

Where Is the Level of the Mortality Plateau?

Roland Rau

University of Rostock & Max Planck Institute for Demographic Research

Marcus Ebeling

University of Rostock & Max Planck Institute for Demographic Research

Frederik Peters

University of Rostock & Max Planck Institute for Demographic Research

Christina Bohk-Ewald

University of Rostock & Max Planck Institute for Demographic Research

Trifon I. Missov

University of Rostock & Max Planck Institute for Demographic Research

Presented at the Living to 100 Symposium
Orlando, Fla.
January 4–6, 2017

Copyright © 2017 by the Society of Actuaries.

All rights reserved by the Society of Actuaries. Permission is granted to make brief excerpts for a published review. Permission is also granted to make limited numbers of copies of items in this monograph for personal, internal, classroom or other instructional use, on condition that the foregoing copyright notice is used so as to give reasonable notice of the Society's copyright. This consent for free limited copying without prior consent of the Society does not extend to making copies for general distribution, for advertising or promotional purposes, for inclusion in new collective works or for resale.

Where Is the Level of the Mortality Plateau?

Roland Rau Marcus Ebeling Frederik Peters

Christina Bohk-Ewald Trifon I. Missov

University of Rostock & Max Planck Institute for Demographic Research*

Using data from the “International Database on Longevity”, Gampe (2010) found a plateau of mortality — which is implied in a logistic model — at ages 110 and above. In her study, Gampe pursued a nonparametric approach. Hence this plateau was not the consequence of forcing a certain parametric shape of the mortality trajectory. Gampe estimated a level of the force of mortality of about $\mu_{110+} = 0.7$, corresponding to a probability of dying of approximately 0.5. Gampe’s results strengthened the initial findings that were reported at the “Living to 100” conference in 2005 (Robine et al., 2005).

Our research question can be expressed simply as: Can we find support for this upper limit of the force of mortality of 0.7, i.e. the chance to survive another year is approximately the chance of tossing a fair coin?

1 Introduction

Few empirical findings are as regular as the exponential increase in mortality at adult ages. From one age to the next, mortality rises with a constant rate of slightly more than

*Corresponding author: Roland Rau. University of Rostock, Chair of Demography, Ulmenstr. 69, 18057 Rostock, Germany. Email: roland.rau@uni-rostock.de

10 percent. This log-linear increase in the force of mortality, $\mu(x)$, is well approximated by Benjamin Gompertz's "law" for the force of mortality, $\mu(x) = ae^{bx}$ with age x , intercept a and slope parameter b . Most researchers agree that the rate of change decreases after about age 80 or 85, a phenomenon commonly called "mortality deceleration" (e.g., Horiuchi and Wilmoth, 1998). As usual when analyzing differences in aggregate measures over time, age, region or any other dimension, there are three possible strains of explanation (Vaupel and Canudas-Romo, 2002). The first among them is problematic data. It is well known that registration of death counts is more precise than the estimates for populations at very high ages, the latter often being inflated. With the same number of events in the numerator but with a larger denominator, age-specific mortality tends to be artificially low. Alternatively, there could be an *actual* decrease in the rate of aging, potentially caused by a slowing down of metabolic process on the individual level (e.g. Ukraintseva and Yashin, 2001).

However, it is selective mortality that is most often referred to when mortality deceleration is discussed. The idea is simple: Populations are heterogeneous. Even if the risk of dying increases at the same pace for all individuals, some are probably frailer than others. Those individuals would die, on average, earlier than their more "robust" peers. With less and less frail individuals alive, the observed force of mortality for the total population tends to level off. The simplest so-called frailty model that can explain the leveling off is probably the one introduced by Vaupel et al. (1979), where the baseline mortality is multiplied for individual i by a factor z_i that follows a Γ distribution with a mean of 1. Vaupel and Yashin (1985) and Vaupel (2010) contain more intuitive illustrations than the more technical initial paper from 1979. A thorough discussion of other mixing distributions than the Γ for univariate frailty models and of multivariate frailty models can be found in Wienke (2010).

But what will be the shape of the trajectory at even higher ages when data become very sparse? Will mortality continue to rise, albeit at a slower pace than at younger ages?

Will it reach a maximum? Using various parametric models, Thatcher et al. (1998) concluded that a model with a logistic shape¹ fitted the data best. Also Thatcher's analysis in 1999 concluded that such a model with a mortality plateau should be employed for the estimation of mortality at advanced ages. In other applications of the logistic model, such an asymptotic maximum is called "carrying capacity."

Intensive research during the past decade supported the notion of such a logistic pattern. Theoretical, mathematical and empirical studies point toward a "Gompertzian" age-specific hazard on the individual level with a gamma distribution for frailty across individuals (e.g., Finkelstein and Esaulova, 2006; Missov and Finkelstein, 2011; Steinsaltz and Wachter, 2006). The findings of the past 10 years are succinctly summarized in Missov and Vaupel (2015, p. 69):

The observed leveling-off of human mortality rates at ages 110+ has four major implications for the generating mechanism. First, plateaus can be modeled in the framework of multiplicative (proportional) or additive hazards, but not by accelerated failure time models. Second, the distribution of unobserved heterogeneity has a regularly-varying-at-zero density at the starting age and converges subsequently to the gamma distribution. Third, in a proportional hazards setting the baseline cumulative hazard is the inverse of the negative logarithm of any completely positive function, and the well-known gamma-Gompertz pair can be derived as a special meaningful case. Fourth, in an additive hazards setting plateaus are generated by taking the latter proportional hazards pattern and adding a frailty-independent term that levels off with age. Many conjugate pairs, i.e., pairs of baseline mortality and frailty distributions, can produce the same mortality pattern. The only demographically meaningful multiplicative model that holds *at* the plateau is the gamma-Gompertz.

¹Either a general logistic model or a simplified version, the Kannisto model. The quadratic model was excluded, since it would lead, ultimately, to a force of mortality of 0.

