Tetraeffective causes of mortality and survivorship

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Abstract
Every tetraeffective cause of mortality and survivorship negatively and positively affects mortality and negatively and positively affects survivorship. There is some scientific evidence of tetraeffective causes of mortality and survivorship, and strong rationales suggest that every cause of mortality and survivorship is tetraeffective. However, until now, tetraeffective causes of mortality and survivorship have remained unidentified, unnamed, unrecognized, unclear, misconceived, unspecified, and unexplained. Here 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-specific component that positively affects mortality and negatively affects survivorship, and "vitacause" refers to a cause-specific component that positively affects survivorship and negatively affects mortality. Best-fitting specifications $\partial Y/\partial(X^p) = a + bX^p$ such that $sign(a) = -sign(bX^p)$ of respective tetradic effects of age, lifespan, contemporary aggregate size, lifespan aggregate size, and historical time on humans' and medflies' mortality and survivorship reveal here mortacauses and vitacauses of respective tetraeffective causes of mortality and survivorship; in these specifications components a and bX^p indicate a mortacause and a vitacause of a tetraeffective cause X of mortality or survivorship Y. Thus mortacauses, vitacauses, and tetraeffective causes of mortality and survivorship are identified, named, recognized, elucidated, conceptualized, specified, explained, and demonstrated.

1. Introduction
1.1 Tetraeffective causes of mortality and survivorship
Any cause that negatively and positively affects mortality and negatively and positively affects survivorship has tetradic – i.e., four kinds of – effects on mortality and survivorship; therefore, I name and identify any such cause as "tetraeffective." Scientific evidence of tetraeffective causes of mortality and survivorship includes evidence of tetradic iatrogenic effects on mortality and survivorship [1,2], including, for example, iatrogenic effects of surgery [3], pharmacologic medication (e.g., antibiotics [4]), or public health campaigns [5-7]. Scientific evidence of tetraeffective causes of mortality and survivorship also includes evidence of Strehler-Mildvan correlations [8-15] and compensations [13,14,16,17] in tetradic effects of age on mortality and survivorship as well as evidence of tetradic effects of wildfires on mortality and survivorship [18]. However, until now, tetraeffective causes of mortality and survivorship have remained unidentified, unnamed, unrecognized, unclear, misconceived, unspecified, and

unexplained. Moreover, tetradic effects on mortality and survivorship appear to be inconsistent with the laws of identity, noncontradiction, and excluded middle that are considered in [19-21]; such inconsistencies are illustrated in the causal structure that is depicted in Figure 1.

Figure 1. A causal structure of tetradic effects on mortality and survivorship. Cause *X* negatively affects mortality *M*, positively affects mortality *M*, negatively affects survivorship *S*, and positively affects survivorship *S*. Arrow → denotes positive effects, and arrow ----> denotes negative effects.

　　　　Here I elucidate that every tetraeffective cause of mortality and survivorship combines corresponding at least one mortacause and at least one vitacause; "mortacause" refers to a cause-specific component that positively affects mortality and negatively affects survivorship, and "vitacause" refers to a cause-specific component that positively affects survivorship and negatively affects mortality. Figure 2 depicts the causal structure of a tetraeffective cause *X* that affects mortality *M* and survivorship *S* through the combined 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 *Xv* on mortality *M* (e.g., illustrating an increasing *Xv* leading to decreasing *M*), and positive effects of an *X*-specific vitacause *Xv* on survivorship *S* (e.g., illustrating an increasing *Xv* leading to increasing *S*). Figure 3 depicts the causal structure of a tetraeffective cause with two mortacauses and three vitacauses. Figures 2 and 3 illustrate that diverse tetraeffective causes of mortality and survivorship could be structured in diverse ways. Additionally, the causal structures that are depicted in Figures 2 and 3 are consistent with the laws of identity, noncontradiction, and excluded middle. Any tetraeffective cause that combines corresponding at least one mortacause and at least one vitacause is consistent with the laws of identity, noncontradiction, and excluded middle.

Figure 2. 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, *Xv* denotes an *X*-specific vitacause, double-dotted lines denote that *Xm* and *Xv* are *X*-specific, arrow → denotes positive effects, and arrow **---->** denotes negative effects.

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

Conceptions of deep structures are found in diverse fields of science and scholarship [22-26]; there are obvious affinities between conceptions of deep structure and the conceptions of mortacauses and vitacauses of respective tetraeffective causes but much remains to be learned about these affinities. Conceptions of frailty, disease, damage, waste, harm, poison, injury, thanatos, destroyer of worlds, and related phenomena are found in diverse cultures, religions, philosophies, and scientific investigations [13,14,27-46]; there are obvious affinities between these conceptions and the conception of mortacauses but remains to be learned about these affinities. Conceptions of vitality, conatus, élan vital, self-preservation, repair, redundancy, defense, nutrition, elixirs, and related phenomena are found in diverse cultures, religions, philosophies, and scientific investigations [8,13,14,17,19,27,28,41,47-61]; there are obvious affinities between these conceptions and the conception of vitacauses but much remains to be learned about these affinities. Moreover, strong rationales suggest that every cause of mortality and survivorship is tetraeffective.

1.2 Rationales for the universality of tetraeffective causes of mortality and survivorship

Mortality refers to cessation of existence, survivorship refers to continuation of existence, and that which continues or ceases to exist is an entity. Tetradic effects mean that effects of mortacauses do not cease during the continuation of existence, and effects of vitacauses do not cease during the cessation of existence. Moreover, previous research elucidates that entities' continuations and cessations of existences are regulated [62-70]; 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 [71,72] – and conceptions of limited existence in diverse (but not all) cultures, religions, and philosophies [73,74] – indicate that every entity's existence is limited. Therefore, existences, regulations, and limitations of existences consistently apply to every entity and to all entities. 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 every entity and 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 every entity and all entities. Similarly, if no causes of mortality and survivorship are tetraeffective, then existences, regulations, and limitations of existences do not consistently apply to every entity and all entities. Therefore, every cause of mortality and survivorship is a tetraeffective cause of mortality and survivorship.

