

Tetraeffective Causes of Mortality and Survivorship

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Abstract

Any cause that negatively affects mortality, positively affects mortality, negatively affects survivorship, and positively affects survivorship is tetraeffective (i.e., having four kinds of effects). However, until now, tetraeffective causes of mortality and survivorship have been unrecognized, unidentified, unnamed, misconceived, unclear, unexplained, and unspecified. Here I recognize, identify, and name these causes, and I show that every tetraeffective cause of mortality and survivorship combines corresponding at least one mortacause and at least one vitacause; “mortacause” refers here to a cause that positively affects mortality and negatively affects survivorship, and “vitacause” refers to a cause that positively affects survivorship and negatively affects mortality. I elucidate and explain the sources, contexts, and implications of tetraeffective causes of mortality and survivorship, I present strong rationales that suggest that every cause of mortality and survivorship is tetraeffective, and I also present references to considerable previous evidence that I interpret to be suggestive evidence of tetraeffective causes of mortality and survivorship. Moreover, rigorous and thorough multivariable analyses of humans’ and medflies’ mortality and survivorship provide here direct evidence of tetraeffective causes of mortality and survivorship, revealing best-fitting specifications $dY/d(X^p) = a + bX^p$ such that $sign(a) = -sign(bX^p)$, where: Y indicates mortality or survivorship; X indicates age, lifespan, contemporary aggregate size, lifespan aggregate size, or historical time; $dY/d(X^p)$ indicates composite effects of X^p on Y ; a indicates an X -specific mortacause or vitacause; bX^p indicates the corresponding opposite X -specific mortacause or vitacause; and coefficients a , b , and p denote respective X -specific and entity-specific constants. Thus recognition, identification, naming, conceptualization, elucidation, explanation, specification, and demonstration of tetraeffective causes of mortality and survivorship usher a new paradigm of causes of mortality and survivorship and enable and promote further scientific research and practical applications.

Introduction

Bieffective and tetraeffective causes of mortality and survivorship

Scientific research on causality remains problematic and challenging [1-6], scientific research on causes of mortality and survivorship remains particularly problematic and challenging [7-15], and mortality and survivorship and their interrelationships are particularly prone to elicit errors and biases [16-21]. Moreover, every bieffective cause has two kinds of effects, every tetraeffective cause has four kinds of effects, and particular problems and challenges are revealed in considerations of bieffective and tetraeffective causes of mortality and survivorship.

“Mortacause” refers here to any bieffective cause that positively affects mortality and negatively affects survivorship; figure 1 depicts a causal structure of a mortacause (e.g., illustrating an increasing mortacause leading to increasing mortality and decreasing survivorship). “Vitacause” refers here to a bieffective cause that positively affects survivorship and negatively affects mortality; figure 2 depicts a causal structure of a bieffective vitacause that positively affects survivorship and negatively affects mortality (e.g., illustrating an increasing vitacause leading to increasing survivorship and decreasing mortality). Every bi-effective cause of mortality and survivorship is consistent with laws of identity, non-contradiction, and excluded middle [1,22,23].

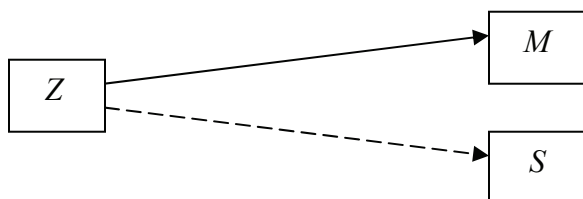


Figure 1. A causal structure of a mortacause (i.e., a bieffective cause that positively affects mortality and negatively affects survivorship). Z denotes a mortacause of mortality M and survivorship S , arrow \rightarrow denotes positive effects, and arrow $---->$ denotes negative effects.

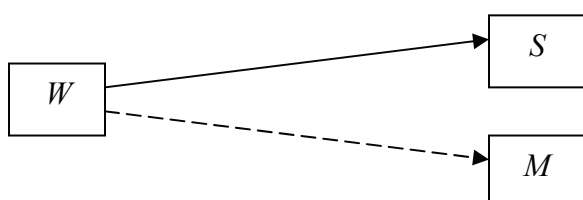


Figure 2. A causal structure of vitacause (i.e., a bieffective cause that positively affects survivorship and negatively affects mortality). W denotes a vitacause of mortality M and survivorship S , arrow \rightarrow denotes positive effects, and arrow $---->$ denotes negative effects.

Every tetraeffective cause of mortality and survivorship negatively affects mortality, positively affects mortality, negatively affects survivorship, and positively affects survivorship. Until now, tetraeffective causes of mortality and survivorship have been unrecognized, unidentified, unnamed, misconceived, unclear, unexplained, and unspecified. Here I recognize, identify, and name these causes, and I present the following discoveries: (i) every tetraeffective cause of mortality and survivorship combines corresponding at least one mortacause and at least one vitacause, (ii) every tetraeffective cause of mortality and survivorship is consistent with laws of identity, non-contradiction, and excluded middle, (iii) every mortacause and every vitacause of every tetraeffective cause of mortality and survivorship is one of dominant and not dominant, and (iv) every tetraeffective cause of mortality and survivorship is hidden in a corresponding bieffective cause of mortality and survivorship[24].

Figure 3 illustrates a causal structure of a tetraeffective cause of mortality and survivorship. Figure 3 depicts positive effects of an X -specific mortacause Xm on mortality M (e.g., illustrating an increasing Xm leading to increasing M), negative effects of an X -specific mortacause Xm on survivorship S (e.g., illustrating an increasing Xm leading to decreasing S), negative effects of an X -specific vitacause Xs on mortality M (e.g., illustrating an increasing Xs leading to decreasing M), and positive effects of an X -specific vitacause Xs on survivorship S (e.g., illustrating an increasing Xv leading to increasing S), where X denotes a tetraeffective cause of mortality and survivorship. Figure 4 depicts effects of corresponding more than one mortacause and more than one vitacause. Figures 3 and 4 illustrate that diverse tetraeffective causes of mortality and survivorship could be structured in diverse ways.

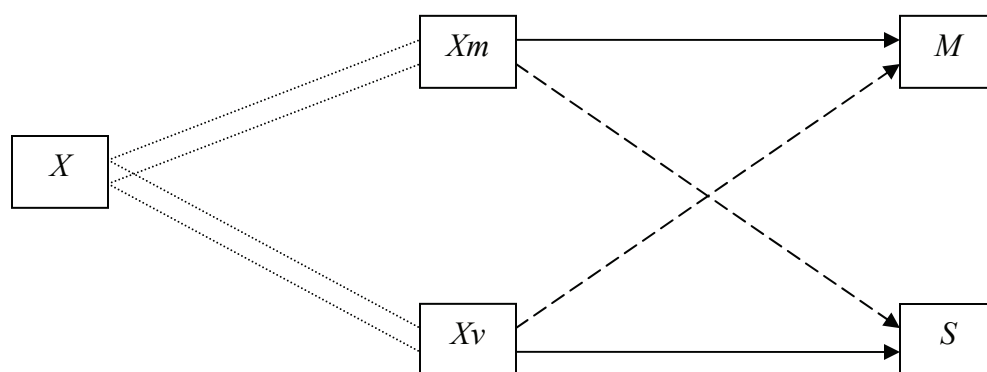


Figure 3. A causal structure of a tetraeffective cause of mortality and survivorship. X denotes a cause of mortality M and survivorship S , Xm denotes an X -specific mortacause, Xs denotes an X -specific vitacause, double dotted lines denote that Xm and Xv are X -specific, arrow \rightarrow denotes positive effects, and arrow $---->$ denotes negative effects.