Using data from the International Database on Longevity,² Gampe (2010) found, indeed, a plateau of mortality—which is implied in a logistic model—at ages 110 and above. In her study, Gampe pursued a nonparametric approach. Hence this plateau was *not* the consequence of forcing a certain parametric shape of the mortality trajectory. Gampe estimated a level of the force of mortality of $\mu(x)_{110+} = 0.7$, corresponding to a probability of dying of $q(x)_{110+} = 1 - e^{-\mu(x)_{110+}} \approx 0.50$. Gampe’s results strengthened the initial findings that were reported at the “Living to 100” conference in 2005 (Robine et al., 2005).

Our research question can be expressed simply as: Can we find support for this upper limit of the force of mortality of 0.7, corresponding to a probability to survive another year of approximately tossing a fair coin?

2 Data and Methods

All our estimates are based on data from the Human Mortality Database (2016). Following the standard literature (e.g. Brillinger, 1986; Carstensen, 2007; Keiding, 1990), we estimated our parametric models in a maximum-likelihood framework assuming that age-specific deaths $D(x) \sim \text{Poisson}(\bar{\mu}(x)N(x))$, where $N(x)$ denotes the number of person-years lived (= exposure time) at age x . The log-likelihood for parameter vector θ that needs to be optimized given the data is:

$$\log -\mathcal{L}(\theta|D(x), N(x)) = \sum_{x=\alpha}^{\beta} [D(x) \log(\bar{\mu}(x)) - \bar{\mu}(x)N(x)]. \quad (1)$$

We typically looked at ages $\alpha = 80$ until $\beta = 109$. In our application, the population level hazard $\bar{\mu}(x)$ in Equation 1 was substituted by the respective hazard function. Our main model was the gamma-Gompertz model that is commonly expressed as (see, for instance Missov and Vaupel, 2015, p. 674):

²This database is available from the Max Planck Institute for Demographic Research at <http://www.supercentenarians.org/>.

$$\bar{\mu}(x) = \frac{ae^{bx}}{1 + \frac{a\gamma}{b}(e^{bx} - 1)} \quad (2)$$

with intercept a and slope b from the Gompertz distributions and the variance of the gamma distribution γ . Thus, the parameter vector θ that needs to be optimized consists of three elements (a, b, γ) .

For reasons of better numerical stability when fitting the data, we used the alternative parametrization via the Gompertz modal age at death M (see, for instance, Missov and Vaupel, 2015):

$$\bar{\mu}(x) = \frac{be^{b(x-M)}}{1 + \gamma e^{-bM}(e^{bx} - 1)} \quad (3)$$

The mortality plateau for this model can be expressed as $\lim_{x \rightarrow \infty} \bar{\mu}(x) = \bar{\mu}^* = b/\gamma$.

3 Results

3.1 Selection of Models

Before we settled on the model of Equation 2 (or Eq. 3), we also estimated alternative models to make sure that the gamma-Gompertz approach is the best model. The simplest model is the pure Gompertz model. It can be expressed in the traditional way and via the mode, which we used in the actual estimation (see Horiuchi et al., 2013, also for the reparametrization of the other models via the mode).

$$\text{traditional : } \bar{\mu}(x) = ae^{bx} \quad \text{via mode : } \bar{\mu}(x) = be^{b(x-M)}. \quad (4)$$

William Makeham (1867) added an age-independent constant to account for a nonsenescent component ("background" mortality):

$$\text{traditional : } \bar{\mu}(x) = ae^{bx} + c \quad \text{via mode : } \bar{\mu}(x) = be^{b(x-M)} + c. \quad (5)$$

Likewise, the gamma-Gompertz model of Equations 2 and 3 can also be extended via a Makeham term:

$$\text{traditional : } \bar{\mu}(x) = \frac{ae^{bx}}{1 + \frac{a\gamma}{b}(e^{bx} - 1)} + c \quad \text{via mode : } \bar{\mu}(x) = \frac{be^{b(x-M)}}{1 + \gamma e^{-bM}(e^{bx} - 1)} + c. \quad (6)$$

Please note that models 4 and 5 do not imply a mortality plateau. Mortality in the Gompertz and Gompertz-Makeham scenarios continues to rise exponentially. Although the overwhelming majority of researchers provide evidence for mortality deceleration, Gavrilov and Gavrilova (2011) argue that there are no signs of mortality deceleration (in their data set for the United States). That is why we have included models 4 and 5.

The best model was selected based on the minimum AIC value for each of the four models. The criterion was estimated using the standard approach as outlined by Akaike (1974, p. 716):

$$\text{AIC} = (-2) \log(\text{maximum likelihood}) + 2(\text{number of independently adjusted parameters within the model})$$

The results for the years 1980–2010 for a selection of seven large countries, separately for women and men, are shown in Figure 1.

The predominant color is green, indicating that in 340 out of the 434 separately estimated models ($\approx 78\%$), the gamma-Gompertz is better suited than the other models. The gamma-Gompertz-Makeham model is the best model in 21% of all cases. But if we had dropped the United States from our sample, a country with weak data quality at those ages, according to Jdanov et al. (2008), this number would drop to 15%. With the Gom-