The total number of causes in an hypothetical system that involves only tetraeffective causes in the regulated regulations and limitations of existence of all entities is smaller than the total number of causes in an hypothetical system that excludes tetraeffective causes of mortality and survivorship – but includes only monoeffective or bieffective causes of mortality and survivorship – in the regulated regulations and limitations of existence of all entities. 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.

There is an extensive and long-standing legacy of considerations of oppositions in religion and philosophy [19,20,27-29,41,73-80], quantum theory [81,82], structuralism [24,26,83], biology [84-86], and art [87-92]. 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. Additionally, these oppositions imply that tetraeffective causes oppose – and are opposed by – other tetraeffective causes. However, if every cause of mortality and survivorship is not tetraeffective (i.e., if tetraeffective causes of mortality and survivorship do not exist) then at least some causes of mortality and survivorship are not opposed by – and do not oppose – other causes of mortality and survivorship, in violation of the requisites of opposition. Therefore, corresponding at least one mortacause and at least one vitacause must be intrinsic to every cause of mortality and survivorship and, therefore, every cause of mortality and survivorship is tetraeffective. These considerations thus amplify, deepen, and elucidate previous considerations of intrinsic and extrinsic causes of mortality and survivorship and previous considerations of essential and coincidental properties. There is ample previous consideration of intrinsic and extrinsic causes of mortality [11,13,17,58,93-105], and there is ample previous consideration of essential and coincidental properties [19,106].

Every tetraeffective cause of mortality and survivorship can be interpreted as a cause whose tetraeffective components are hidden (e.g., unknown, unobserved, ignored, or misconceived). Additionally, every cause that is known or observed to be a non-tetraeffective cause can be interpreted to be a tetraeffective cause whose components are hidden. Moreover, hiddenness of at least one mortacause of a tetraeffective cause of mortality and survivorship does not imply the following: (i) the at least one mortacause does not exist, and (ii) the cause is not tetraeffective. Similarly, hiddenness of at least one vitacause of a tetraeffective cause of mortality and survivorship does 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 – e.g., every cause, any cause, a specific cause – of mortality and survivorship is not a tetraeffective cause.

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.

1.3 Specifications of tetraeffective causes of mortality and survivorship

Specifications $\partial Y/\partial(X^p) = a + bX^p$ such that $sign(a) = -sign(bX^p)$ – and corresponding longer polynomials (e.g., $\partial Y/\partial(X^p) = a + bX^p + c(X^p)^2$ such that $sign(a) = -sign(bX^p)$) – specify mortacauses and vitacauses of tetraeffective causes of mortality and survivorship, where X denotes an ordinal or higher level tetraeffective cause of mortality or survivorship Y, $Y = M$ denotes mortality, $Y = S$ denotes survivorship, $\partial Y/\partial(X^p)$ denotes the partial derivative of Y with respect to X^p, and coefficients a, b, and p denote respective specific constants. Partial derivatives $\partial Y/\partial(X^p)$ in specifications $\partial Y/\partial(X^p) = a + bX^p$ such that $sign(a) = -sign(bX^p)$ – and in corresponding longer polynomials – are 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 $\partial Y/\partial(X^p) = a + bX^p$ is interpreted as indicative of the intensity of the effects of an X-specific mortacause or vitacause on mortality or survivorship Y, and component bX^p of $\partial Y/\partial(X^p) = a + bX^p$ is interpreted as indicative of the intensity of the effects of the opposite X-specific mortacause or vitacause on mortality or survivorship Y. Relationship $sign(a) = -sign(bX^p)$ means that if component a indicates the intensity of the effects of an X-specific mortacause then component bX^p indicates the intensity of the effects of an X-specific vitacause, and if component a indicates the intensity of the effects of an X-specific vitacause then component bX^p indicates the intensity of the effects of an X-specific mortacause. The focus on intensity here follows Gompertz's interpretation of derivatives as indicative of intensity (Gompertz 1825). Specifications $\partial Y/\partial(X^p) = a + bX^p$ such that $sign(a) = -sign(bX^p)$ are consistent with – and convey – the causal structure that is depicted in Figure 2. Additionally, by allowing diverse kinds of polynomials (e.g., $\partial Y/\partial(X^p) = a + bX^p + c(X^p)^2$), these specifications are also consistent with the causal structure that is depicted in Figure 3.

      Models $M_A = M_0 + aA^p$ have been usefully employed in previous research on effects of age A and mortality rate M_0 at an initial age A_0 on mortality rate M_A at age A, with constants a and p [102-104]. Models $Z = a + bG^p$ have been usefully employed in previous research on metabolic scaling of diverse phenomena Z, focusing on effects of mass G, and corresponding constants a, b, and p [107]. However, employment of models $\partial Y/\partial(X^p) = a + bX^p$ in previous research on mortality or survivorship is not easy to find. Models $\partial Y/\partial(X^p) = a + bX^p$ are powered polynomial models [108], they are also fractional polynomial models [109,110], and they are also some of several kinds of

Weibull models [13,14,102-104,111-115]. Specifications $\partial Y/\partial(X^p) = a + bX^p$ – and higher level polynomials – are investigated here in further analyses of causes of humans' and medflies' mortality and survivorship.