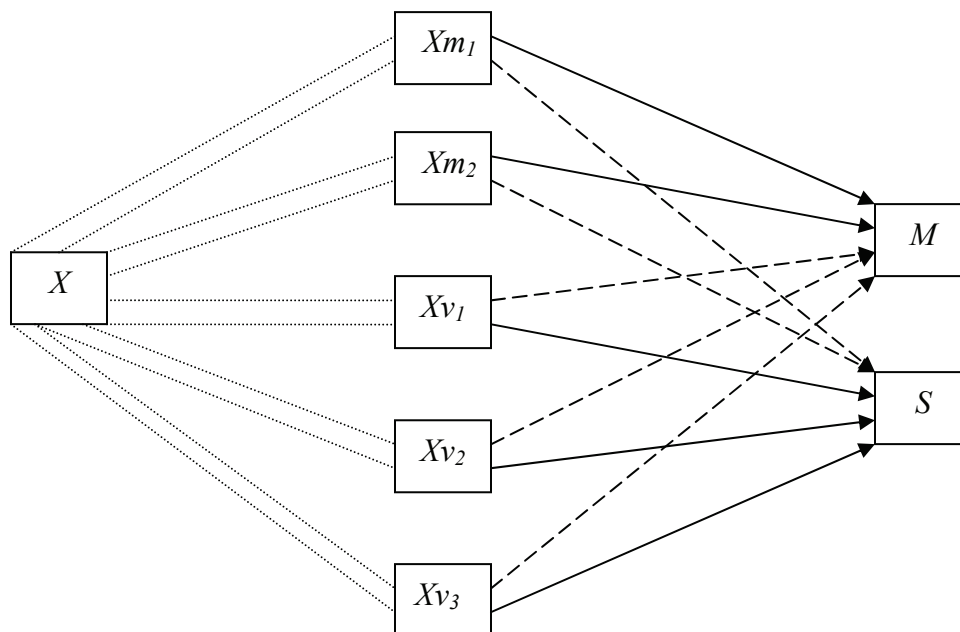


Figure 4. A causal structure of a tetraeffective cause of mortality and survivorship. X denotes a cause of mortality M and survivorship S , Xm denotes an X -specific mortacause, Xs denotes an X -specific vitacause, double dotted lines denote that Xm and Xs are X -specific, arrow \rightarrow denotes positive effects, and arrow $---->$ denotes negative effects.

Figures 3 and 4 illustrate causal structures of tetraeffective causes of mortality and survivorship that are consistent with laws of identity, non-contradiction, and excluded middle. This consistency with laws of identity, non-contradiction, and excluded middle is elucidated when contrasted with the causal structure in Figure 5; Figure 5 illustrates a causal structure of a non-existent and invalid tetraeffective cause of mortality and survivorship that is inconsistent with laws of identity, non-contradiction, and excluded middle.

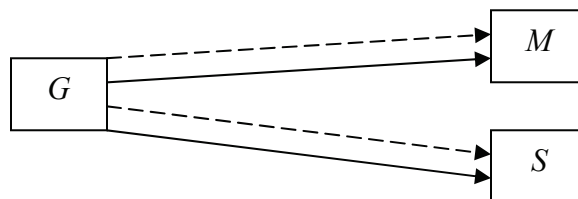


Figure 5. A causal structure of non-existent and invalid tetraeffective cause of mortality and survivorship that is inconsistent with laws of identity, non-contradiction, and excluded middle. G denotes a cause of mortality M and survivorship S , arrow \rightarrow denotes positive effects, and arrow $---->$ denotes negative effects.

Dominance in a tetraeffective cause of mortality and survivorship could be indicated by one or more characteristics (e.g., intensity, effectiveness, force, potency) of respective corresponding cause-specific at least one mortacause and at least one vitacause. Every tetraeffective cause of mortality and survivorship is characterized by one of the following conditions of dominance: (i) total at least one cause-specific mortacause is more dominant than the corresponding total at least one cause-specific vitacause, (ii) total at least one cause-specific mortacause is as dominant as its corresponding total at least one cause-specific vitacause, and (iii) total at least one cause-specific mortacause is less dominant than its corresponding total at least one cause-specific vitacause. If the total at least one cause-specific mortacause is more dominant than the total at least one cause-specific vitacause then the effects of the tetraeffective cause are hidden and they appear as restricted effects of a bieffective mortacause. If the total at least one cause-specific vitacause is the more dominant than the total at least one cause-specific mortacause then the effects of the tetraeffective cause are hidden but they appear as restricted effects of a bieffective vitacause. If the total at least one cause-specific mortacause is as dominant as its corresponding total at least one cause-specific vitacause then the effects of the tetraeffective cause are hidden and they do not appear as restricted effects of a bieffective cause (i.e., no effects are apparent).

I suggest that dominance in tetraeffective causes of mortality and survivorship leads to absence of recognition, absence of identification, and absence of naming of tetraeffective causes of mortality and survivorship as well as prevalence of misconception and unclarity of these tetraeffective causes. Moreover, I suggest that absence of recognition, absence of identification, and absence of naming of tetraeffective causes of mortality and survivorship – and prevalence of misconception and unclarity of these tetraeffective causes – involve the following implicit dominant conventional invalid assumptions: (i) Every cause of mortality and survivorship is exclusively bieffective, (ii) tetraeffective causes of mortality and survivorship do not exist, and (iii) any tetraeffective cause of mortality and survivorship that would exist would be invalidly inconsistent with laws of identity, non-contradiction, and excluded middle. Furthermore, I present strong rationales that suggest that every cause of mortality and survivorship is tetraeffective (i.e., these strong rationales suggest that tetraeffective causes of mortality and survivorship are universal), and I also present references to considerable previous evidence of causes of mortality and survivorship that I interpret to be suggestive evidence of tetraeffective causes of mortality and survivorship.

Rationales for the universality of tetraeffective causes of mortality and survivorship

There is an extensive and long-standing legacy of considerations of oppositions in religion and philosophy [1,22,25-36], quantum theory [37,38], structuralism [39-41], biology [42-44], and art [45-50]. These considerations imply that every mortacause is opposed by – and

opposes – a corresponding at least one vitacause; these considerations also imply that every vitacause is opposed by – and opposes – a corresponding at least one mortacause. These considerations further imply at least one of the following: (i) every cause of mortality and survivorship is bieffective, and (ii) every cause of mortality and survivorship is tetraeffective. Every cause that is observed to be a bieffective cause can be interpreted to be a tetraeffective cause whose components are unknown, unobserved, or ignored. Misconceiving, ignoring, not observing, or not knowing at least one mortacause of a tetraeffective cause of mortality and survivorship do not imply the following: (i) the at least one mortacause does not exist, and (ii) the cause is not tetraeffective. Similarly, misconceiving, ignoring, not observing, or not knowing at least one vitacause of a tetraeffective cause of mortality and survivorship do not imply the following: (i) the at least one vitacause does not exist, and (ii) the cause is not tetraeffective. Therefore, it is invalid to conclude that a cause of mortality and survivorship is exclusively bieffective cause and it is invalid to conclude that this cause is not a tetraeffective cause. Moreover, the extensive and long-standing legacies of considerations of oppositions imply that it is invalid to conclude that a mortacause is not opposed by a corresponding vitacause; similarly, these legacies imply that it is invalid to conclude that a vitacause is not opposed by a corresponding mortacause; these legacies further imply that every cause-specific mortacause requires and is accompanied by a corresponding at least one vitacause; similarly, these legacies further imply that every vitacause requires and is accompanied by a corresponding cause-specific at least one mortacause. Therefore, the universality of tetraeffective causes of mortality and survivorship is tautological (i.e., the statement that every cause of mortality and survivorship is tetraeffective is true in every possible interpretation). However, consistent absence of evidence of tetraeffective causes of mortality and survivorship in rigorous and thorough searches for such causes will render these tautologies to the realm of irrelevance and meaninglessness.

Mortality refers to cessation of existence, survivorship refers to continuation of existence, that which continues or ceases to exist is an entity, and previous research elucidates that entities' existences are regulated [51-59]; thus implying that mortality, survivorship, and their causes are regulated regulators of existence. Additionally, the cessation of existence of all previous entities, and the expected cessation of existence of the universe and all its entities [60,61] – and conceptions of limited existence in diverse (but not all) cultures, religions, and philosophies [30,31] – indicate that every entity's existence is limited. Therefore, if every cause of mortality and survivorship is a tetraeffective cause of mortality and survivorship, then tetraeffective causes are intimately involved in the existence – and the regulation and limitation of existence – of all entities. However, if diverse but not all causes of mortality and survivorship are tetraeffective causes of mortality and survivorship, then existences – and regulations and limitations of existences – do not consistently apply to all entities. Similarly, if no causes of mortality and survivorship are

tetraeffective causes of mortality and survivorship, then existences, regulations, and limitations of existences do not consistently apply to all entities. Therefore, every cause of mortality and survivorship is a tetraeffective cause of mortality and survivorship. Moreover, the number of causes in a system that involves mortacauses and vitacauses in the regulated regulations and limitations of existence of all entities is smaller than the number of causes that are involved in a corresponding system that is restricted to bieffective causes of mortality and survivorship but excludes tetraeffective causes of mortality and survivorship. Therefore, a system that involves tetraeffective causes of mortality and survivorship in the regulated regulations and limitations of existence of all entities is more parsimonious than a corresponding system that excludes tetraeffective causes of mortality and survivorship. This parsimony provides an additional rationale for the universality of tetraeffective causes of mortality and survivorship. Furthermore, symbioses between corresponding at least one cause-specific mortacause and at least one cause-specific vitacause of every tetraeffective cause of mortality and survivorship are illustrated by the observation that any positive effect of age on mortality and any negative effect of age on survivorship require entities of ages greater than zero, and any entity of age greater than zero requires corresponding negative effects of age on mortality and positive effects of age on survivorship; these symbioses and requirements imply that effects of age on mortality and survivorship are tetraeffective, further implying that age is a tetraeffective cause of mortality and survivorship. Similar symbioses, requirements, and implications apply to every entity and every cause of mortality and survivorship.