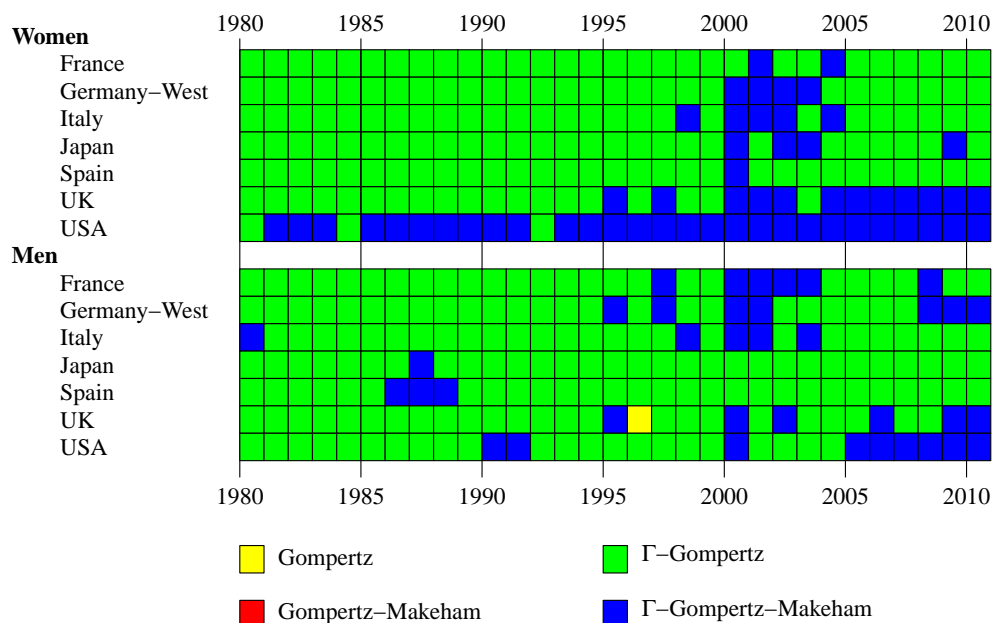


Figure 1: Best Models When Comparing Gompertz, Gompertz-Makeham, Gamma-Gompertz and Gamma-Gompertz-Makeham Models Note: For seven large countries, 1980–2010, separately for women and men. Source: Own estimations based on data from the Human Mortality Database (2016).

pertz model being best in the United Kingdom in 1996, there is only one case where the data supported a model without deceleration and, therefore, without a plateau.

3.2 Graphical Fit of the Gamma-Gompertz Model

Selecting the best out of a variety of models does not guarantee in any way that the model fits the data well, regardless of the selection criterion (AIC, BIC, r^2 , ...). As a simple example: A quadratic model is (necessarily) at least as close to the data as a straight line in linear regression. But it might not capture the essential features of the data to be fitted (e.g., a third-order polynomial). Likewise, we need to check that the gamma-Gompertz model is not only the best of the four models but also a close fit to the data. We did this on a visual basis: The blue lines in the six panels of Figure 2 show, for 2010 data for females

from Japan, Spain, Sweden, France, Germany and Italy, how extremely well the selected gamma-Gompertz model actually fits the observed death rates (plotted with plus signs). Hence, we cannot expect that our estimates for $\bar{\mu}^*$ are the spurious outcome of a poor model choice. In each graph, the green horizontal line denotes the value of 0.7, which we would have anticipated from Gampe (2010). The red horizontal line depicts the level of the mortality that derived from the parameter estimates ($\hat{b}/\hat{\gamma}$, see Missov and Vaupel, 2015). With the exception of Spain (upper right panel), our estimates suggest a mortality plateau that is slightly higher than the expected 0.7 but lower than 0.8.

3.3 Estimates of the Mortality Plateau

Having identified the gamma-Gompertz model as providing a good fit to the data and being statistically superior to the other models according to the AIC, we next estimated the mortality plateau for Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany (West), Italy, Japan, Norway, Spain, Sweden, Switzerland, the United Kingdom and the United States. The empirical discrete density function for all years and countries for women and men is depicted in Figure 3 with gray vertical bars. According to our estimates, the actual level of the mortality plateau would be higher than 0.7. It would be slightly below a population hazard level of 1, which can be translated to a probability of dying of $1 - \exp(-1) = 0.6321206 \approx 63\%$. The red and blue lines depict estimated densities for females and males, respectively. These results suggest that the median of the estimated mortality plateaus is lower for women than for men. The median estimate for the plateau of the population hazard of males is about 1.2 or 70%. We would not claim that our results contradict Gampe's estimates: 573 out of the 637 individuals that formed the basis for Gampe's estimate of 0.7 were females (Gampe, 2010, p. 224). We would have also expected that the estimates are less dispersed. The variation in mortality plateaus for males is particularly striking.

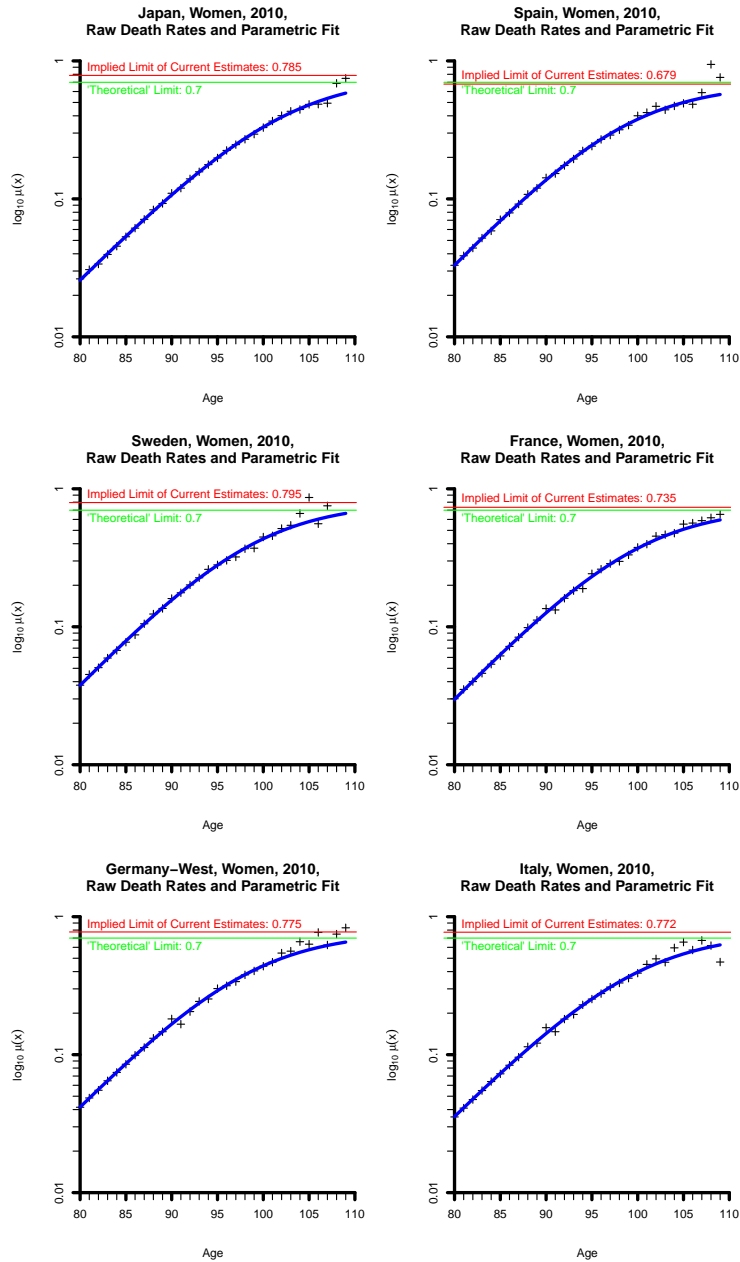


Figure 2: Raw Death Rates (+) and Fitted Force of Mortality (Blue Lines) at Ages 80–109 Note: For Japan, Spain, Sweden, France, Germany-West and Italy in 2010. Additional horizontal reference lines were included in green for Gampe’s (2010) “theoretical” limits of 0.7 and in red for the ones obtained from our estimation. Source: Own estimation and illustration based on data from the Human Mortality Database (2016).