2. Materials and methods

In [108] I present analyses of 188,087 weighted cases with 79,164,608 events of death or survival of all individuals that were born in Sweden in decennial years 1760 – 1930 and died between 1760 and 2008. In [108] I also present analyses of 50,716 weighted cases with 2,211,782 events of death or survival of caged Mediterranean fruit flies, *Ceratitis capitata*, commonly known as medflies. In [108] I employ AIC and BIC information criteria in tests of the following multivariable individual-level longitudinal limited powered polynomials binary random-effects regression models of mortality and survivorship:

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

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 humans' year or medflies' day 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, $f(\eta_{ij})$ denotes a binary transformation of η_{ij}, $\boldsymbol{\beta}$ denote regression coefficients β, $\boldsymbol{X_q}$ denote ordinal or higher-level variables X, and $\boldsymbol{W_v}$ denote categorical variables W. The specific $\boldsymbol{X_q}$ variables in this investigation are: $X = A$ denotes humans' or medflies' age, $X = L$ denotes humans' or medflies' lifespan, $X = C$ denotes humans' or 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), $X = \Lambda$ denotes humans' or medflies' lifespan aggregate size (i.e., this size refers to the number of individuals whose lifespan and contemporary aggregate are identical to those of the criterion individual), and $X = H$ denotes humans' historical time (i.e., a specific year). The specific $\boldsymbol{W_v}$ variables in this investigation are: $W = F$ denotes being female in reference to humans' or medflies' sex, and $W = Q$ 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. Conventional transformations $f(\eta_{ij})$ include, for example, a logit transformation $f(\eta_{ij}) = exp(\eta_{ij})/\{1+ exp(\eta_{ij})\}$), a probit transformation $f(\eta_{ij}) = \Phi(\eta_{ij})$, or a complementary log-log transformation $f(\eta_{ij}) = 1 - exp\{-exp(\eta_{ij})\}$ [116-119]. Equations 5 – 8 and Tables 1 – 4 in [108] present regression coefficients and respective 95% confidence intervals of the best-fitting models of humans' and medflies' mortality and survivorship. For example, the best-fitting model of humans' mortality and survivorship that is presented in Equation 5 in [108] is specified with

$$\eta_{ij} = 511.78 - 1074.55(A_{ij}^{0.16}) + 546.12(A_{ij}^{0.16})^2 - 17.12(L_{ij}^{0.88})$$
$$+ 0.101(L_{ij}^{0.88})^2 + 0.006 (C_{ij}^{0.75}) - (4.39e-7)(C_{ij}^{0.75})^2$$
$$+ 6.19(\Lambda_{ij}^{0.30}) - 0.35(\Lambda_{ij}^{0.30})^2 - 0.008(H_{ij}^{1.41}) \quad (2)$$
$$+ (1.92e-6)(H_{ij}^{1.41})^2 - (7.97e-10)(H_{ij}^{1.41})^3$$
$$- 1.13(F_{ij}), \text{ such that } P(M_{ij}) = exp(\eta_{ij})/\{1+ exp(\eta_{ij})\}.$$

Further information on the data and regression analyses is available in [108].

If $r_q = 1$ for an ordinal or higher level cause X_q then then $\beta_{q1}(X_{qij})^{p_q}$ in Model 1, where β_{q1} denotes the respective regression coefficient for this cause, such that $\beta_1 X^p$ for a generic cause X. If $r_q = 2$ for an ordinal or higher level cause X_q then $\beta_{q1}(X_{qij})^{p_q}$ and

$\beta_{q2}\{(X_{qij})^{p_q}\}^2$ in Model 1, where β_{q1} and β_{q2} denote the respective regression coefficients for this cause, such that $\beta_1 X^p$ and $\beta_2(X^p)^2$ for a generic cause X. Similar procedures apply to $r_q > 2$ in Model 1. Here I calculate

$$\partial Y / \partial(X^p) = \sum_{k=1}^{r} k\beta_k\{(X^p)^{(k-1)}\}, \quad (3)$$

where $\partial Y/\partial(X^p)$ denotes the partial derivative of Y with respect to a variable X^p while holding all other components of Model 1 as known and constant, β_k denotes a k regression coefficient of a generic variable X^p in Model 1, and r denotes the length of the polynomial of variable X^p in Model 1. If $r = 2$ in Model 3, then $\partial Y/\partial(X^p) = a + bX^p$, where $\beta_1 = a$ and $2\beta_2 = b$. If $r = 3$ in Model 3, then $\partial Y/\partial(X^p) = a + bX^p + c(X^p)^2$ where $\beta_1 = a$, $2\beta_2 = b$, and $3\beta_3 = c$. Similar procedures apply to $r > 3$ in Model 3. As noted, specifications $\partial Y/\partial(X^p) = a + bX^p$ – and higher level polynomials – are investigated here in analyses of causes of humans' and medflies' mortality and survivorship.

3. Results

Table 1 presents best-fitting values of coefficients a, b, c, and p for respective Y, X, and humans or medflies based upon the best-fitting models in [108]. For example, Equations 5 – 8 and Tables 1 – 4 in [108] reveal that each of the best-fitting models include β_{A1} and β_{A2} regression coefficients for respective effects of age A^p on humans' and medflies' mortality and survivorship, where A denotes age, and where p denotes the respective power coefficient for age. Therefore, using $a = \beta_{A1}$ and $b = 2\beta_{A2}$ yields corresponding partial derivatives $\partial Y/\partial(A^p) = a + bA^p$, holding all other variables as known and constant in each respective best-fitting model of humans' and medflies' mortality and survivorship. Similarly, Equations 5 – 6 and Tables 1 – 2 in [108] reveal that the best-fitting models include β_{H1}, β_{H2}, and β_{H3} regression coefficients for effects of historical time H^p on humans' mortality and survivorship, where H denotes historical time, and where p denotes the respective power coefficient for historical time. Therefore, using $a = \beta_{H1}$, $b = 2\beta_{H2}$, and $c = 3\beta_{H3}$ yields corresponding partial derivatives $\partial Y/\partial(H^p) = a + bH^p$

$+ c(H^p)^2$, holding all other variables as known and constant in each respective best-fitting model of humans' mortality and survivorship.