Utilitarian, practical, moral, and ethical considerations provide additional rationales for the universality of tetraeffective causes of mortality and survivorship. These rationales emphasize that considerations of tetraeffective causes of mortality and survivorship are useful, practical, moral, and ethical. For example, tetraeffective causes of mortality and survivorship imply that it is useful, practical, moral, and ethical to search for vitacauses and mortacauses in effects of cancer or any other disease as well as in effects of nuclear holocaust, global warming, poverty, injustice, or any other cause of mortality and survivorship. Similarly, tetraeffective causes of mortality and survivorship imply that it is useful, practical, moral, and ethical to assume that every vitality is accompanied by an opposite frailty and vice versa, every damage is accompanied by an opposite repair and vice versa, every injury or disease is accompanied by an opposite remedy and vice versa, and so on; further implying that it is invalid, impractical, immoral, unethical, and not useful to assume that elixirs, thanatoses, or destroyers of worlds are unopposed.

Suggestive evidence of tetraeffective causes of mortality and survivorship

New interpretations of previous evidence of causes of mortality and survivorship provide suggestive evidence of tetraeffective causes of mortality and survivorship. Iatrogenic effects

on mortality and survivorship refer to negative and positive medical effects on mortality and survivorship [62,63]; such effects include, for example, iatrogenic effects of surgery [64], pharmacologic medication (e.g., antibiotics [65]), or public health campaigns [66-68]; iatrogenic medical effects on mortality and survivorship provide suggestive evidence of medical-specific mortacauses and vitacauses that negatively and positively affect mortality and survivorship. Similarly, nonlinear or varying respective effects of radiation [49,69-71], insecticides [72], and food intake [73] on mortality and survivorship provide suggestive evidence of respective radiation-specific, insecticides-specific, and food-intake-specific mortacauses and vitacauses that negatively and positively affect mortality and survivorship. Additionally, Strehler-Mildvan correlations [74-80], compensations [70,79-81], decelerations [71,80,82-94], and hysteresis or delays [72,95,96] in effects of age on mortality and survivorship provide suggestive evidence of age-specific mortacauses and vitacauses that negatively and positively affect mortality and survivorship. Moreover, there is ample evidence of nonlinear or varying effects of socioeconomic causes on mortality and survivorship; such socioeconomic effects include, for example, effects of educational attainment, race, gender, income, and economic development [15,66-68,97-107]; nonlinear or varying effects provide suggestive evidence of socioeconomic-specific mortacauses and vitacauses that negatively and positively affect mortality and survivorship. Furthermore, evidence of negative and positive effects of wildfires on mortality and survivorship also provides suggestive evidence of mortacauses and vitacauses that negatively and positively affect mortality and survivorship [108]. Thus diverse effects on mortality and survivorship provide suggestive evidence of tetraeffective causes of mortality and survivorship. Additionally, rigorous and thorough multivariable analyses of humans' and medflies' mortality and survivorship are employed here in a search for direct evidence of tetraeffective causes of mortality and survivorship.

Materials and methods¹

The analyses here expand the analyses that are presented in [109]. The analyses here and in [109] analyze 188,087 weighted cases with 79,164,608 events of death or survival of all entities that were born in Sweden in decennial years 1760 – 1930 and died between 1760 and 2008. The analyses here and in [109] also analyze 50,716 weighted cases with 2,211,782 events of death or survival of caged Mediterranean fruit flies, *Ceratitis capitata*, commonly known as medflies. The analyses here and in [109] analyze these data employing AIC and BIC information criteria and the following multivariable binary limited powered polynomial random-effects regression model of an individual's mortality and survivorship

¹ I applied for a related patent.

$$\eta_{ij} = \beta_0 + \sum_{q=1}^n \sum_{k=1}^{r_q} [\beta_{qk} \{(X_{qij})^{p_q}\}^k] + \sum_{v=1}^u (\beta_v W_{vij}) + \xi_{ij}, \quad (1)$$

such that $P(Y_{ij}) = f(\eta_{ij})$,

where Y_{ij} denotes mortality M_{ij} or survivorship S_{ij} of individual i that continues to exist (i.e., $M_{ij} = 0$ and $S_{ij} = 1$) or ceases to exist (i.e., $M_{ij} = 1$ and $S_{ij} = 0$) at observation j , $P(Y_{ij})$ denotes the probability of mortality (i.e., $P(M_{ij})$) or the probability of survivorship (i.e., $P(S_{ij})$) of individual i at observation j , $f(\eta_{ij})$ is a link function that denotes a transformation of η_{ij} (e.g., a logit transformation $P(Y_{ij}) = \exp(\eta_{ij}) / \{1 + \exp(\eta_{ij})\}$), β denote regression coefficients β , X_q denote ordinal or higher-level variables, and W_v denote categorical variables. The specific X_q variables in this investigation are: $X = A$ denotes humans' and medflies' age, $X = L$ denotes humans' and medflies' lifespan, $X = C$ denotes humans' and medflies' contemporary aggregate size (i.e., this size refers to the number of individuals whose age, sex, and location in time or space are identical to those of the criterion individual), $X = A$ denotes humans' and medflies' lifespan aggregate size (i.e., this size refers to the number of individuals whose lifespan, age, sex, and location in time or space are identical to those of the criterion individual), and $X = H$ denotes humans' historical time (i.e., a specific year). The specific W_v variables in this investigation are: $W = F$ denotes being female in reference to humans' and medflies' sex, and $W = Z$ denotes medflies' respective cages. Coefficients q denote sequential indicators of n distinct variables X , p_q denotes a power coefficient of variable X_q , k are sequential indicators of the r_q polynomial length of variable X_q . Coefficients v denote sequential indicators of u distinct variables W , and each ξ_{ij} denotes a random-effects coefficient corresponding to individual i at observation j . The original data for this investigation are available in [110,111]; further information on the data and regression analyses is available in [109].

Going beyond the analyses in [109], respective best-fitting models 1 yield here

$$dY / d(X^p) = \sum_{k=1}^{r_x} k \beta_k \{(X^p)^{(k-1)}\}, \quad (2)$$

where $dY/d(X^p)$ denotes the first derivative of Y with respect to X^p , p denotes a power coefficient of variable X in model 1, β_k denotes a k regression coefficient of variable X^p in model 1, and r_x denotes the limited length of the polynomial of variable X^p in model 1. Model 2 transforms to $dY/d(X^p) = a + bX^p$ when $r_x = 2$, but Model 2 transforms to $dY/d(X^p) = a + bX^p + c(X^p)^2$ when $r_x = 3$, and so on for higher values of r_x . Best-fitting values of coefficients a , b , c , p , and r_x for a specific Y , a specific X , and a specific kind of entities are determined by the AIC and BIC information criteria that are employed in the respective regression analyses. Derivatives – and respective confidence intervals for these derivatives – for humans' and medflies' mortality or survivorship with respect to age, lifespan,

contemporary aggregate size, lifespan aggregate size are calculated here using procedure “margins” in the Stata software for best-fitting regression models [109,112], verifying consistency with model 2.

Specifications $dY/d(X^p) = a + bX^p$ are employed here for testing whether $dY/d(X^p) = a + bX^p$ such that $\text{sign}(a) = -\text{sign}(bX^p)$. Specifications $dY/d(X^p) = a + bX^p$ such that $\text{sign}(a) = -\text{sign}(bX^p)$ provide simple, parsimonious, and meaningful specifications for tetraeffective causes of mortality and survivorship. First derivative $dY/d(X^p)$ in specifications $dY/d(X^p) = a + bX^p$ is interpreted here as indicative of the intensity of the total effects of at least one X -specific mortacause and at least one X -specific vitacause on mortality or survivorship Y , component a of $dY/d(X^p) = a + bX^p$ is interpreted as indicative of the intensity of the effects of one of an X -specific mortacause and an X -specific vitacause on mortality or survivorship Y , and component bX^p of $dY/d(X^p) = a + bX^p$ is interpreted as indicative of the intensity of the effects of the other one of an X -specific mortacause and an X -specific vitacause on mortality or survivorship Y .