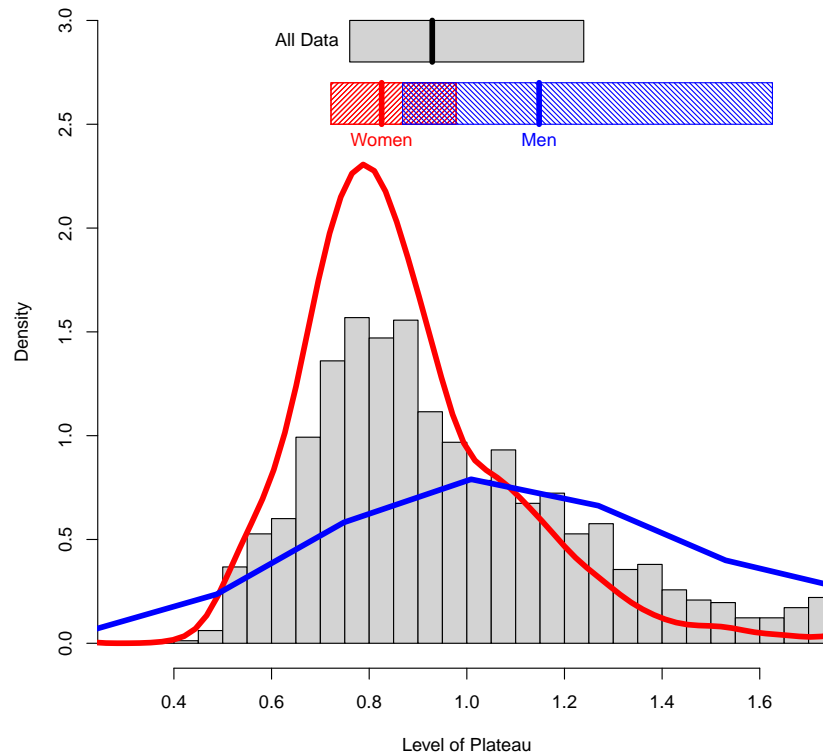


Figure 3: Empirical Density Function of Mortality Plateaus for Women and Men, 1960–2010 Note: Data for Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany (West), Italy, Japan, Norway, Spain, Sweden, Switzerland, United Kingdom and United States. Red curve is estimated density for women, and blue curve is estimated density for men for the same countries and years. Horizontal rectangles are interquartile ranges of mortality plateau for both sexes combined (gray), women (red) and men (blue). Source: Own estimation and illustration based on data from the Human Mortality Database (2016).

Therefore, we decided to simulate data with a “true” signal of $b = 0.14$ and $\gamma = 0.2$, which results in a mortality plateau of $\bar{\mu}^* = b/\gamma = 0.14/0.2 = 0.7$ but different “population sizes.” The simulations were repeated 1,000 times for each of the three population sizes of 1,000, 10,000, and 100,000 individuals. The results of the simulation are shown in

Figure 4. The median values in each of the three simulations are close to what we would have expected. We can also see that the variation around the median strongly depends on the population size. If only 1,000 individuals were alive at age 80, which is the beginning of the age interval for our estimations, “typical” values would range from about 0.58 to 0.92. The variation we have observed in our empirical data cannot be explained by small population size alone, though. Even in 1960, more than 10,000 women as well as men age 80 were alive in Sweden, one of the smallest countries we have analyzed.

That is why we analyzed the data by decade (see Figure 5) and by country (see Figure 6) separately for women and men. The results are inconclusive, however. If the mortality plateau had changed systematically from one decade to the next, we might have observed a decrease in the variation. With the exception of the last decade, we have seen a fairly stable median for women during the 40 years of analysis with little change in the variation. The lack of change in the variation is even more the case for males, as both panels of Figure 5 illustrate. Also, the analysis by country did not improve our understanding of the mortality plateau substantially. If each country had its own specific level of mortality, a large part of the variation observed in Figure 3 could have been easily explained. Unfortunately, this is not the case.

The level of the mortality plateau is the ratio of the slope parameter b and the variance of the gamma distribution γ . We investigated, therefore, whether the variation in the mortality plateau is mainly attributable to one of the two factors. Figure 7 shows a range of theoretical b values on the x -axis and of theoretical gamma values. Their ratios, i.e., the level of the respective mortality plateaus, are included as contour lines. The color images in the panels depict a two-dimensional density plot of 1,632 gamma-Gompertz estimates, which also formed the basis for Figure 3.³ Shades of red denote relatively low densities, whereas blue, purple and violet colors show the areas of many estimates being close to each other. It seems that the variation in the level of the mortality plateau is primarily

³51 calendar years for 16 countries, separately for women and men.