Table 1. Values of *a* and *b* of humans' and medflies' best-fitting $\partial Y/\partial(X^p) = a + bX^p$.

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

Notes: $Y = M$ denotes mortality, $Y = S$ denotes survivorship, $X = A$ denotes age, $X = L$ denotes lifespan, $X = C$ denotes contemporary aggregate size, $X = \Lambda$ denotes lifespan aggregate size for humans and medflies, and $X = H$ denotes humans' historical time. Respective low and high *a* and *b* values respectively denote low and high values of these coefficients at the 95% confidence intervals. Where $\partial Y/\partial(H^p) = a + bH^p + c(H^p)^2$, $c = -2.39E-09$ (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 $Y = S$.

Values for coefficients *a* and *b* in Table 1 show that best-fitting specifications of humans' and medflies' $\partial M/\partial(A^p)$, $\partial S/\partial(A^p)$, $\partial M/\partial(L^p)$, $\partial S/\partial(L^p)$, $\partial M/\partial(C^p)$, $\partial S/\partial(C^p)$, $\partial M/\partial(\Lambda^p)$, and $\partial S/\partial(\Lambda^p)$ reveal here that $\partial Y/\partial(X^p) = a + bX^p$ such that $sign(a) = -sign(bX^p)$, indicating respective mortacauses and vitacauses of respective tetradic effects of age, lifespan, contemporary aggregate size, and lifespan aggregate size on humans' and medflies' mortality and survivorship. Table 1 also shows that best-fitting specifications

of humans' $\partial M/\partial(H^p)$ and $\partial S/\partial(H^p)$ reveal here that $\partial Y/\partial(H^p) = a + bH^p + c(H^p)^2$ *such that sign(a) = -sign(bH^p)*, indicating a corresponding mortacause and vitacause of respective tetradic effects of historical time on humans' mortality or survivorship. These results show and elucidate that every cause of humans' and medflies' mortality and survivorship in the present analyses is a respective tetraeffective cause that is composed of at least one mortacause and at least one vitacause; each mortacause positively affects mortality and negatively affects survivorship, and each vitacause positively affects survivorship and negatively affects mortality.

4. Discussion

This investigation shows tetraeffective causes of mortality and survivorship wherein each tetraeffective cause combines corresponding at least one mortacause and at least one vitacause. As noted, every tetraeffective cause of mortality and survivorship that combines at least one mortacause and at least one vitacause is consistent with laws of identify, noncontradiction, and excluded middle. As also noted, strong rationales suggest that every cause of mortality and survivorship is tetraeffective, and there is considerable previous scientific evidence of tetraeffective causes of mortality and survivorship. Analyses of humans' and medflies' mortality and survivorship reveal here best-fitting specifications $\partial Y/\partial(X^p) = a + bX^p$ *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; these results provide evidence of mortacauses and vitacauses of diverse tetraeffective causes of diverse kinds of individuals in diverse situations. Thus mortacauses, vitacauses, and tetraeffective causes of mortality and survivorship are identified, named, recognized, elucidated, conceived, specified, explained, and demonstrated; thus ushering a new paradigm of causes of mortality and survivorship, and enabling and promoting further scientific research and practical applications [120,121].

As noted, 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 [13,14,27-46]. Similarly, as noted, 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 [8,13,14,17,19,27,28,41,47-61]. 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 further research.

Scientific research on causality remains problematic and challenging [19,122-126], scientific research on causes of mortality and survivorship remains particularly

problematic and challenging [127-135], and mortality and survivorship and their interrelationships are particularly prone to elicit errors and biases [136-141]. 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 addresses here diverse problems and challenges by employing 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 [108]. Furthermore, scientists and artists typically attribute force to mortality [88,89,91,92,114,142,143]; therefore, the consideration of mortacauses and vitacauses usefully elucidates here that attributions that ignore intensity, force, and other characteristics of mortacauses and vitacauses commit errors of omission; additionally, attributions that attribute to mortality the intensity, force, or other characteristics 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 [136,137,139-141,144-146]. Thus considerations of tetraeffective causes of mortality and survivorship – and the methods and procedures that are employed here – address diverse problems and meet diverse challenges. However, other diverse problems and challenges remain, and other diverse 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 [19,122-135].

As noted, specifications $\partial Y/\partial(X^p) = a + bX^p$ are interpreted here as indicative of the respective intensity of respective tetraeffective causes, mortacauses, and vitacauses. As also noted, the focus on intensity follows Gompertz's interpretation of derivatives as indicative of intensity [142]. However, it is instructive to note that insights from Newton's analyses of motion promote the interpretation of partial derivative $\partial Y/\partial(X^p)$ 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 [147-150]. Furthermore, insights from the Hamiltonian conceptions of energy promote the interpretation of $\partial Y/\partial(X^p)$ 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 [151,152]. Other specifications and interpretations could involve, for example, wave functions [153,154]. These possible interpretative insights show that mortacauses, vitacauses, and tetraeffective causes 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 mortacauses, vitacauses, and tetraeffective causes of mortality and survivorship.

