{(m-1)} \cdot \exp(-t^m/a) \quad \text{for } t \geq 0 \] (2)

where \( m \) and \( a \) are the Weibull parameters. The quantity \(-dS(t)/dt\) is the failure density function in the theory of reliability. It is the number of deaths in 1 year from all people in the first year. \( S(t) \) is survival curve (percent living people at time \( t \) of all born people) and it is given by the relationship (Eq. (3)):

\[ S(t) = \exp(-t^m/a) \] (3)

Then the mortality rate which corresponds to the Weibull distribution function is given by the relationship (Eq. (4)):

\[ \text{for } t \geq 1 \text{ is } R(t) = R(1) \cdot t^{(m-1)} \quad -1 < (m - 1) < 0 \] (4)

with logarithm, then:

\[ \ln(R(t)) = \ln(R(1) + (m - 1) \cdot \ln(t)) \] (5)
where \( R(1) = m/a \) is the mortality rate at the age of 1 and \((m - 1)\) is the slope of the linear decrease.

The mortality reduction after the birth has two explanations:

(a) Population adapts itself to the far biggest change of conditions represented by the birth. The population is taken as a homogeneous entity and the particular reduction results from gradual improvement of resistance against dominant cause of death in this age (0–10 years).

(b) Population is a heterogeneous entity. It includes weaker individuals who are dying out and, consequently, the total mortality gets lower (Riggs, 1990; Dolejs, 1998). Observations made for non-living systems (automotives, electronic machines and equipment, buildings, etc.) proved the failure density function declines like the Weibull one immediately after the system has been put into operation, becoming constant and later growing in the course of time. Since we cannot expect an adaptation period in the case of non-living systems and the Weibull function applies, there is no need to take the adaptation period into account when explaining the Weibull function in the living systems. While it doesn’t mean there is no adaptation after the birth, the adaptation has no influence on the number of death and it is not likely to occur in the case of congenital anomalies for which the function applies even over the age of 10. The depletion of the affected individuals offers a better explanation of the reduction of mortality than the process of adaptation does. The following summary characterizes the core difference between (a) and (b): when observed from the outside point of view the population as a unit improves itself. In case (a), the individuals should also experience the process of improvement—adaptation due to which the term ‘homogeneous’ was used. Mechanism (b) says the change in state of individuals in the course of time is not important for the whole unit—population. What matters most is the diversification of individuals after the birth and a relative decline in number of the affected ones.

2. Materials and methods

The mortality rate was calculated using the WHO database (the internet address: www.who.ch/whoisis/mort/mort.htm) for men and women in the US, in Japan and in former Czechoslovakia. The database contains the numbers of living people and the numbers of death for all causes. The Basic Tabulation List from ICD9 (WHO, 1977) was used in this database for the definition of the causes of death. The mortality rate was calculated for dominant causes of death at the interval 1–10 years. The age categories 1, 2, 2, 3, 4, 5–9 and 10–14 years were used in the database. The age category 0 was not used. The individuals younger than 1 year are at this category. This category is important but unfortunately it could not be used. The mortality is extremely dependent on age at the interval 0–1 year. Therefore the expected value is somewhere at the interval 0–0.5 years (the number of death declines). The error of estimation of expected value is extreme for this category. For formal reason (the logarithm is not defined for 0 year) the value 0 could not be used as estimation of the expected value and the mortality rate was examined from the age of 1 year.
The number of death was added up in a particular age category for the entire period 1979–1991. The number of living people was calculated by the same method. The mortality rate was calculated per 1000 living people and it describes the entire period 1979–1991. The ICD9 revision coding system was valid at this period. The examined causes participate in more than 1% at the interval 1–10 years. (The spina bifida is the exception from that because its share is 0.6%). The linear area was over the age of 10 for congenital anomalies. Therefore, the mortalities from congenital anomalies were calculated for all age categories (1, 2, 3, 4, 5–9, 10–14, 15–19, 20–24, 25–29, 30–34, 35–39, 40–44, 45–49, 50–54, 55–59, 60–64, 65–69, 70–74, 75–79, 80–84 years). The averages of these intervals (7, 12, 17...82) were used for calculations of a linear parameters (I supposed that the expected value is near the average for these categories). The natural logarithm was used at this paper. The Euler number is more convenient as the base of logarithm to arrange mathematical relationships. (The slope does not depend of the base of logarithm in case both axes have the logarithmic scale).