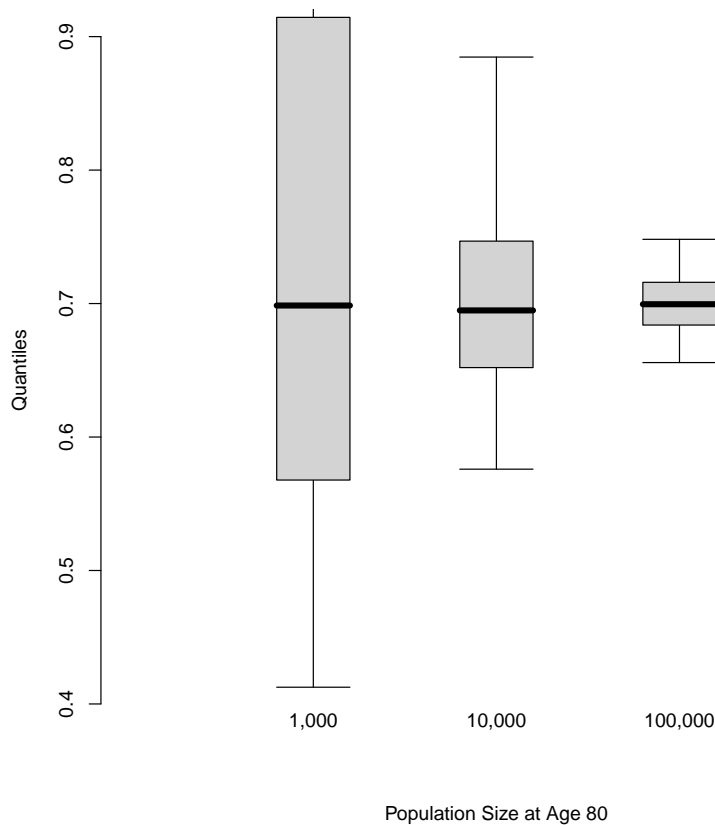


Figure 4: Boxplots of Simulation with Sample Sizes 1,000, 10,000 and 100,000 Where the “True Signal” Equals $\bar{\mu}^* = b/\gamma = 0.14/0.2 = 0.7$ Note: Simulations were repeated 1,000 times for each population size. The gray vertical bars depict the interquartile range, containing a solid black line for the median value. The “whiskers” of the boxplot represent the empirical 95% interval. Source: Own simulations.

due to variation in the estimates for γ , while the estimates for the slopes cover a much smaller range (0.10 to 0.15 for women; 0.07 to 0.12 for men).

We also analyzed another potential impact on our estimates: the numerical method employed. All our estimates were based on standard numerical methods, i.e., the Nelder-Mead algorithm, which is the default setting for R’s general purpose optimizer `optim` (R

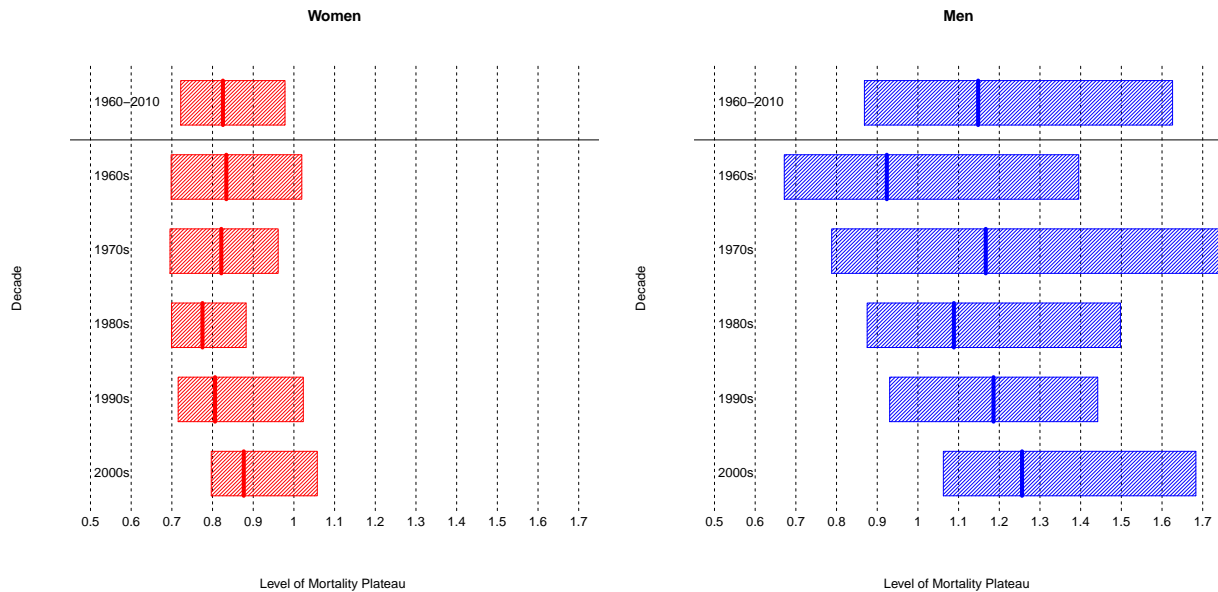


Figure 5: Interquartile Range and Median Values for Mortality Plateaus by Decade of Analysis, 1960–2010 Note: Data for women (left in red) and men (right in blue) in Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany (West), Italy, Japan, Norway, Spain, Sweden, Switzerland, United Kingdom and United States. Source: Own estimation and illustration based on data from the Human Mortality Database (2016).

Core Team, 2015). Using the same set of countries with high data quality as in Figure 2, we compared the estimates of `optim` with a genetic algorithm as implemented in the R package `rgenoud` (Mebane, Jr. and Sekhon, 2011; Sekhon and Mebane, Jr., 1998), using population sizes of 1,000 and 100 generations. We used the same starting values of $b_0 = 0.1$, $M_0 = 85$, and $\gamma_0 = 0.3$, i.e., values that are in the same order of magnitude but not too close, for `optim` as well as `rgenoud`. The results are presented in Table 1 (page 16).