Results

Best-fitting specifications of humans' and medflies' $dM/d(A^p)$, $dS/d(A^p)$, $dM/d(L^p)$, $dS/d(L^p)$, $dM/d(C^p)$, $dS/d(C^p)$, $dM/d(A^p)$, and $dS/d(A^p)$ reveal here that $dY/d(X^p) = a + bX^p$, where $Y = M$ denotes mortality, $Y = S$ denotes survivorship, $X = A$ denotes age, $X = L$ denotes lifespan, $X = C$ denotes contemporary aggregate size, $X = A$ denotes lifespan aggregate size, $dY/d(X^p)$ indicates total effects of X^p on Y , a indicates an X -specific mortacause or vitacause, bX^p indicates the corresponding opposite X -specific mortacause or vitacause, and coefficients a , b , and p denote respective X -specific and entity-specific constants. Additionally, best-fitting specifications of humans' $dM/d(H^p)$ and $dS/d(H^p)$ reveal here that $dY/d(H^p) = a + bH^p + c(H^p)^2$, where $X = H$ denotes humans' historical time. Table 1 shows that $\text{sign}(a) = -\text{sign}(bX^p)$ in best-fitting specifications $dY/d(X^p) = a + bX^p$ of $dM/d(A^p)$, $dS/d(A^p)$, $dM/d(L^p)$, $dS/d(L^p)$, $dM/d(C^p)$, $dS/d(C^p)$, $dM/d(A^p)$, and $dS/d(A^p)$. Table 1 also shows that $\text{sign}(a) = -\text{sign}(bH^p)$ in best-fitting specifications $dY/d(H^p) = a + bH^p + c(H^p)^2$ of $dM/d(H^p)$ and $dS/d(H^p)$.

The results of the multivariable analyses of humans' and medflies' mortality and survivorship reveal tetraeffective causes of mortality and survivorship. Every cause of mortality and survivorship in this investigation is revealed to be a tetraeffective cause of mortality and survivorship; every cause in these analyses is a composite of at least one cause-specific mortacause (positively affecting mortality and negatively affecting survivorship) and at least one vitacause (positively affecting survivorship and negatively affecting mortality).

Table 1. Values of coefficients of humans' and medflies' best-fitting $dY/d(X^p) = a + bX^p$.*

Entities	Y	X	p	a	b	low a	high a	low b	high b
humans	M	A	0.16	-1074.55	1092.24	-1076.92	-1072.19	1089.83	1094.64
humans	S	A	0.16	1074.55	-1092.24	1072.19	1076.92	-1094.64	-1089.83
medflies	M	A	0.13	-2648.52	2591.52	-2681.20	-2615.85	2559.53	2623.51
medflies	S	A	0.16	1402.49	-1413.24	1373.72	1431.25	-1442.58	-1383.90
humans	M	L	0.88	-17.12	0.20	-17.16	-17.08	0.20	0.20
humans	S	L	0.88	17.12	-0.20	17.08	17.16	-0.20	-0.20
medflies	M	L	0.98	-16.67	0.19	-16.88	-16.46	0.19	0.19
medflies	S	L	0.94	19.24	-0.24	18.82	19.65	-0.24	-0.23
humans	M	C	0.75	0.00623	-8.78E-07	0.00619	0.00627	-8.92E-07	-8.66E-07
humans	S	C	0.75	-0.00623	8.78E-07	-0.00627	-0.00619	8.66E-07	8.92E-07
medflies	M	C	1.02	-0.00632	1.37E-06	-0.00652	-0.00612	1.31E-06	1.43E-06
medflies	S	C	1.02	0.00407	-8.06E-07	0.00388	0.00427	-8.64E-07	-7.48E-07
humans	M	Λ	0.3	6.18689	-0.69738	6.16888	6.20490	-0.69938	-0.69536
humans	S	Λ	0.3	-6.18689	0.69737	-6.20490	-6.16888	0.69537	0.69938
medflies	M	Λ	0.95	-0.09025	0.00053	-0.09327	-0.08723	0.00051	0.00055
medflies	S	Λ	0.88	0.11285	-0.00098	0.10723	0.11846	-0.00104	-0.00092
humans	M	H	1.41	-7.84E-03	3.84E-06	-7.91E-03	-7.77E-03	3.72E-06	3.94E-06
humans	S	H	1.41	7.84E-03	-3.84E-06	7.77E-03	7.91E-03	-3.94E-06	-3.72E-06

*Y = M denotes mortality, Y = S denotes survivorship, X = A denotes age, X = L denotes lifespan, X = C denotes contemporary aggregate size, X = Λ denotes lifespan aggregate size, and respective low and high a and b values respectively denote low and high 95% confidence intervals. Best-fitting specifications $dY/d(H^p) = a + bH^p + c(H^p)^2$ where X = H denotes humans' historical time, c = -2.39E-09 (low c = -2.44E-09, high c = -2.35E-09 at confidence interval CI_{0.95}) at Y = M, and c = 2.39E-09 (low c = 2.35E-09, high c = 2.44E-09 at CI_{0.95}) at Y = S.

These analyses reveal mortacauses and vitacauses in best-fitting specifications $dY/d(X^p) = a + bX^p$ such that $sign(a) = -sign(bX^p)$; where $dY/d(X^p)$ denotes the first derivative of mortality or survivorship Y with respect to X^p ; coefficients a, b, and p denote specific X-specific and entity-specific constants; and X respectively indicates age, lifespan, contemporary aggregate size, lifespan aggregate size, or historical time. In these best-fitting specifications, component a is interpreted as indicative of the intensity and effectiveness of effects of an invariant X-specific mortacause or vitacause, such that the invariant X-specific mortacause positively affects mortality and negatively affects survivorship, and such that the invariant X-specific vitacause negatively affects mortality and positively affects survivorship. In these

best-fitting specifications, component bX^p is interpreted as indicative of the intensity and effectiveness of effects of a variable X -specific mortacause or vitacause, such that the variable X -specific mortacause positively affects mortality and negatively affects survivorship, and such that the variable X -specific vitacause negatively affects mortality and positively affects survivorship. These results demonstrate and elucidate that every cause of humans' and medflies' mortality and survivorship in the present analyses is composed of at least one mortacause (positively affecting mortality and negatively affecting survivorship) and at least one vitacause (positively affecting survivorship and negatively affecting mortality). These empirical analyses provide direct evidence of tetraeffective causes of mortality and survivorship and direct evidence of mortacauses and vitacauses.

Discussion

This investigation shows that every tetraeffective cause of mortality and survivorship combines corresponding at least one mortacause and at least one vitacause and is, therefore, consistent with laws of identity, non-contradiction, and excluded middle. Additionally, strong rationales suggest here that every cause of mortality and survivorship is tetraeffective, and considerable previous evidence is interpreted here as being suggestive evidence of tetraeffective causes of mortality and survivorship. Moreover, results of rigorous and through multivariable analyses of humans' and medflies' mortality and survivorship provide here direct evidence of tetraeffective causes of mortality and survivorship, revealing best-fitting specifications $dY/d(X^p) = a + bX^p$ such that $sign(a) = -sign(bX^p)$ for effects of age, lifespan, contemporary aggregate size, lifespan aggregate size, and historical time on mortality and survivorship. Thus tetraeffective causes of mortality and survivorship are recognized, identified, named, conceptualized, elucidated, explained, specified, and demonstrated; thus ushering a new paradigm of causes of mortality and survivorship [24,113], and enabling and promoting further scientific research and practical applications.

There is an obvious affinity between the conception of mortacauses and conceptions of frailty, disease, damage, waste, harm, poison, injury, thanatos, destroyer of worlds, and related phenomena in diverse cultures, religions, philosophies, and scientific investigations [33-36,79,80,114-129]. Similarly, there is an obvious affinity between the conception of vitacauses and conceptions of vitality, conatus, élan vital, selfishness, repair, redundancy, defense, nutrition, elixirs, and related phenomena in diverse cultures, religions, philosophies, and scientific investigations [1,33,34,36,74,79-81,94,130-143]. The universality of tetraeffective causes of mortality and survivorship implies that frailty, disease, damage, waste, harm, poison, injury, thanatos, destroyer of worlds, and related phenomena are mortacause-dominant tetraeffective causes of mortality and survivorship. The universality of tetraeffective causes of mortality and survivorship also implies that vitality, conatus, élan

vital, selfishness, repair, redundancy, defense, nutrition, elixirs, and related phenomena are vitacause-dominant tetraeffective causes of mortality and survivorship. The scientific investigation of these implications requires empirical analyses in further research.