Specification $\partial Y/\partial(X^p) = a + bX^p$ such that $sign(a) = -sign(bX^p)$ 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 diverse ordinal or higher level tetraeffective causes, mortacauses, and vitacauses. Therefore, it is prudent to investigate the scope of the simple, succinct, and meaningful specification $\partial Y/\partial(X^p) = a + bX^p$ such that $sign(a) = -sign(bX^p)$; the investigation of this scope will indicate whether this specification provides best-fitting specifications of diverse kinds of tetraeffective causes of mortality and survivorship – and diverse kinds of mortacauses and vitacauses – of diverse kinds of entities in diverse times and places [155,156]. Moreover, a major limitation of specifications with derivatives is that these specifications are not applicable to categorical tetraeffective causes (e.g., sex, race). Additional questions about scope apply to learning whether diverse kinds of binary outcomes (e.g., disease and health, war and peace, failure and success) are affected by tetraeffective causes. Furthermore, in spite of considerable progress and promising leads, models and laws of mortality and survivorship remain elusive and the search for such models and laws remains inconclusive [8,11,13,14,17,38,39,54-59,94,95,113-115,142,157-174]; specification $dY/d(X^p) = a + bX^p$ such that $sign(a) = -sign(bX^p)$ provides a potentially valuable addition to this search for models and laws of mortality and survivorship. Much remains to be learned about the lawfulness and universality of tetraeffective causes of mortality and survivorship, and much also remains to be learned about the lawfulness, best-fit, and scope of specification $dY/d(X^p) = a + bX^p$ such that $sign(a) = -sign(bX^p)$.

As noted, previous researchers distinguish between intrinsic and extrinsic causes of mortality and survivorship [11,13,17,58,93-105]. Additionally, previous investigations of mortality and survivorship focus on diverse kinds of entities (e.g., living and non-living entities [85,86,175-178]). The tetraeffective causes that are examined in this investigation (i.e., age, lifespan, concurrent aggregate size, lifespan aggregate size, and historical time) differ with respect to their extrinsicness and intrinsicness, and the investigation here reveals similarities and differences between diverse kinds of entities (i.e., humans and medflies). These considerations show that much remains to be learned about the scope of diverse characteristics of mortacauses and vitacauses of extrinsic and intrinsic tetraeffective causes of mortality and survivorship of diverse kinds of entities [155,156]. Moreover, previous research reveals diverse kinds of resolutions of oppositions [19,20,41,75,76,78-80,179,180]; implying that much remains to be learned about resolutions of the following oppositions: Oppositions between respective at least one mortacause and at least one vitacause, oppositions between mortality and survivorship, oppositions between negatively and positively affected mortality, and oppositions between negatively and positively affected survivorship. Moreover, previous research suggests that *modi operandi* of diverse phenomena determine – and are determined by – their respective *habitus* and *opus operatum* [19,179-183]. These considerations show that much remains to be learned about *modi operandi*, *habitus*, and *opus operatum* of mortacauses and vitacauses of tetraeffective causes of mortality and survivorship.

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References

1. Illich I (1974) Medical Nemesis: The Expropriation of Health. London: Caler & Boyars.
2. Wikipedia (2015) Entry on "iatrogenesis." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed May 3.
3. Wikipedia (2015) Entry on "Perioperative mortality." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed July 17.
4. Wikipedia (2015) Entries on "antibiotics" and "antimicrobial resistance." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed October 7.
5. 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.
6. Mosk C, Johansson SR (1986) Income and mortality: Evidence from modern Japan. Population Development Review 12: 415-440.
7. Riley JC (2008) Low Income, Social Growth, and Good Health: A History of Twelve Countries. Berkeley: University of California Press.
8. Strehler BL, Mildvan AS (1960) General theory of mortality and aging. Science 132: 14-21.
9. Riggs JE (1993) The Gompertz function: Distinguishing mathematical from biological limitations. Mechanisms of Ageing and Development 69: 33-36.
10. Finkelstein M (2012) Discussing the Strehler-Mildvan model of mortality. Demographic Research 26: 191-206.
11. Li T, Anderson JJ (2015) The Strehler-Mildvan correlation from the perspective of a two-process vitality model. Population Studies 69: 91-104.
12. 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.
13. Gavrilov LA, Gavrilova NL (1991) The Biology of Lifespan: A Quantitative Approach. New York: Harwood Academic.
14. 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.
15. Zheng H, Yang Y, Land K (2011) Heterogeneity in the Strehler-Mildvan general theory of mortality and aging. Demography 48: 267-290.
16. 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.

17. 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.

18. Wikipedia (2016) Wildfire. Wikipedia, the free encyclopedia http://en.wikipedia.org/wiki/. Accessed February 10.

19. Aristotle ([Fourth Century BCE] 1984) The Complete Works of Aristotle; Barnes J, editor. Princeton, N. J.: Princeton University Press.

20. Horn LR (2015) Contradiction. The Stanford Encyclopedia of Philosophy, http://plato.stanford.edu/entries/contradiction/ accessed May 15.

21. Wikipedia (2015) Law of thought. Wikipedia, the free encyclopedia http://en.wikipedia.org/wiki/. Accessed December 3.

22. Chomsky N (1957) Syntactic Structure. Hague: Mouton.

23. de Saussure F (1916) Cours de Linguistique Générale; Bally C, Sechehaye A, editors. Paris: Payot.

24. Lévi-Strauss C (1958) Anthropolgie Structurale. Paris: Plon.

25. Wikipedia (2015) Entry on "Deep structure and surface structure" Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed September 11.

26. Wikipedia (2015) Entry on "Structuralism." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed September 11.

27. Freud S (1922) Beyond the Pleasure Principle. London, Vienna: International Psycho-Analytical.

28. Freud S (1930) Civilization and Its Discontents. London: Hogarth Press.

29. Wikipedia (2015) Entries on "Death drive" and "Thanatos." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed September 11.

30. Vaupel JW, Manton KG, Stallard E (1979) The impact of heterogeneity in individual frailty on the dynamics of mortality. Demography 16: 439-454.

31. Duchateau L, Janssen P (2008) The Frailty Model. New York: Springer.

32. 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.

33. 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.

34. 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.

35. Mitnitski A, Song X, Rockwood K (2013) Assessing biological aging: the origin of deficit accumulation. Biogerontology 14: 709-717.

36. Kirkwood TBL (1977) Evolution of ageing. Nature 270: 301-304.

37. Medawar PB ([1952] 1957) An unsolved problem in biology. In: Medawar PB, editor. The Uniqueness of the Individual. London: Methuen. pp. 44-70.