It is easy to see that maximizing the log-likelihood with the standard approach or with a genetic algorithm yields estimates that are nondistinguishable. The largest difference was 0.0046 for the modal age at death M . The parameters b and γ differed by 0.00042 or

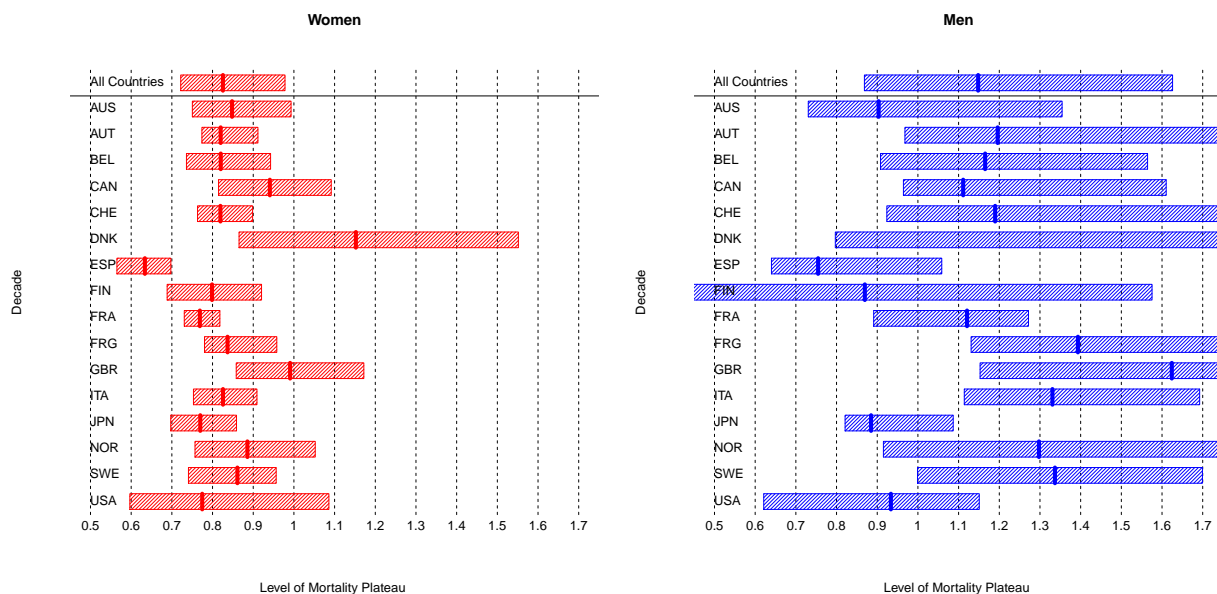


Figure 6: Interquartile Range and Median Values for Mortality Plateaus by Country, 1960–2010 Note: Data for women (left in red) and men (right in blue). Source: Own estimation and illustration based on data from the Human Mortality Database (2016).

less. Hence, the large variation in mortality plateaus cannot be attributed to non- or ill-converging numerical methods. But we discovered that with the exception of Spain, the remaining five countries estimated the variance to be close to 0.20 and also little variation for the slope.

We therefore decided to estimate the gamma-Gompertz model for countries with the best grade for data quality (Jdanov et al., 2008), with high life expectancy and a population size of at least 10 million for the years 2005–2010, to be safe to have sufficient numbers at old ages to not run into numerical problems. The final set of countries consisted of Belgium, France, Germany, West Germany, Italy and Japan. We added the estimates for b and γ for these countries in these years as white dots to the density plot. The results are given in Figure 8 (page 17). Those recent estimates for countries with low mortality, excellent data quality and large numbers suggest that there seems to be in fact a mortality plateau—

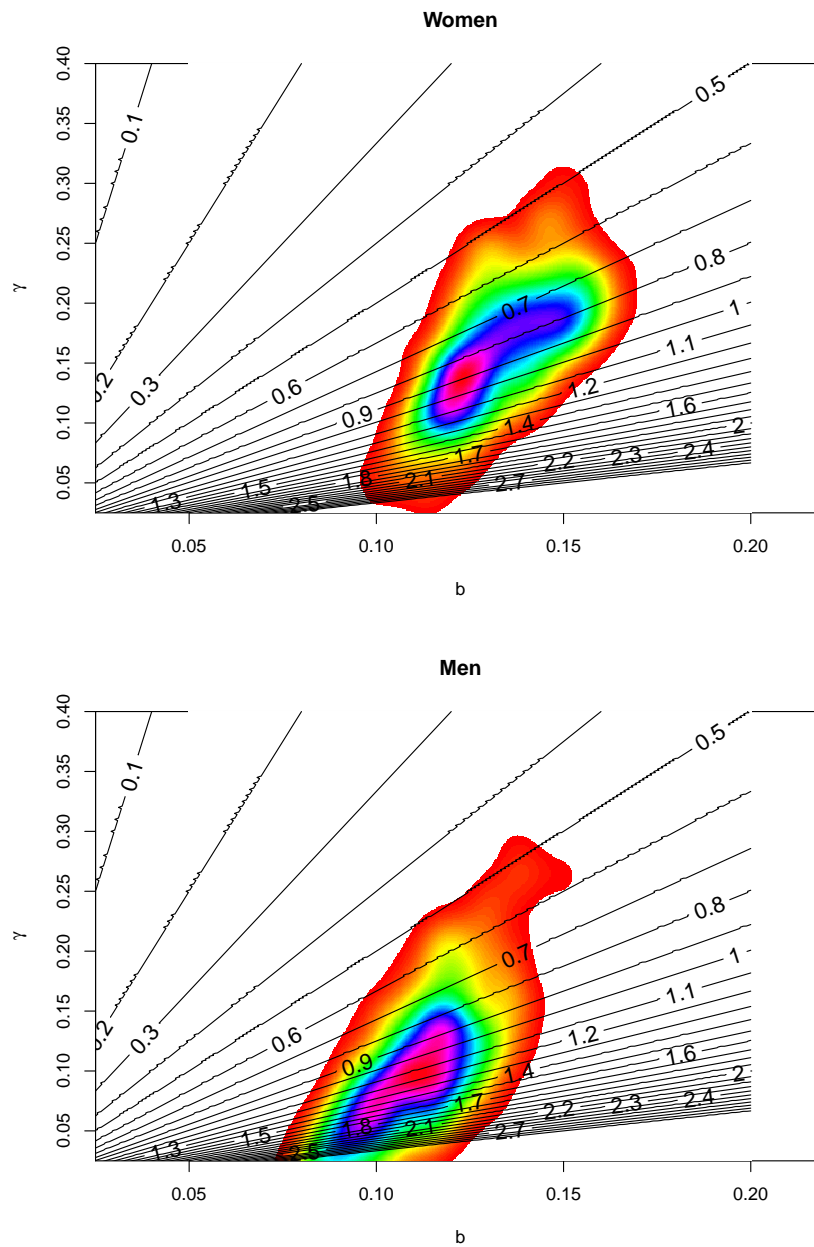


Figure 7: Two-Dimensional Density Plot of Estimates for b and γ with Corresponding Levels of the Mortality Plateau for Women and Men Note: Plateau levels shown as contour lines for women (upper panel) and men (lower panel). Source: Own estimation and illustration based on data from the Human Mortality Database (2016).