Dominances between corresponding mortacauses and vitacauses of tetraeffective causes of mortality and survivorship are some of the diverse characteristics, processes, or operations that are involved in these tetraeffective causes. Subject to critiques and debates [144-149], previous research shows that power laws and other invariants modulate effects on mortality and survivorship [98,109,150-155]. Previous research also shows that mortality and survivorship are affected by an invariant kind of frailty [33-35,114,115], a variable kind of frailty [116-119], an invariant kind of vitality [33,34,36,130,132,136], and a variable kind of vitality [74,94,137-142]. Furthermore, every mortacause and every vitacause – and interrelationships among mortacauses and vitacauses – could be stable or changing. Therefore, every mortacause and every vitacause is at least: one of dominant and not dominant, one of modulated and not modulated, one of invariant and variable, and one of stable and changing. Much remains to be learned about dominance, non dominance, modulation, non modulation, invariance, variability, stability, change, and other characteristics, processes, or operations of respective cause-specific mortacauses, vitacauses, and their interrelationships.

As noted, scientific research on causality remains problematic and challenging [1-6], scientific research on causes of mortality and survivorship remains particularly problematic and challenging [7-15], and mortality and survivorship and their interrelationships are particularly prone to elicit errors and biases [16-21]. The investigation here shows that consideration of tetraeffective causes of mortality and survivorship usefully elucidates – and deepens the consideration of, and expands the scope of scientific research on – causes of mortality and survivorship. Moreover, the empirical investigation employs here best-fitting multivariable powered polynomial individual-level binary regression models of mortality and survivorship – and respective methods and procedures – that eliminate or reduce susceptibility to the following errors and biases: (i) errors of conflation of age and lifespan, (ii) errors of conflation of age, period, and cohort, (iii) ecological or aggregation errors or biases, (iv) errors of omission or conflation of contemporary-specific aggregates and lifespan-specific aggregates, (v) specification errors associated with omitted – or lurking, or confounding, or underlying – variables, (vi) unobserved heterogeneity bias, and (vii) specification errors associated with nonlinearity [109]. Furthermore, scientists and artists typically attribute force to mortality [46,47,49,50,92,156,157]; therefore, the consideration of mortacauses and vitacauses usefully elucidates here that attributions that ignore force and other characteristics of mortacauses and vitacauses commit errors of omission; additionally, attributions that attribute to mortality the forces of mortacauses and vitacauses commit errors of commission; attribution errors, errors of omission, and errors of commission belong to a

large class of errors and biases that are commonly conceptualized as cognitive errors and biases [16,17,19-21,158-160]. Thus considerations of tetraeffective causes of mortality and survivorship – and the methods and procedures that are employed here – solve some problems and meet some challenges. However, diverse problems and challenges remain, and other problems and challenges come – and will continue to come – into focus; many of these problems and challenges require further research with diverse methods and procedures [1-15].

Specifications $dY/d(X^p) = a + bX^p$ are interpreted here as indicative of the respective intensity or effectiveness of respective tetraeffective causes, mortacauses, and vitacauses. This focus on intensity follows Gompertz's interpretation of derivatives as indicative of intensity [156], and the focus on effectiveness is based here upon implications of relationship $\text{sign}\{dY/d(X^p)\} = \text{sign}(a)$ or $\text{sign}\{dY/d(X^p)\} = \text{sign}(bX^p)$ of $dY/d(X^p) = a + bX^p$ such that $\text{sign}(a) = -\text{sign}(bX^p)$. However, it is instructive to note that insights from Newton's analyses of motion promote the interpretation of derivative $dY/d(X^p)$ as indicative of the velocity of overall effects of X^p on mortality or survivorship Y , the interpretation of coefficient $|b|$ as indicative of the speed of the effects of the variable X -specific mortacause or vitacause, and the interpretation of component bX^p as indicative of the force of the effects of the variable X -specific mortacause or vitacause [161-164]. Furthermore, insights from the Hamiltonian conceptions of energy promote the interpretation of $dY/d(X^p)$ as indicative of the total energy of the overall effects of X^p on mortality or survivorship, the interpretation of component a as indicative of the potential energy of the invariant X -specific mortacause or vitacause, and the interpretation of component bX^p as indicative of the kinetic energy of the variable X -specific mortacause or vitacause [165,166]. Other specifications and interpretations could involve, for example, wave functions [167,168]. These possible interpretative insights show that causes, mortacauses, and vitacauses could be specified and interpreted in diverse fruitful productive ways that could yield diverse insights; these considerations promote further exploration of diverse specifications and diverse interpretations of meanings and characteristics of tetraeffective causes of mortality and survivorship, mortacauses, and vitacauses.

Specifications $Y = a + bX^p$ have been usefully employed in previous research on mortality and survivorship as well as in other research in biology, physics, chemistry, and other branches of science [169-172], but employment of specifications $dY/d(X^p) = a + bX^p$ in previous research on mortality or survivorship is not easy to find. Model $Y = a + bX^p$ is a powered first-degree polynomial model [109], it is also a first-degree fractional polynomial model [173,174], and it is also one of several kinds of Weibull models [79,80,90,92,169-171,175-177]. Model $dY/d(X^p) = a + bX^p$ differs from model $Y = a + bX^p$. Moreover, specification $dY/d(X^p) = a + bX^p$ such that $\text{sign}(a) = -\text{sign}(bX^p)$ is included in specification $dY/d(X^p) = a + bX^p + c(X^p)^2$ such that $\text{sign}(a) = -\text{sign}(bX^p)$, as illustrated here in analyses of

effects of historical time on humans' mortality and survivorship. As noted, model $dY/d(X^p)$ $= a + bX^p$ is based here upon differential equations analyses of best-fitting multivariable powered polynomial individual-level binary regression models of mortality and survivorship [109]. The best-fitting specification $dY/d(X^p) = a + bX^p$ such that $\text{sign}(a) = -\text{sign}(bX^p)$ that is found in this investigation is a specification of tetraeffective causes, mortacauses, and vitacauses. Specification $dY/d(X^p) = a + bX^p$ such that $\text{sign}(a) = -\text{sign}(bX^p)$ is simple, succinct, and meaningful but it is not the only possible simple, succinct, and meaningful mathematical specification – and may not be the best-fitting specification – of tetraeffective causes, mortacauses, and vitacauses. Therefore, it is prudent to investigate the scope of the simple, succinct, and meaningful specification $dY/d(X^p) = a + bX^p$ such that $\text{sign}(a) = -\text{sign}(bX^p)$ [178,179]; the investigation of this scope will indicate whether this specification provides best-fitting specifications of diverse kinds of tetraeffective causes – and diverse kinds of mortacauses and vitacauses – of diverse kinds of entities in diverse times and places.

Acknowledgements

I thank my wife Marcia, our daughters Batia Elsa and Hannah Edna, and our friend Robert D. Tanner.

References

1. Aristotle ([Fourth Century BCE] 1984) The Complete Works of Aristotle; Barnes J, editor. Princeton, N. J.: Princeton University Press.
2. Granger CW (1980) Testing for causality: A personal viewpoint. Journal of Economic Dynamics and Control 2: 329–352.
3. Pearl J (2009) Causality: Models, Reasoning and Inference. Cambridge: Cambridge University Press.
4. Heckman JJ (2005) The scientific model of causality. Sociological Methodology 35: 1-97.
5. Kleinberg S (2013) Causality, probability, and time. New York: Cambridge University Press.
6. Kleinberg S (2015) Why: A guide to finding and using causes. Sebastopol, CA: O'Reilly Media.
7. Hill AB (1965) The environment and disease: Association or causation? Proceedings of the Royal Society of Medicine 58: 295–300.
8. Kleinberg S, Hripesak G (2011) A review of causal inference for biomedical informatics. Journal of Biomedical Informatics 44: 1102-1112.
9. Krieger N (1994) Epidemiology and the web of causation: has anyone seen the spider? Social Science & Medicine 39: 887-903.
10. Krieger N (2008) Proximal, distal, and the politics of causation: What's level got to do with it? American Journal of Public Health 98: 221-230.
11. Ward A (2009) Causal criteria and the problem of complex causation. Medicine, Health Care and Philosophy 12: 333-343.