38. Milne EMG (2008) The natural distribution of survival. Journal of Theoretical Biology 255: 223–236.

39. Munch SB, Mangel M (2006) Evaluation of mortality trajectories in evolutionary biodemography. Proceedings of the National Academy of Sciences 103: 16604-16607.
40. Szilard L (1959) On the nature of the aging process. Proceedings of the National Academy of Sciences 45: 30-45.
41. Brown NO (1959) Life Against Death: The Psychoanalytical Meaning of History. New York: Vintage Books.
42. Wikipedia (2015) Thanatos. Wikipedia, the free encyclopedia http://en.wikipedia.org/wiki/. Accessed December 3
43. Wikipedia (2015) Bhagavad Gita. Wikipedia, the free encyclopedia http://en.wikipedia.org/wiki/. Accessed December 3
44. Hijiya JA (2000) The Gita of J. Robert Oppenheimer. Proceedings of the American Philosophical Society 144: 123-167.
45. Mathers CD, Loncar D (2006) Projections of global mortality and burden of disease from 2002 to 2030. Plos Medicine.
46. WHO (2013) International Classification of Diseases. http://www.who.int/classifications/icd/en/. Accessed 2013 March 12
47. Spinoza B ([1677] 2008) Opera Posthuma; Mignini F, editor. Amsterdam: Quodlibet.
48. Bergson H (1907) L'évolution créatice. Paris: F. Alcan.
49. Wikipedia (2015) Entries on "conatus," "élan vital,"and "selfish gene." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed May 3
50. Dawkins R ([1976] 2006) The selfish gene. Oxford: Oxford University Press.
51. Wikipedia (2015) Entry on "Antibody." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed May 3
52. Wikipedia (2015) Entry on "Selfish gene." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed May 3
53. Pearl R (1928) The Rate of Living. New York: Alfred A. Knopf.
54. 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.
55. Anderson JJ (2000) Vitality-based model relating stressors and environmental properties to organism survival. Ecological Monographs 70: 445-470.
56. Anderson JJ, Gildea MC, Williams DW, Li T (2008) Linking growth, survival, and heterogeneity through vitality. The American Naturalist 171: E20-E43.
57. Anderson JJ, Li T (2015) A two-process mortality model with extensions to juvenile mortality, population dynamics, and evolution. Population Association of America Meeting.
58. Li T, Anderson J (2013) Shaping human mortality patterns through intrinsic and extrinsic vitality processes. Demographic Research 28: 341-372.
59. Li T, Anderson JJ (2009) The vitality model: A way to understand population survival and demographic heterogeneity. Theoretical Population Biology 76: 118-131.
60. 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.

61. Wikipedia (2015) Elixir of life. Wikipedia, the free encyclopedia http://en.wikipedia.org/wiki/. Accessed December 3

62. Malthus TR ([1798] 1992) An Essay on the Principle of Population; Winch D, editor. Cambridge: Cambridge University Press. 392 p.

63. 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.

64. Lee RD (1987) Population dynamics of humans and other animals. Demography 24: 443-465.

65. 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.

66. 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.

67. Belbas SA (2006) Control systems with hysteresis. In: Bertotti G, Mayergoyz I, editors. The Science of Hysteresis. New York: Elsevier. pp. 605-638.

68. Carnes BA (2011) What is lifespan regulation and why does it exist? Biogerontology 12: 367-374.

69. Koshland DE (1973) Protein shape and biological control. Scientific American 229: 52-64.

70. Tanner RD, DeAngelis LH (1974) Sigmoidal and growth rate kinetic hysteresis in biochemical systems. The Chemical Engineering Journal 8: 113-123.

71. ESO (2015) European Southern Observatory, "Charting the slow death of the universe." www.eso.org/public/usa/news/eso1533/ Accessed August 18.

72. Wikipedia (2015) Entry on "ultimate fate of the universe." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed August 18.

73. Reynolds FE, Waugh EH (1977) Religious Encounters with Death. University Park: The Pennsylvania State University Press.

74. Johnston M (2010) Surviving Death. Princeton: Princeton University Press.

75. Wang RR (2012) Yinyang : The Way of Heaven and Earth in Chinese Thought and Culture. Cambridge, GBR: Cambridge University Press.

76. Nāgārjuna A (1986) The Philosophy of the Middle Way: Mūlamadhyamakakārikā. Albany: State University of New York Press.

77. Wikipedia (2015) Entry on "Unity of opposites." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed December 4.

78. Hegel GWF ([1831] 2001) Vorlesungen über die Logik. Hamburg: Meiner.

79. 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.

80. Wikipedia (2015) Entry on "Heraclitus." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed May 3

81. Schrödinger E (1935) Die gegenwärtige Situation in der Quantenmechanik. Naturwissenschaften 23: 807-812; 823-828; 844-849.

82. Deutsch D, Ekert A (2012) Beyond the quantum horizon. Scientific American 307: 84-89.

83. de Coppet D (1970) 1, 4, 8; 9, 7. La monnaie; Presence de morts et mesure du temps. L'Homme 10: 17-39.

84. Benecke M (2002) Eternal Life: Biomedicine, Aging, and Immortality. New York: Columbia University Press.

85. Villarreal LP (2004) Are viruses alive? Scientific American 291: 101-105.

86. Rasmussen S, Bedau MA, Chen L, Deamer D, Krakauer DC, et al., editors (2009) Protocells: Bridging Non-living and Living Matter. Cambridge: MIT Press.

87. Joyce J ([1907] 1993) Dubliners. New York: Garland.

88. Neruda P (1935) Residencia en la Tierra. Madrid: Ediciones del Árbol.

89. Pearson K (1897) The Chances of Death and Other Studies in Evolution. London: Arnold.

90. Saramago J ([1997] 1999) All the Names. New York: Harcourt.

91. Strehler BL (1977) Time, Cells, and Aging. New York: Academic Press.

92. 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.