Table 1: Parameter Estimates for Gamma-Gompertz Model for Women, Using Two Different Estimation Strategies Note: Data for women in Japan, Spain, Sweden, France, Germany (West) and Italy in 2010 (see Figure 2 for actual fit). For estimation strategies, `optim` refers to standard numerical optimization in R, while `genetic` employs the genetic optimization approach of package `rgenoud` (Mebane, Jr. and Sekhon, 2011; Sekhon and Mebane, Jr., 1998) with a population size of 1,000 and 100 generations. Both approaches used the same starting values of $b = 0.1$, $M = 85$, and $\gamma = 0.3$.

Country	Estimated Parameter and Estimation Approach					
	b		M		γ	
	<code>optim</code>	<code>genetic</code>	<code>optim</code>	<code>genetic</code>	<code>optim</code>	<code>genetic</code>
Japan	0.15350	0.15365	91.40880	91.40417	0.19548	0.19680
Spain	0.16006	0.15998	89.55558	89.55333	0.23565	0.23524
Sweden	0.15942	0.15948	88.75105	88.75217	0.20049	0.20069
France	0.15914	0.15925	90.27741	90.27510	0.21657	0.21707
Germany (West)	0.15724	0.15723	88.09153	88.09380	0.20280	0.20282
Italy	0.15433	0.15428	89.20267	89.20105	0.19981	0.19957

at least for females—that is located at about 0.8 (median: 0.799; IQR: 0.775–0.829). This translates into a probability of dying of 0.55%. The picture remains blurred for males. Even the reduction to the set of countries for which we would expect highly reliable data did not result in an estimate for a mortality plateau with little variation around it. The median value for the population hazard was 1.246 (equivalent to a probability of dying of about 71%) with an interquartile range of 1.1324–1.5487, mainly due to considerable differences in the estimates for γ .

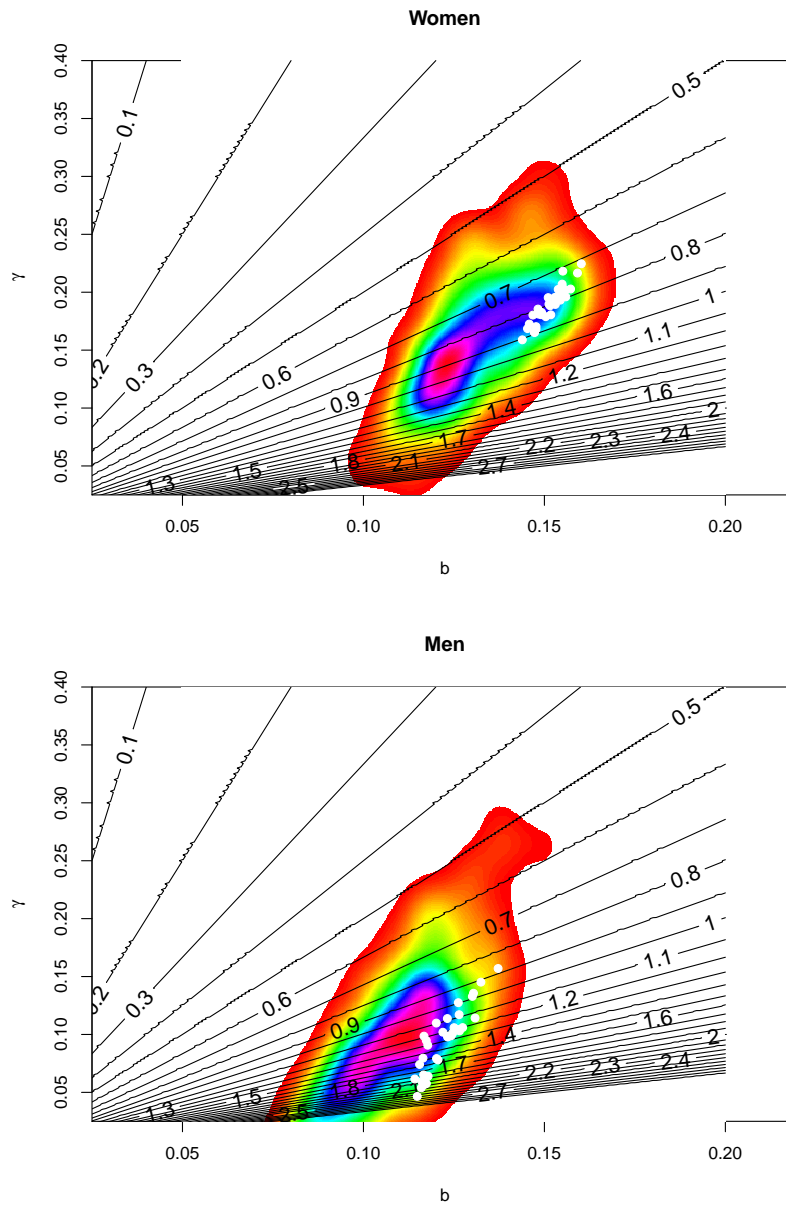


Figure 8: Two-Dimensional Density Plot of Estimates for b and γ with Corresponding Levels of the Mortality Plateau and Estimates by Country Note: Plateau levels shown as contour lines for women (upper panel) and men (lower panel). White dots represent estimates for Belgium, France, Germany, Germany-West, Italy and Japan. Source: Own estimation and illustration based on data from the Human Mortality Database (2016).

4 Summary

The goal of our analysis was to test whether we can find support for a plateau of the population hazard at a level of 0.7 as estimated by Gampe (2010) with a nonparametric approach. Recent theoretical, mathematical and statistical findings suggest that “the only demographically meaningful multiplicative model that holds *at* the plateau is the gamma-Gompertz” (Missov and Vaupel, 2015, p. 69).