12. Ward A (2009) The role of causal criteria in causal inferences: Bradford Hill's "aspects of association". *Epidemiologic Perspectives & Innovations* 6: 2.
13. Kaplan GA (2004) What's wrong with social epidemiology, and how can we make it better? *Epidemiologic Reviews* 26: 124-135.
14. Rothman KJ, Greenland S, Poole C, Lash TL (2008) Causation and causal inference. In: J. RK, Greenland S, Poole C, Lash TL, editors. *Modern Epidemiology*. Third ed. Philadelphia, PA: Lippincott, Williams and Wilkins. pp. 6-31.
15. Kawachi I, Adler N, Dow WH (2010) Money, schooling, and health: Mechanisms and causal evidence. *Annals of the New York Academy of Sciences* 1186: 56–68.
16. Kahneman D, Tversky A (1979) Prospect theory: An analysis of decision under risk. *Econometrica* 47.
17. Tversky A, Kahneman D (1981) The framing of decisions and the psychology of choice. *Science* 211: 453-458.
18. Wikipedia (2014) Entry on "survivorship bias," <http://en.wikipedia.org/wiki/>. Accessed on January 5.
19. Olivola CY, Sagara N (2009) Distributions of observed death tolls govern sensitivity to human fatalities. *Proceedings of the National Academy of Sciences* 106: 22151-22156.
20. Elton EJ, Gruber MJ, Blake CR (1996) Survivor bias and mutual fund performance. *Review of Financial Studies* 9: 1097-1120.
21. Ioannidis JPA (2005) Why Most Published Research Findings Are False. *PLoS Med* 2: e124.
22. Horn RL (2015) Contradictions. *The Stanford Encyclopedia of Philosophy*, <http://plato.stanford.edu/entries/contradiction/> accessed May 15.
23. Wikipedia (2015) Law of thought. *Wikipedia, the free encyclopedia* <http://en.wikipedia.org/wiki/>. Accessed December 3.
24. Schickore J (2014) Scientific discovery. *Stanford Encyclopedia of Philosophy*, <http://plato.stanford.edu/entries/scientific-discovery/>.
25. Wang RR (2012) *Yinyang : The Way of Heaven and Earth in Chinese Thought and Culture*. Cambridge, GBR: Cambridge University Press.
26. Nāgārjuna A (1986) *The Philosophy of the Middle Way: Mūlamadhyamakakārikā*. Albany: State University of New York Press.
27. Wikipedia (2015) Entry on "Unity of opposites." *Wikipedia, The Free Encyclopedia* <http://en.wikipedia.org/wiki/>. Accessed December 4.
28. Hegel GWF ([1831] 2001) *Vorlesungen über die Logik*. Hamburg: Meiner.
29. Eliade M (1977) *Mythologies of death: An introduction*. In: Reynolds FE, Waugh EH, editors. *Religious Encounters With Death: Insights from the History and Anthropology of Religions*. University Park: The Pennsylvania State University Press. pp. 13-23.
30. Johnston M (2010) *Surviving Death*. Princeton: Princeton University Press.
31. Reynolds FE, Waugh EH (1977) *Religious Encounters with Death*. University Park: The Pennsylvania State University Press.
32. Wikipedia (2015) Entry on "Heraclitus." *Wikipedia, The Free Encyclopedia* <http://en.wikipedia.org/wiki/>. Accessed May 3
33. Freud S (1922) *Beyond the Pleasure Principle*. London, Vienna: International Psycho-Analytical.
34. Freud S (1930) *Civilization and Its Discontents*. London: Hogarth Press.
35. Wikipedia (2015) Entries on "Death drive" and "Thanatos." *Wikipedia, The Free Encyclopedia* <http://en.wikipedia.org/wiki/>. Accessed September 11.
36. Brown NO (1959) *Life Against Death: The Psychoanalytical Meaning of History*. New York: Vintage Books.

37. Schrödinger E (1935) Die gegenwärtige Situation in der Quantenmechanik. *Naturwissenschaften* 23: 807-812; 823-828; 844-849.
38. Deutsch D, Ekert A (2012) Beyond the quantum horizon. *Scientific American* 307: 84-89.
39. Lévi-Strauss C (1958) *Anthropologie Structurale*. Paris: Plon.
40. Wikipedia (2015) Entry on "Structuralism." Wikipedia, The Free Encyclopedia <http://en.wikipedia.org/wiki/>. Accessed September 11.
41. de Coppet D (1970) 1, 4, 8; 9, 7. La monnaie; Presence de morts et mesure du temps. *L'Homme* 10: 17-39.
42. Benecke M (2002) *Eternal Life: Biomedicine, Aging, and Immortality*. New York: Columbia University Press.
43. Villarreal LP (2004) Are viruses alive? *Scientific American* 291: 101-105.
44. Rasmussen S, Bedau MA, Chen L, Deamer D, Krakauer DC, et al., editors (2009) *Protocells: Bridging Non-living and Living Matter*. Cambridge: MIT Press.
45. Joyce J ([1907] 1993) *Dubliners*. New York: Garland.
46. Neruda P (1935) *Residencia en la Tierra*. Madrid: Ediciones del Árbol.
47. Pearson K (1897) *The Chances of Death and Other Studies in Evolution*. London: Arnold.
48. Saramago J ([1997] 1999) *All the Names*. New York: Harcourt.
49. Strehler BL (1977) *Time, Cells, and Aging*. New York: Academic Press.
50. Turner EL, Hanley JA (2010) Cultural imagery and statistical models of the force of mortality: Addison, Gompertz and Pearson. *Journal of the Royal Statistical Society: Series A (Statistics in Society)* 173: 483-499.
51. Malthus TR ([1798] 1992) *An Essay on the Principle of Population*; Winch D, editor. Cambridge: Cambridge University Press. 392 p.
52. Coleman D (1986) Population regulation: A long range view. In: Coleman D, Schofield R, editors. *The State of Population Theory: Forward from Malthus*. Oxford: Basil Blackwell. pp. 14-41.
53. Lee RD (1987) Population dynamics of humans and other animals. *Demography* 24: 443-465.
54. Sinclair ARE (1988) Population regulation in animals. In: Cherrett JM, editor. *Ecological Concepts: The Contribution of Ecology to an Understanding of the Natural World*. Oxford: Blackwell. pp. 197-241.
55. Sibly RM, Barker D, Denham MC, Hone J, Pagel M (2005) On the regulation of populations of mammals, birds, fish, and insects. *Science* 309: 607-610.
56. Belbas SA (2006) Control systems with hysteresis. In: Bertotti G, Mayergoyz I, editors. *The Science of Hysteresis*. New York: Elsevier. pp. 605-638.
57. Carnes BA (2011) What is lifespan regulation and why does it exist? *Biogerontology* 12: 367-374.
58. Koshland DE (1973) Protein shape and biological control. *Scientific American* 229: 52-64.
59. Tanner RD, DeAngelis LH (1974) Sigmoidal and growth rate kinetic hysteresis in biochemical systems. *The Chemical Engineering Journal* 8: 113-123.
60. ESO (2015) European Southern Observatory, "Charting the slow death of the universe." www.eso.org/public/usa/news/eso1533/ Accessed August 18.
61. Wikipedia (2015) Entry on "ultimate fate of the universe." Wikipedia, The Free Encyclopedia <http://en.wikipedia.org/wiki/>. Accessed August 18.
62. Illich I (1974) *Medical Nemesis: The Expropriation of Health*. London: Caler & Boyars.
63. Wikipedia (2015) Entry on "iatrogenesis." Wikipedia, The Free Encyclopedia <http://en.wikipedia.org/wiki/>. Accessed May 3
64. Wikipedia (2015) Entry on "Perioperative mortality." Wikipedia, The Free Encyclopedia <http://en.wikipedia.org/wiki/>. Accessed July 17.