93. Kirkwood TBL (1985) Comparative and evolutionary aspects of longevity. In: Finch CE, Schneider EL, editors. Handbook of the Biology of Aging. New York: Reinhold. pp. 27-43.

94. Carnes BA, Olshansky SJ, Grahn D (1996) Continuing the search for a law of mortality. Population and Development Review 22: 231-264.

95. Carnes BA, Holden LR, Olshansky SJ, Witten MT, Siegel JS (2006) Mortality partitions and their relevance to research on senescence. Biogerontology 7: 183-198.

96. Carnes BA, Witten TM (2014) How long must humans live? The Journals of Gerontology Series A: Biological Sciences and Medical Sciences 69: 965-970.

97. Olshansky SJ (2010) The law of mortality revisited: Interspecies comparisons of mortality. Journal of Comparative Pathology 142: S4-S9.

98. Stearns SC (1992) The evolution of life histories. Oxford: Oxford University Press.

99. Stearns SC, Ackermann M, Doebeli M, Kaiser M (2000) Experimental evolution of aging, growth, and reproduction in fruitflies. Proceedings of the National Academy of Sciences 97: 3309-3313.

100. Monaghan P, Chamantier A, Nussey DH, Ricklefs RE (2008) The evolutionary ecology of senescence. Functional Ecology 22: 371-378.

101. Reznick DN, Bryant MJ, Roff DA, Ghalambor CK, Ghalambor DE (2004) Effects of extrinsic mortality on the evolution of senescence in guppies. Nature 431: 1095-1099.

102. 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.

103. Ricklefs RE (2010) Life-history connections to rates of aging in terrestrial vertebrates. Proceedings of the National Academy of Sciences 107: 10314-10319.

104. Ricklefs RE, Scheuerlein A (2001) Comparison of aging-related mortality among birds and mammals. Experimental gerontology 36: 845-857.
105. Williams PD, Day T (2003) Antagonistic pleiotropy, mortality source interactions, and the evolutionary theory of senescence. Evolution 57: 1478-1488.
106. Robertson T, Atkins P (2015) Essential vs. accidental properties, The Stanford Encyclopedia of Philosophy, http://plato.stanford.edu/entries/contradiction/ accessed May 15.
107. White CR, Kearney MR (2014) Metabolic scaling in animals: methods, empirical results, and theoretical explanations. Comprehensive Physiology 4: 231-256.
108. Epelbaum M (2014) Lifespan and aggregate size variables in specifications of mortality or survivorship. PLoS ONE 9: e84156.
109. 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.
110. 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.
111. Weibull WA (1939) A statistical theory of the strength of materials. Ingeniorsvetenskapsakademiens Handlingar 151: 5–45.
112. Weibull WA (1951) A statistical distribution function of wide applicability. Journal of Applied Mechanics 18: 293–297.
113. 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.
114. Bebbington M, Lai C-D, Zitikis RA (2011) Modelling deceleration in senescent mortality. Mathematical Population Studies 18: 18-37.
115. Bebbington M, Green R, Lai C-D, Zitikis R (2012) Beyond the Gompertz law: exploring the late-life mortality deceleration phenomenon. Scandinavian Actuarial Journal: 1-19.
116. Chambers EA, Cox DR (1967) Discrimination between alternative binary response models. Biometrika 54: 573-578.
117. Collett D (2003) Modelling Binary Data. Boca Raton: Chapman & Hall/CRC.
118. Cox DR (1970) The Analysis of Binary Data. London: Chapman and Hall.
119. Koenker R, Yoon J (2009) Parametric links for binary choice models: A Fisherian-Bayesian colloquy. Journal of Econometrics 152: 120-130.
120. Kuhn TS ([1962] 1970) The structure of scientific revolutions. Chicago: University of Chicago Press.
121. Schickore J (2014) Scientific discovery. Stanford Encyclopedia of Philosophy, http://platostanfordedu/entries/scientific-discovery/.
122. Granger CW (1980) Testing for causality: A personal viewpoint. Journal of Economic Dynamics and Control 2: 329–352.
123. Heckman JJ (2005) The scientific model of causality. Sociological Methodology 35: 1-97.
124. Kleinberg S (2013) Causality, probability, and time. New York: Cambridge University Press.

125. Kleinberg S (2015) Why: A guide to finding and using causes. Sebastopol, CA: O'Reilly Media.
126. Pearl J (2009) Causality: Models, Reasoning and Inference. Cambridge: Cambridge University Press.
127. Hill AB (1965) The environment and disease: Association or causation? Proceedings of the Royal Society of Medicine 58: 295–300.
128. Kleinberg S, Hripcsak G (2011) A review of causal inference for biomedical informatics. Journal of Biomedical Informatics 44: 1102-1112.
129. Krieger N (1994) Epidemiology and the web of causation: has anyone seen the spider? Social Science & Medicine 39: 887-903.
130. 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.
131. Ward A (2009) Causal criteria and the problem of complex causation. Medicine, Health Care and Philosophy 12: 333-343.
132. Ward A (2009) The role of causal criteria in causal inferences: Bradford Hill's "aspects of association". Epidemiologic Perspectives & Innovations 6: 2.
133. Kaplan GA (2004) What's wrong with social epidemiology, and how can we make it better? Epidemiologic Reviews 26: 124-135.
134. Rothman KJ, Greenland S, Poole C, Lash TL (2008) Causation and causal inference. In: Rothman KJ, Greenland S, Poole C, Lash TL, editors. Modern Epidemiology. Third ed. Philadelphia, PA: Lippincott, Williams and Wilkins. pp. 6-31.
135. 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.
136. Kahneman D, Tversky A (1979) Prospect theory: An analysis of decision under risk. Econometrica 47: 263-291.
137. Tversky A, Kahneman D (1981) The framing of decisions and the psychology of choice. Science 211: 453-458.
138. Wikipedia (2014) Entry on "survivorship bias," http://en.wikipedia.org/wiki/. Accessed on January 5.
139. 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.
140. Elton EJ, Gruber MJ, Blake CR (1996) Survivor bias and mutual fund performance. Review of Financial Studies 9: 1097-1120.
141. Ioannidis JPA (2005) Why Most Published Research Findings Are False. PLoS Med 2: e124.
142. 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.
143. 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.
144. Nisbett RE, Ross LD (1980) Human Inference: Strategies and Shortcomings of Social Judgment. Englewood Cliffs, NJ: Prentice-Hall.