3. Results

The linear dependence of logarithm of mortality on logarithm of age was examined for following dominant causes: (the share of particular cause on total number of death in the US in 1979–1991 up 10 years is in the brackets), all death (100%), all except accidents (44%), congenital anomalies of heart and circulatory system—ICD9: 745–747 (9%), spina bifida—ICD9: 741, 742.3 (0.6%), diseases of the nervous system ICD9: 320–359 (6.7%), other diseases of the respiratory system 466, 480–519 (4.4%). These linear dependencies are shown in Fig. 1. for men and women in the US.

The linear dependence in log–log scale was examined for men and women in the US, in Japan, in former Czechoslovakia for the period 1979–1991. The parameters of linear regression are shown in Table 1. The linear area is over the age of 10 years for congenital anomalies. The intervals for congenital anomalies were inspected visually (Figs. 2–7). The parameters of linear regression were calculated for these intervals for congenital anomalies. These parameters and the intervals are shown in Table 1.

The dependence of mortality rate on age was not confirmed for following dominant causes at the interval 1–10 years (Fig. 1) (Dolejs, 1997): accidents—ICD9: E800–E999 (56%), malignant neoplasm of lymphatic and haemopoietic tissue—ICD9: 200–208 (5.3%), malignant neoplasm of brain—ICD9: 191 (1.6%).

The slopes are near $-1$ for congenital anomalies for all populations (Table 1). The average is $-1.007$ with the standard deviation 0.077 for six populations and for two diseases (12 slopes). Furthermore, I assumed the slope is $-1$. The distribution function of the probability of death has to agree with this result. The general relationship (Eq. (6)) is valid between survival curve and mortality rate:
Table 1
Parameters of linear regression

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>USA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All without accident</td>
<td>1–10 -0.566</td>
<td>1–10 -0.669</td>
</tr>
<tr>
<td>USA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congenital anomalies of heart and circulatory system</td>
<td>1–10 -0.597</td>
<td>1–10 -0.759</td>
</tr>
<tr>
<td>USA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spina bifida and hydrocephalus</td>
<td>1–10 -0.990</td>
<td>1–10 -0.752</td>
</tr>
<tr>
<td>USA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diseases of the nervous system</td>
<td>1–10 -0.992</td>
<td>1–10 -0.752</td>
</tr>
<tr>
<td>USA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other diseases of the respiratory system</td>
<td>1–10 -0.997</td>
<td>1–10 -0.959</td>
</tr>
<tr>
<td>Japan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>1–10 -0.646</td>
<td>1–10 -0.803</td>
</tr>
<tr>
<td>Japan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All without accident</td>
<td>1–10 -0.786</td>
<td>1–10 -0.835</td>
</tr>
<tr>
<td>Poland</td>
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<td></td>
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<tr>
<td>Congenital anomalies of heart and circulatory system</td>
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<td>1–10 -0.835</td>
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<tr>
<td>Poland</td>
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<tr>
<td>Spina bifida and hydrocephalus</td>
<td>1–10 -0.999</td>
<td>1–10 -0.835</td>
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<td>Poland</td>
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<tr>
<td>Diseases of the nervous system</td>
<td>1–10 -0.992</td>
<td>1–10 -0.835</td>
</tr>
<tr>
<td>Poland</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other diseases of the respiratory system</td>
<td>1–10 -0.997</td>
<td>1–10 -0.835</td>
</tr>
<tr>
<td>Czechoslovakia</td>
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<td></td>
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<tr>
<td>All</td>
<td>1–10 -0.453</td>
<td>1–10 -0.606</td>
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<tr>
<td>Czechoslovakia</td>
<td></td>
<td></td>
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<tr>
<td>All without accident</td>
<td>1–10 -0.615</td>
<td>1–10 -0.676</td>
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<tr>
<td>Czechoslovakia</td>
<td></td>
<td></td>
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<tr>
<td>Congenital anomalies of heart and circulatory system</td>
<td>1–10 -0.504</td>
<td>1–10 -0.676</td>
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<td>Czechoslovakia</td>
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<td></td>
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<tr>
<td>Spina bifida and hydrocephalus</td>
<td>1–10 -0.924</td>
<td>1–10 -0.676</td>
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<tr>
<td>Czechoslovakia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diseases of the nervous system</td>
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<td>1–10 -0.676</td>
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<tr>
<td>Czechoslovakia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other diseases of the respiratory system</td>
<td>1–10 -0.997</td>
<td>1–10 -0.979</td>
</tr>
</tbody>
</table>
\[ R(t) \equiv -\frac{dS(t)/dt}{S(t)} \] (6)