65. Wikipedia (2015) Entries on "antibiotics" and "antimicrobial resistance." Wikipedia, The Free Encyclopedia <http://en.wikipedia.org/wiki/>. Accessed October 7.
66. Johansson SR, Mosk C (1987) Exposure, resistance, and life expectancy: Disease and death during the economic development of Japan, 1900-1960. *Population Studies* 41: 207-235.
67. Mosk C, Johansson SR (1986) Income and mortality: Evidence from modern Japan. *Population Development Review* 12: 415-440.
68. Riley JC (2008) *Low Income, Social Growth, and Good Health: A History of Twelve Countries*. Berkeley: University of California Press.
69. Atlan H, Miquel J, Helmle LC, Dolkas CB (1976) Thermodynamics of aging in *Drosophila melanogaster*. *Mechanisms of Ageing and Development* 5: 371-387.
70. Rosenberg B, Kemeny G, Smith LG, Skurnick ID, Bandurski MJ (1973) The kinetics and thermodynamics of death in multicellular organisms. *Mechanisms of Ageing and Development* 2: 275-293.
71. Carnes BA, Holden LR, Olshansky SJ, Witten MT, Siegel JS (2006) Mortality partitions and their relevance to research on senescence. *Biogerontology* 7: 183-198.
72. Banks HT, Banks JE, Joyner SL (2008) Estimation in time-delay modeling of insecticide-induced mortality. CRSC Technical Reports, CRSC-TR08-15.
73. Fontana L, Partridge L, Longo VD (2010) Extending healthy life span from yeast to humans. *Science* 328: 321-326.
74. Strehler BL, Mildvan AS (1960) General theory of mortality and aging. *Science* 132: 14-21.
75. Riggs JE (1993) The Gompertz function: Distinguishing mathematical from biological limitations. *Mechanisms of Ageing and Development* 69: 33-36.
76. Finkelstein M (2012) Discussing the Strehler-Mildvan model of mortality. *Demographic Research* 26: 191-206.
77. Li T, Anderson JJ (2015) The Strehler-Mildvan correlation from the perspective of a two-process vitality model. *Population Studies* 69: 91-104.
78. Yashin AI, Begun AS, Boiko SI, Ukraintseva SV, Oeppen J (2001) The new trends in survival improvement require a revision of traditional gerontological concepts. *Experimental Gerontology* 37: 157-167.
79. Gavrilov LA, Gavrilova NL (1991) *The Biology of Lifespan: A Quantitative Approach*. New York: Harwood Academic.
80. Gavrilov LA, Gavrilova NS (2006) Models of systems failure in aging. In: Conn PM, editor. *Handbook of Models for Human Aging*. Burlington, MA: Elsevier Academic Press. pp. 45-68.
81. Gavrilov LA, Gavrilova NS (2006) Reliability theory of aging and longevity. In: Masoro EJ, Austad SN, editors. *Handbook of Models for Human Aging*. Oxford: Academic Press. pp. 3-42.
82. Greenwood M, Irwin JO (1939) The biostatistics of senility. *Human Biology* 11: 1-23.
83. Carey JR, Liedo P, Orozco D, Vaupel JW (1992) Slowing of mortality rates at older ages in large medfly cohorts. *Science* 258: 457-461.
84. Curtsinger JW, Fukui HH, Townsend DR, Vaupel JW (1992) Demography of genotypes: failure of the limited life-span paradigm in *Drosophila melanogaster*. *Science* 258: 461-463.
85. Mueller LD, Rauser CL, Rose MR (2011) *Does Aging Stop?* New York: Oxford University Press.
86. Vaupel JW, Carey JR, Christensen K, Johnson TE (1998) Biodemographic trajectories of longevity. *Science* 280: 855-860.
87. Witten M (1989) Quantifying the concepts of rate and acceleration/deceleration of aging. *Growth, Development and Aging* 53: 7-16.

88. Witten TM, Eakin T (1997) Multiphasic models of survival: Background and early developments. *Experimental Gerontology* 32: 259-285.
89. Fukui HH, Xiu L, Curtsinger JW (1993) Slowing of age-specific mortality rates in *Drosophila melanogaster*. *Experimental Gerontology* 28: 585-599.
90. Bebbington M, Green R, Lai C-D, Zitakis R (2012) Beyond the Gompertz law: exploring the late-life mortality deceleration phenomenon. *Scandinavian Actuarial Journal*: 1-19.
91. Bebbington M, Lai C-D, Zitakis R (2007) Modeling human mortality using mixtures of bathtub shaped failure distributions. *Journal of Theoretical Biology* 245: 528-538.
92. Bebbington M, Lai C-D, Zitakis RA (2011) Modelling deceleration in senescent mortality. *Mathematical Population Studies* 18: 18-37.
93. Golubev A (2009) How could the Gompertz-Makeham law evolve. *Journal of Theoretical Biology* 258: 1-17.
94. Li T, Anderson JJ (2009) The vitality model: A way to understand population survival and demographic heterogeneity. *Theoretical Population Biology* 76: 118-131.
95. Amal Y, Pinto MC (2010) Global solutions for an age-dependent model of nucleation, growth and ageing with hysteresis. *Discrete and Continuous Dynamical Systems - B* 13: 517 - 535.
96. Takeuchi KA (2008) Scaling of hysteresis loops at phase transitions into a quasiabsorbing state. *Physical Review E* 77: 1-4.
97. Preston SH (1976) *Mortality Patterns In National Populations*. New York: Academic Press. 201 p.
98. Epelbaum M (1990) Sociomontary patterns and specifications. *Social Science Research* 19: 322-347.
99. Krieger N, Rowley DL, Herman AA, Avery B, Phillips MT (1993) Racism, sexism, and social class: Implications for studies of health, disease, and well-being. *American Journal of Preventive Medicine* 9: 82-122.
100. Wrigley EA, Oeppen JE, Schofield RS (1997) *English Population History from Family Reconstitution 1580-1937*. Cambridge: Cambridge University Press.
101. Lynch J, Davey Smith G, Harper S, Kaplan GA, Wolfson M (2004) Is income inequality a determinant of population health? Part 1. A systematic review. *The Milbank Quarterly* 82: 5-99.
102. Smith JP (2004) Unraveling the SES-health connection. *Population and Development Review* 30: 108-132.
103. Cutler DM, Deaton A, Lleras-Muney A (2006) The determinants of mortality. *Journal of Economic Perspectives* 20: 97-120.
104. Krieger N, Rehkopf DH, Chen JT, Waterman PD, Marcelli E, et al. (2008) The fall and rise of US inequities in premature mortality: 1960-2002. *PLoS Med* 5: e46.
105. Lynch J, Davey Smith G, Harper S, Hillemeier M (2004) Is income inequality a determinant of population health? Part 2. U.S. national and regional trends in income inequality and age-and cause-specific mortality. *The Milbank Quarterly* 82: 355-400.
106. Adler NE, Boyce T, Chesney MA, Cohen S, Folkman S, et al. (1994) Socioeconomic status and health: The challenge of the gradient. *American Psychologist* 49: 15-24.
107. Antonovsky A (1967) Social class, life expectancy, and overall mortality. *Milbank Memorial Fund Quarterly* 45: 31-73.
108. Wikipedia (2016) Wildfire. Wikipedia, the free encyclopedia <http://en.wikipedia.org/wiki/>. Accessed February 10
109. Epelbaum M (2014) Lifespan and aggregate size variables in specifications of mortality or survivorship. *PLoS ONE* 9: e84156.

110. Original data on medflies are available at <https://ucdavis.app.box.com/s/ovoxdo0h8l4s1jym0ugihnfjpwj8iou>.
111. Original data on humans in Sweden are available at www.mortality.org.
112. StataCorp (2009) Stata Statistical Software Release 11. College Station, TX: Stata Corporation.
113. Kuhn TS ([1962] 1970) The structure of scientific revolutions. Chicago: University of Chicago Press.
114. Vaupel JW, Manton KG, Stallard E (1979) The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography* 16: 439-454.
115. Duchateau L, Janssen P (2008) The Frailty Model. New York: Springer.
116. Rockwood K, Mitnitski A (2007) Frailty in relation to the accumulation of deficits. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences* 62: 722-727.
117. Mohler MJ, Fain MJ, Wertheimer AM, Najafi B, Nikolich-Augich J (2014) The Frailty Syndrome: Clinical measurements and basic underpinnings in humans and animals. *Experimental Gerontology* 54: 6-13.
118. Bergman H, Ferrucci L, Guralnik J, Hogan DB, Hummel S, et al. (2007) Frailty: An emerging research and clinical paradigm: Issues and controversies. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences* 62: 731-737.
119. Mitnitski A, Song X, Rockwood K (2013) Assessing biological aging: the origin of deficit accumulation. *Biogerontology* 14: 709-717.
120. Kirkwood TBL (1977) Evolution of ageing. *Nature* 270: 301-304.
121. Medawar PB ([1952] 1957) An unsolved problem in biology. In: Medawar PB, editor. *The Uniqueness of the Individual*. London: Methuen. pp. 44-70.
122. Milne EMG (2008) The natural distribution of survival. *Journal of Theoretical Biology* 255: 223-236.
123. Munch SB, Mangel M (2006) Evaluation of mortality trajectories in evolutionary biodemography. *Proceedings of the National Academy of Sciences* 103: 16604-16607.
124. Szilard L (1959) On the nature of the aging process. *Proceedings of the National Academy of Sciences* 45: 30-45.
125. Wikipedia (2015) Thanatos. Wikipedia, the free encyclopedia <http://en.wikipedia.org/wiki/>. Accessed December 3
126. Wikipedia (2015) Bhagavad Gita. Wikipedia, the free encyclopedia <http://en.wikipedia.org/wiki/>. Accessed December 3
127. Hijiya JA (2000) The Gita of J. Robert Oppenheimer. *Proceedings of the American Philosophical Society* 144: 123-167.
128. Mathers CD, Loncar D (2006) Projections of global mortality and burden of disease from 2002 to 2030. *Plos Medicine*.
129. WHO (2013) International Classification of Diseases. <http://www.who.int/classifications/icd/en/>. Accessed 2013 March 12
130. Spinoza B ([1677] 2008) *Opera Posthuma*; Mignini F, editor. Amsterdam: Quodlibet.
131. Bergson H (1907) *L'évolution créatrice*. Paris: F. Alcan.
132. Wikipedia (2015) Entries on "conatus," "élan vital," and "selfish gene." Wikipedia, The Free Encyclopedia <http://en.wikipedia.org/wiki/>. Accessed May 3
133. Dawkins R ([1976] 2006) *The selfish gene*. Oxford: Oxford University Press.
134. Wikipedia (2015) Entry on "Antibody." Wikipedia, The Free Encyclopedia <http://en.wikipedia.org/wiki/>. Accessed May 3
135. Wikipedia (2015) Entry on "Selfish gene." Wikipedia, The Free Encyclopedia <http://en.wikipedia.org/wiki/>. Accessed May 3
136. Pearl R (1928) *The Rate of Living*. New York: Alfred A. Knopf.