145. Ross LD, Nisbett RE (1991) The Person and the Situation: Perspectives of Social Psychology. New York: McGraw-Hill.

146. Wikipedia (2014) Entries on "cognitive bias" and "list of cognitive biases," http://en.wikipedia.org/wiki/. Accessed on January 5.

147. Newton IS (1687) Philosophiae Naturalis Principia Mathematica. Londini: S. Pepys, Reg. Soc. Praeses.

148. Wikipedia (2015) Entries on "velocity," "acceleration," and "speed." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed July 7.

149. Wikipedia (2015) Entry on "force." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed July 7.

150. Wikipedia (2015) Entry on "Newton's laws of motion." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed July 7.

151. Hamilton WR ([1834-1835] 2015) On a General Method in Dynamics. www.maths.tcd.ie/pub/HistMath/People/Hamilton/Dynamics/ Accessed July 23.

152. Wikipedia (2015) Entry on "Hamiltonian mechanics." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed July 7.

153. Schrödinger E (1926) An andulatory theory of the mechanics of atoms and molecules. Physical Review 28: 1049-1070.

154. Wikipedia (2015) Entry on "Schrödinger equation." Wikipedia, The Free Encyclopedia http://en.wikipedia.org/wiki/. Accessed July 7.

155. 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.

156. Walker HA, Cohen BP (1985) Scope statements: Imperatives for evaluating theory. American Sociological Review 50: 288-301.

157. Makeham WM (1867) On the law of mortality and the construction of annuity tables. Journal of the Institute of Actuaries and Assurance 13: 325-367.

158. Witten TM, Eakin T (1997) Multiphasic models of survival: Background and early developments. Experimental Gerontology 32: 259-285.

159. Yashin AI, Iachine IA, Begun AS (2000) Mortality modeling: A review. Mathematical Population Studies 8: 305-332.

160. Bebbington M, Lai C-D, Zitikis R (2007) Modeling human mortality using mixtures of bathtub shaped failure distributions. Journal of Theoretical Biology 245: 528-538.

161. Olshansky SJ, Carnes BA (1997) Ever since Gompertz. Demography 34: 1-15.

162. Smith D, Keyfitz N (1977) Mathematical Demography. Berlin: Springer-Verlag. 514 p.

163. Corbaux F (1833) On the Natural and Mathematical Laws Concerning Population, Vitality, and Mortality. London: W. Wilson.

164. Azbel' MY (1994) Universal biological scaling and mortality. Proceedings of the National Academy of Sciences 91: 12453-12457.

165. Azbel' MY (1996) Unitary mortality law and species-specific age. Proceedings of the Royal Society of London Series B: Biological Sciences 263: 1449-1454.

166. Azbel' MY (1999) Empirical laws of survival and evolution: The universality and implications. Proceedings of the National Academy of Sciences 96: 15368-15373.

167. Azbel' MY (1999) Phenomenological theory of mortality evolution: Its singularities, universality, and superuniversality. Proceedings of the National Academy of Sciences 96: 3303-3307.
168. Azbel' MY (2002) An exact law can test biological theories of mortality. Experimental Gerontology 37: 859-869.
169. Azbel' MY (2002) The law of universal mortality. Physical Review E 66: 1-9.
170. Azbel' MY (2003) Conservation laws of metabolism and mortality. Physica A: Statistical Mechanics and its Applications 329: 436-450.
171. Li T, Yang YC, Anderson JJ (2013) Mortality Increase in Late-Middle and Early-Old Age: Heterogeneity in Death Processes as a New Explanation. Demography 50: 1563-1591.
172. Golubev A (2009) How could the Gompertz-Makeham law evolve. Journal of Theoretical Biology 258: 1-17.
173. Milne EMG (2009) A new model of ageing and mortality. British Actuarial Journal 15: 213-234.
174. Milne EMG (2010) Dynamics of human mortality. Experimental Gerontology 45: 180-187.
175. 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.
176. Daepp MIG, Hamilton MJ, West GB, Bettencourt LMA (2015) The mortality of companies. Journal of The Royal Society Interface 12: 1-8.
177. Dowling MR, Milutinović D, Hodgkin PD (2005) Modelling cell lifespan and proliferation: is likelihood to die or to divide independent of age? Journal of The Royal Society Interface 2: 517-526.
178. Vaupel JW, Carey JR, Christensen K, Johnson TE (1998) Biodemographic trajectories of longevity. Science 280: 855-860.
179. Bourdieu P (1972) Esquisse d'une théorie de la pratique, précédé de trois études d'ethnologie Kabyle. Geneve: Droz.
180. Bourdieu P (1979) La distinction: Critique sociale du jugement. Paris: Les éditions de minuit.
181. Elias N ([1939] 1994) The Civilizing Process. Sociogenetic and Psychogenetic Investigations. Oxford: Basil Blackwell.
182. Mauss M ([1923-1924] 1950) Sociologie et Anthropologie. Paris: Presses Universitaires de France.
183. Wacquant L (2014) Putting habitus in its place: Rejoinder to the symposium, *Body & Society* 20: 118. doi: 10.1177/1357034X14530845.