In congenital anomalies the Weibull distribution is not valid because the slope \( m - 1 \) is equal to \(-1\) and the parameter \( m \) could not be zero. The relationship (Eq. (7)) was examined for congenital anomalies:

\[ R(t) = \frac{R(1)}{t} \] (7)

where \( R(1) \) is mortality rate in the first year. In the case where we are interested in the mechanism which is responsible of the dependence (Eq. (7)), we have to know the distribution function of the probability of death. We examined the quantity that describes the congenital anomalies caused mortality against the entire population. The death caused by congenital anomaly can occur only within the subpopulation. Quantities describing the subpopulation are important with respect to the mechanism that causes the decline of congenital anomalies caused mortality after the birth. The index \( s \) is used to describe the quantities of subpopulation with congenital anomaly. \( L_s(t) \) is the number of living people inside the subpopulation,

![Fig. 1. Plot of logarithm of mortality rates from dominant causes for men in the US versus logarithm of age up to the age of 10 (1979–1991).](image-url)
Fig. 2. Plot of logarithm of actual mortality rates and logarithm of theoretical mortality rates for men in the US versus logarithm of age for the period 1979–1991.

$S_s(t)$ is the survival curve of the subpopulation, $D_s(t)$ is the number of death in year $t$ from congenital anomaly and $R_s(t)$ is the mortality rate inside the subpopulation (as calculated using the number of living people inside the subpopulation at age $t$). The number of living people inside the subpopulation is given by Eq. (8):

Fig. 3. Plot of logarithm of actual mortality rates and logarithm of theoretical mortality rates for women in the US versus logarithm of age for the period 1979–1991.
Fig. 4. Plot of logarithm of actual mortality rates and logarithm of theoretical mortality rates for men in Japan versus logarithm of age for the period 1979–1991.

\[ L_s(t) = L(1) \cdot S_s(t) \cdot p \]  

(8)

where \( p \) is the proportional size of the subpopulation at the age of 1 and \( L(1) \) is the number of all living people at the age of 1. The number of death \( D_s(t) \) from congenital anomaly in year \( t \) is given by Eq. (9):

\[ D_s(t) = R_s(t) \cdot L_s(t) = R_s(t) \cdot S_s(t) \cdot p \cdot L(1) \]  

(9)

Fig. 5. Plot of logarithm of actual mortality rates and logarithm of theoretical mortality rates for women in Japan versus logarithm of age for the period 1979–1991.
The number of all living people is given by Eq. (10):  

$$L(t) = L(1) \cdot S(t)$$  \hspace{1cm} (10)
The mortality rate of congenital anomaly in year $t$, $R(t)$ as calculated against entire population is defined as $D_s(t)$ over $L(t)$. The mortality $R(t)$ is given by Eq. (11):

$$R(t) \equiv \frac{D_s}{L} = \frac{L(1)pS_sR_s}{L(1)S} = \frac{p}{S} \left( -\frac{dS_s}{dt} \right)$$

(11)

This relationship follows from the definitions of these quantities. The relationship (Eq. (7)) is valid simultaneously for the mortality $R(t)$ (This fact was examined). If the size of the entire population does not suffer any substantial change (situation after the birth), the survival curve of all population $S(t)$ is practically equal to 1.

We examined that the numerator in Eq. (11) is inversely proportional to the age. From the relationship (Eq. (6)), it follows that the numerator in Eq. (11) is equal to $-p \cdot S_s(1)/dt$. It is number of death in 1 year from all people with congenital anomaly in the first year.

In the case of a subpopulation, the examined quantity does not correspond with the mortality rate $R_s(t)$, but it does so with a failure density function $dS_s/(1)dt$. The examined relationship was the distribution (Eq. (12)):

$$-\frac{dS_s}{dt} = \frac{(R(1)/p) \cdot 1}{t} \equiv \frac{R_s(1)}{t}$$

(12)

The survival curve inside the subpopulation $S_s(t)$ we obtain as the integral of Eq. (12). The relationship (Eq. (13)) was deduced from Eq. (12) with condition $S(1) \equiv 1$:

$$S_s(t) = 1 - R_s(1) \cdot \ln(t)$$

(13)

where $t_{\text{max}} \geq t \geq 1$; $t_{\text{max}} = e^{1/R_s(1)}$ and $R_s(1)$ is mortality rate from congenital anomaly as calculated inside the subpopulation in the first year. The distribution (Eq. (12)) has only one parameter $R_s(1)$. The quantity $S_s(t)$ is equal zero at the year $t_{\text{max}}$. The main difference from the Weibull distribution is that the $S_s(t)$ curve is not exponential (Eq. (3)) but it is given by the relationship (Eq. (13)). The cumulative distribution function of this distribution $(1 - S_s(t))$ is equal to $R_s(1) \cdot \ln(t)$.