137. Anderson JJ (1992) A vitality-based stochastic model for organism survival. In: DeAngelis DL, Gross LJ, editors. Individual-based models and approaches in ecology. New York: Chapman & Hall. pp. 256-277.
138. Anderson JJ (2000) Vitality-based model relating stressors and environmental properties to organism survival. *Ecological Monographs* 70: 445-470.
139. Anderson JJ, Gildea MC, Williams DW, Li T (2008) Linking growth, survival, and heterogeneity through vitality. *The American Naturalist* 171: E20-E43.
140. Anderson JJ, Li T (2015) A two-process mortality model with extensions to juvenile mortality, population dynamics, and evolution. *Population Association of America Meeting*.
141. Li T, Anderson J (2013) Shaping human mortality patterns through intrinsic and extrinsic vitality processes. *Demographic Research* 28: 341-372.
142. Zuev SM, Yashin AI, Manton KG, Dowd E, Pogojev IB, et al. (2000) Vitality index in survival modeling: how physiological aging influences mortality. *Journal of Gerontology A* 55: B10-19.
143. Wikipedia (2015) Elixir of life. Wikipedia, the free encyclopedia <http://en.wikipedia.org/wiki/>. Accessed December 3
144. Dodds PS, Rothman DH, Weitz JS (2001) Re-examination of the '3/4-law' of metabolism. *Journal of Theoretical Biology* 209: 9-27.
145. Kolokotronis T, Savage VM, Deeds EJ, Fontana W (2010) Curvature in metabolic scaling. *Nature* 464: 753-756.
146. Savage VM, White EP, Moses ME, Ernest SKM, Enquist BJ, et al. (2006) Comment on 'The illusion of invariant quantities in life histories'. *Science* 312: 198b.
147. White CR (2010) There is no single p. *Nature* 464: 691-692.
148. Nee S, Colegrave N, West SA, Grafen A (2005) The illusion of invariant quantities in life histories. *Science* 309: 1236-1239.
149. Stumpf MPH, Porter MA (2012) Critical truths about power laws. *Science* 335: 665-666.
150. Juckett DA, Rosenberg B (1982) The kinetics and thermodynamics of lysis of young and old sheep red blood cells. *Mechanisms of Ageing and Development* 18: 33-45.
151. Bevacqua D, Melià P, De Leo GA, Gatto M (2011) Intra-specific scaling of natural mortality in fish: the paradigmatic case of the European eel. *Oecologia* 165: 333-339.
152. Brown JH, Gillooly JF, Allen AP, Savage VM, West GB (2004) Toward a metabolic theory of ecology. *Ecology* 85: 1771-1789.
153. West GB, Brown JH, Enquist BJ (1997) A general model for the origin of allometric scaling laws in biology. *Science* 276: 122-126.
154. Charnov EL (1993) *Life History Invariants; Some Explorations of Symmetry in Evolutionary Ecology*. Oxford: Oxford University Press. 167 p.
155. Beverton RJH, Holt SJ (1959) A review of the lifespans and mortality rates of fish in nature and their relation to growth and other physiological characteristics. In: Wolstenholme GEW, O'Connor M, editors. *CIBA Foundation Colloquia on Ageing Volume V: The Lifespan of Animals*. Boston: Little, Brown. pp. 142-180.
156. Gompertz B (1825) On the nature of the function expressive of the law of human mortality, and on a new mode of determining the value of life contingencies. *Philosophical Transactions of the Royal Society of London* 115: 513-585.
157. Missov TI, Lenart A, Nemeth L, Canudas-Romo V, Vaupel JW (2015) The Gompertz force of mortality in terms of the modal age at death. *Demographic Research* 32/36.
158. Nisbett RE, Ross LD (1980) *Human Inference: Strategies and Shortcomings of Social Judgment*. Englewood Cliffs, NJ: Prentice-Hall.
159. Ross LD, Nisbett RE (1991) *The Person and the Situation: Perspectives of Social Psychology*. New York: McGraw-Hill.

160. Wikipedia (2014) Entries on "cognitive bias" and "list of cognitive biases," <http://en.wikipedia.org/wiki/>. Accessed on January 5.
161. Newton IS (1687) *Philosophiae Naturalis Principia Mathematica*. Londini: S. Pepys, Reg. Soc. Praeses.
162. Wikipedia (2015) Entries on "velocity," "acceleration," and "speed." Wikipedia, The Free Encyclopedia <http://en.wikipedia.org/wiki/>. Accessed July 7.
163. Wikipedia (2015) Entry on "force." Wikipedia, The Free Encyclopedia <http://en.wikipedia.org/wiki/>. Accessed July 7.
164. Wikipedia (2015) Entry on "Newton's laws of motion." Wikipedia, The Free Encyclopedia <http://en.wikipedia.org/wiki/>. Accessed July 7.
165. Hamilton WR ([1834-1835] 2015) On a General Method in Dynamics. www.maths.tcd.ie/pub/HistMath/People/Hamilton/Dynamics/ Accessed July 23.
166. Wikipedia (2015) Entry on "Hamiltonian mechanics." Wikipedia, The Free Encyclopedia <http://en.wikipedia.org/wiki/>. Accessed July 7.
167. Schrödinger E (1926) An undulatory theory of the mechanics of atoms and molecules. *Physical Review* 28: 1049-1070.
168. Wikipedia (2015) Entry on "Schrödinger equation." Wikipedia, The Free Encyclopedia <http://en.wikipedia.org/wiki/>. Accessed July 7.
169. Ricklefs RE (1998) Evolutionary theories of aging: confirmation of a fundamental prediction, with implications for the genetic basis and evolution of life span. *The American Naturalist* 152: 24-44.
170. Ricklefs RE (2010) Life-history connections to rates of aging in terrestrial vertebrates. *Proceedings of the National Academy of Sciences* 107: 10314-10319.
171. Ricklefs RE, Scheuerlein A (2001) Comparison of aging-related mortality among birds and mammals. *Experimental gerontology* 36: 845-857.
172. White CR, Kearney MR (2014) Metabolic scaling in animals: methods, empirical results, and theoretical explanations. *Comprehensive Physiology* 4: 231-256.
173. Royston P, Altman DG (1994) Regression using fractional polynomials of continuous covariates: parsimonious parametric modelling (with discussion). *Journal of the Royal Statistical Society Series C (Applied Statistics)* 43: 429-467.
174. Royston P, Sauerbrei W (2008) *Multivariable Model-Building: A Pragmatic Approach to Regression Analysis Based on Fractional Polynomials for Modelling Continuous Variables*. Chichester, England: John Wiley.
175. Weibull WA (1939) A statistical theory of the strength of materials. *Ingeniorsvetenskapsakademiens Handlingar* 151: 5-45.
176. Weibull WA (1951) A statistical distribution function of wide applicability. *Journal of Applied Mechanics* 18: 293-297.
177. Juckett DA, Rosenberg B (1993) Comparison of the Gompertz and Weibull functions as descriptors for human mortality distributions and their intersections. *Mechanisms of Ageing and Development* 69: 1-31.
178. Krakauer DC, Collins JP, Erwin D, Flack JC, Fontana W, et al. (2011) The challenges and scope of theoretical biology. *Journal of Theoretical Biology* 276: 269-276.
179. Walker HA, Cohen BP (1985) Scope statements: Imperatives for evaluating theory. *American Sociological Review* 50: 288-301.