We estimated these gamma-Gompertz models with data from the Human Mortality Database. With about a level for the plateau of 0.8, our results for women were slightly higher than the expected 0.7. The mortality plateau for males was even higher, though. We estimated a level of approximately 1.2. Translated into probabilities of dying, we could not replicate the chance of the toss of a fair coin but about 55% for women and 70% for men. Since Gampe’s original estimates were based on primarily female data, we were not surprised by the difference between women and men. More worrisome was the large variation in our estimates for 16 countries for the years 1960–2010. Differentiating by country or by decade did not provide any insights why there was such a large variation in mortality plateaus. Also, small population sizes were not able to explain the large variation as simulation studies have shown.

Only when we restricted ourselves to large countries (more than 10 million inhabitants), high data quality according to Jdanov et al. (2008), low mortality and recent years to have enough survivors at advanced ages were we able to narrow down the corridor for females. Our mortality plateau of 0.8 for women is slightly higher than the one predicted by Gampe (2010) with a slope parameter b of about 0.14–0.15 and a γ parameter slightly below 0.2. Our refined approach for males was less successful. Even after selecting highly reliable data only, we estimated still large variation for the mortality plateau, mainly caused by γ estimates ranging from 0.05 to 0.15.

To conclude: We would claim that there is, indeed, support for a mortality plateau for

women. At 0.8, it is slightly higher than suggested by Gampe (2010). If a mortality plateau exists for males, it is higher than for females, but our estimates were not convincing. This could be the outcome of there being still very few old men alive at very advanced ages. Hence, a mortality plateau might not exist at all, or we have to wait until enough centenarians and semi-supercenarians are male to have more robust estimates.

References

- Akaike, H., A New Look at the Statistical Model Identification. *IEEE Transactions on Automatic Control* 19(6): 716–723. 1974
- Brillinger, D.R., The Natural Variability of Vital Rates and Associated Statistics. *Biometrics* 42: 693–734. 1986
- Carstensen, B., Ageperiodcohort models for the Lexis diagram. *Statistics in Medicine* 26: 3018–3045. 2007
- Finkelstein, M. and Esaulova, V., Asymptotic behavior of a general class of mixture failure rates. *Advances in Applied Probability* 38(1): 244–262. 2006
- Gampe, J., Human mortality beyond age 110. In H. Maier, J. Gampe, B. Jeune, J.M. Robine, and J.W. Vaupel, eds., *Supercenarians*, volume 7 of *Demographic Research Monographs*, pp. 219–230, Heidelberg: Springer. 2010
- Gavrilov, L.A. and Gavrilova, N.S., Mortality measurement at advanced ages: a study of the Social Security Administration Death Master File. *North American actuarial journal* 15(3): 432–447. 2011
- Horiuchi, S., Ouellette, N., Cheung, S.L.K., and Robine, J.M., Modal age at death: lifespan indicator in the era of longevity extension. *Vienna Yearbook of Population Research* 11: 37–69. 2013

- Horiuchi, S. and Wilmoth, J.R., Deceleration in the Age Pattern of Mortality at Older Ages. *Demography* 35(4): 391–412. 1998
- Jdanov, D.A., Jasilionis, D., Soroko, E.L., Rau, R., and Vaupel, J.W., Beyond the Kannisto-Thatcher Database on Old Age Mortality: An Assessment of Data Quality at Advanced Ages. Working Paper MPIDR Working Paper WP-20083-013, Max Planck Institute for Demographic Research, Rostock, Germany. 2008
- Keiding, N., Statistical Inference in the Lexis Diagram. *Philosophical Transactions: Physical Sciences and Engineering* 332(1627): 487–509. 1990
- Makeham, W.M., On the law of mortality. *Journal of the Institute of Actuaries* 13: 325–358. 1867
- Mebane, Jr., W.R. and Sekhon, J.S., Genetic Optimization Using Derivatives: The rgenoud Package for R. *Journal of Statistical Software* 42(11): 1–26. 2011
- Missov, T. and Finkelstein, M., Admissible mixing distributions for a general class of mixture survival models with known asymptotics. *Theoretical Population Biology* 80: 64–70. 2011
- Missov, T.I. and Vaupel, J.W., Mortality Implications of Mortality Plateaus. *SIAM Review* 57(1): 61–70. 2015
- R Core Team, *R: A Language and Environment for Statistical Computing*. R Foundation for Statistical Computing, Vienna, Austria. 2015
- Robine, J., Cournil, A., Gampe, J., and Vaupel, J.W., IDL, the International Database on Longevity. In Society of Actuaries, ed., *Living to 100 and Beyond*. 2005
- Sekhon, J.S. and Mebane, Jr., W.R., Genetic Optimization Using Derivatives: Theory and Application to Nonlinear Models. *Political Analysis* 7: 189–213. 1998

- Steinsaltz, D.R. and Wachter, K.W., Understanding Mortality Deceleration and Heterogeneity. *Mathematical Population Studies* 13: 19–37. 2006
- Thatcher, A.R., The long-term pattern of adult mortality and the highest attained age. *Journal of the Royal Statistical Society* 162(1): 5–43. 1999
- Thatcher, R.A., Kannisto, V., and Vaupel, J.W., *The force of mortality at ages 80 to 120*. Monographs on Population Aging, 3, Odense, DK: Odense University Press. 1998
- Ukraitseva, S.V. and Yashin, A.I., How individual age-associated changes may influence human morbidity and mortality patterns. *Mechanisms of Ageing and Development* 122(13): 1447–1460. 2001
- University of California, Berkeley (USA), and Max Planck Institute for Demographic Research, Rostock, (Germany), Human Mortality Database. Available at www.mortality.org. 2016
- Vaupel, J.W., Biodemography of human aging. *Nature* 464: 536–542. 2010
- Vaupel, J.W. and Canudas-Romo, V., Decomposing demographic change into direct vs. compositional components. *Demographic Research* 7: 1–14. 2002
- Vaupel, J.W., Manton, K.G., and Stallard, E., The Impact of Heterogeneity in Individual Frailty on the Dynamics of Mortality. *Demography* 16: 439–454. 1979
- Vaupel, J.W. and Yashin, A.I., Heterogeneity's Ruses: Some Surprising Effects of Selection on Population Dynamics. *The American Statistician* 39(3): 176–185. 1985
- Wienke, A., *Frailty models in survival analysis*. CRC Press. 2010