Eq. (12) describes the number of death in 1 year $t$ divided by number of all people with congenital anomaly in the first year. This distribution can be interpreted as if the level of malformation determines the lifetime. The number of death from congenital anomaly at year $t$ corresponds to the number of people with definite level of malformation at the moment of birth. The death is doubtless a stochastic process. In the case of congenital anomalies, the degree of chance is not enforced. The lifetime is predominate with a uncertainty. The uncertainty is not observable in the case where it is smaller than the interval of the age category (1 year up the age of 4 and 5 years for older people).

The mortality from congenital anomaly was calculated theoretically using the relationships (Eqs. (11) and (12)). The resultant theoretical curve is given by Eq. (14):

$$R(t) = \frac{p}{S(t)} \frac{R_s(1)}{t} \equiv \frac{R(1)}{S(t)} \frac{1}{t}$$

(14)

The actual value $R(1)$ was used to fit the data. This parameter was calculated as the number of death from congenital anomaly in the first year over all living people at
the age of 1. This parameter is shown in Table 1 for all populations. The actual survival curves $S(t)$ were calculated for all populations for the period 1979–1991. The theoretical curves were calculated using the parameter $R(1)$ and the actual curves $S(t)$. The theoretical curves educed from the distribution (Eq. (12)) and the actual mortality rate from congenital anomalies are in Figs. 2–7. In a case where the depletion of the entire population is significant if compared with the subpopulation, the theoretical curves increased. The phenomenon is demonstrated for higher age categories and it is caused by decreasing of the quantity $S(t)$ in the relationship (Eq. (14)). The theoretical curves capture the actual mortality rate for spina bifida and hydrocephalus in the US (Figs. 2 and 3). The theoretical curves fit the data for all age categories even the mortality curves are increasing.

In the case where the subpopulation is small, the subpopulation is depleted faster than all population. This situation is shown in Figs. 4 and 5 for spina bifida and hydrocephalus in Japan and for both congenital anomalies in former Czechoslovakia in Figs. 6 and 7. We can state that the actual mortality curves from congenital anomalies of heart and circulatory system increase for the US and Japan before the theoretical ones (Figs. 2–5).

4. Discussion

There is no doubt about the statement the death caused by congenital anomalies can occur only within the subpopulation characterized by this particular congenital anomaly. The subpopulation is defined already after the birth. If the size of the entire population does not suffer any substantial change (situation after the birth), changes within the survival curve of the entire population $S(t)$ can be ignored. Consequently, the value of $S(t)$ equals 1. Quantities describing the subpopulation are important with respect to the mechanism that causes the decline of congenital anomalies caused mortality after the birth. We examined the quantity that describes the congenital anomalies caused mortality calculated against the entire population. In the case of a subpopulation, the examined quantity doesn’t correspond with the mortality inside subpopulation, but it does so with a relative number of deaths in a time unit, i.e. with a failure density function. In other words, if the death of congenital anomalies can only occur within a relatively small group, the mortality rate within that group does not correspond with the Weibull function (This statement does not depend of the fact that the slope is equal $-1$).

Regarding the character of congenital anomalies and the similarity between the mortality rate dependence on age under 10 years and the non-living systems failure curve, an adaptation has not been considered. In our case, the adaptation describes a process that would be carried out by majority of affected organisms, reducing the probability of death of any particular individual. To explain the mortality rates decline, the extinction of different subpopulations was tested for both constant and the Gompertzian decline of mortality rate. The resulting theoretical mortality rates calculated from those models turned to fall more slowly, when compared with the actual dependence (the mortality is inversely proportional to the age). Fig. 8
Fig. 8. Plot of logarithm of actual mortality rates and logarithm of theoretical mortality rate decline caused by the extinction of a subpopulation with constant mortality.

demonstrates the mortality rate decline caused by the extinction of a subpopulation with constant mortality.

Another test of the congenital anomalies mortality decline was based on the assumption the individual mortality rate is constant in time, on condition that the subpopulation is divided into several categories, each with a different constant mortality rate. The individual constant mortality rate corresponds with the level of malformation of an individual. Neither that model fitted the actual curves. They (the curves) correspond well if we assume Eq. (12) distribution is valid. The theoretical equations for men and women in the US (Eq. (14)) fit the actual data for all age categories for spina bifida (Figs. 2 and 3). The actual mortality from congenital anomalies of heart and circulatory system over the age of 15 exceeds the theoretical one, in the US (corresponding to the distribution: Eq. (12)). In case of men and women in Japan, the difference between the actual mortality from congenital anomalies of heart and circulatory system and the theoretical one is not so big (Figs. 4 and 5). Eq. (14) did not apply for some death of congenital anomalies of heart and circulatory system in case of elderly people in the US and, partially, in Japan, too. In all other cases of higher age categories, the actual mortality rate falls more quickly than the theoretical one, due to the depletion of individuals of the subpopulation (the subpopulation with congenital anomalies) (Figs. 4–7). The individuals with congenital anomaly are not very likely to live until the age of 60 years and the cases are only found within a large population. Over the period 1979–1991, that condition was met only in the US for both congenital anomalies, and in Japan for congenital anomalies of heart and circulatory system.

Eq. (12) defines the number of deaths in 1 year dependent on age divided by the number of individuals with congenital anomaly. The logarithm of congenital anomalies mortality declines along with the logarithm of age with the slope equal
to $-1$. No probability distribution function is known which would correspond with the described function. If an adaptation does not take place, the mortality rate decline is a consequence of depletion of the affected individuals. In case of spina bifida, several levels of malformation can be identified. The distribution described by Eq. (12) is understood as if the level of malformation determines the lifetime (the variance of predetermination can be 5 years for the age over 4 years). Having accepted that interpretation, it follows that the more affected the individuals are the more is present among those who are being born (see Eq. (12)). That interpretation is backed by the fact that the number of spontaneous abortions caused by a developmental disability gets smaller within the time of pregnancy. The level of the developmental disability decreases throughout the pregnancy, too (Boue et al., 1975). Only 50% of conceptions are estimated to develop into a birth of a living child (Roberts and Lowe, 1975). Spontaneous abortions exercise large elimination of developmental disabilities in the course of pregnancy. The decline of mortality from congenital anomalies after the birth seems to be a continuation of that process.

In case of other respiratory diseases (as classified by ICD9—466, 480–519), the logarithm of mortality declines proportionally to the logarithm of age, with the slope equal to $-1$. An average of six slopes from Table 1 was calculated for the six observed populations and for other respiratory diseases. The average equals $-0.989$ with a standard deviation of 0.023. Regarding a marked similarity between the congenital anomalies mortality decline and the figures of respiratory diseases...
mortality, the conclusions derived from both observations seem to be identical. In
the case of death occurred through the age of 10 years where the distribution (Eq.
(12)) applies the following general conclusion can be made—the death resulted
from the state of organism immediately after the birth. The casualty consisted only
in the very moment of death, for which there was a tolerance of 1 year in the case
of death before the age of 4, and 5 years in the case of death over the age of 4.

Logarithm of the total mortality along with the age logarithm declines in a
slower pace when compared to the logarithm of all death excluding accidents (see
Fig. 1). The result reflects the fact that accident caused mortality is in fact
independent of age. If we add accidents to the other causes of death, the mortality
rate decline will slow down. The effect is demonstrated in Fig. 9.

A situation similar to the previous one could occur in the case of nervous system
diseases mortality (ICD9 revision: 320–359). The slope for the nervous system
diseases is also greater than $-1$ (mortality rate falls more slowly). The case cannot
be ruled out that the set of deaths includes some causes independent of age. The
mortality rate decline of the previous category could correspond with the distribu-
tion (Eq. (12)) too. By adding up two types of the mortality rate dependence on
age, we get a theoretical explanation of every Weibull function (mortality rate
decline) with the slope greater than $-1$. The assumption cannot be verified in the
case of nervous system disease as the data describing the number of death cannot
be further divided into more detailed categories.

An explanation cannot be ruled out that the Weibull decline of failure curve in
case of non-living systems is defined by the sum of time independent values of the
failure curve and the distribution (Eq. (12)).

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