The Janus Model of Life-Course Dynamics

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Content

Nascentes Morimur
From the moment we are born, we begin to die
(Marcus Manilius, 1st century AD)

Man is born, grows up, and dies, according to certain laws which have never been properly investigated, either as whole or in the mode of their mutual reactions.
(Adolphe Quetelet, 1835)

Introduction

The human life-course can be portrayed as a straight line with a beginning and an end, or – less simply – as a pyramid or platform with people of all ages arranged chronologically from birth to death. The traditional calendars of life are an excellent illustration of the archetypical view that the first half of life is a question of development or growth and the second half – almost inevitably – a matter of aging or decline. In 1835, the Flemish scientist Adolphe Quetelet broadened this view in *A treatise on Man and the Development of his Faculties*, the first research report on human development and aging. The data covered topics such as birth rate, mortality trends by age, stature, weight, and strength, as well as the development of the 'moral and intellectual qualities of man.' On the first page of the treatise, Quetelet describes an extensive research program for the study of life-course dynamics:

> "… they *(i.e. Quetelet's colleagues*) have neglected to put forward (….) the study of his physical *development* (bodily growth), and they have neglected to mark by numbers how individual man increases with respect to weight and height – how, in short, his forces are developed, the sensibility of his organs, and his other physical (*and mental*) faculties. They have not determined the age at which his faculties reach their *maximum* or highest energy, nor the time when they commence to *decline*." (p. 1) (italics added JS).

Currently, Quetelet's conception of the human life-course is known as a sequential two-phase model of growth and decline, which emphasizes the 'developmental' aspects of individual life.

A decade before Quetelet, the English actuary Benjamin Gompertz (1825) had emphasized the 'aging' aspects in a paper *On the Nature of the Function Expressive of the Law of Human Mortality*. He made the observation – based on the death and population records in England, Sweden, and France – that there is an exponential rise in death rates between the ages of 20 and 60, *i.e*. the ‘law of mortality.’ Partly due to the work of Makeham (1860), the age range was extended from 10 to 80 (Olshansky & Carnes, 1997). In the 20th century, Gompertz’s name was commonly linked to the full mortality curve of population data, from birth to death. It is important to note that this curve not only refers to the 20-60 or 10-80 age range, but also to the first age period of decreasing mortality from 0 to 10 or 20 years. In other words, the Gompertz curve is characterized by two sequential phases of decreasing and increasing mortality, commonly interpreted as development and aging (senescing), with the minimum varying from 10 to 20 or even 30 years. As such, the Gompertz curve could be conceived as the inverse of Quetelet’s growth and decline curve. Both curves confirm the traditional view that the human life course consists of two sequential processes of change, development and aging, with the transition point (maximum or minimum) at maturity or adulthood (Anderson, 1964). This view raises the crucial question of how the transition from developmental processes into aging processes can be explained.

Human ontogenesis

In 1960, Birren presented a general theory of aging as a counterpart of development. The use of the metaphor ‘counterpart’ is meant to express the idea that there are latent structures of behavior (emotions, cognitions, and motivations) – carried forward from earlier experience – that interact with present situations. Aging is viewed as a transformation of the biological and behavioral development of the organism expressed in a ‘counterpart manner’ in variable ecological contexts. Counterpart theory primarily describes the diachronic relationship between development and aging and does not explicitly address the issue of their synchronic relations. To fill the gap, Schroots (1982; Birren & Schroots, 1984) developed a simple diagram of human ontogenesis, much later called the ‘butterfly,’ in which development and aging are conceptualized as two parallel but related processes of change, or as the two sides of a unitary life trajectory (Figure 1).

The butterfly diagram demonstrates that at the start of ontogenesis (conception), the developmental process is most visible or manifest, while the signs of aging are at the time still obscure or latent – and *vice versa* at the end of life. It should be noted that across the lifespan, the transition point varies from function to function, from system to system, and from individual to individual.

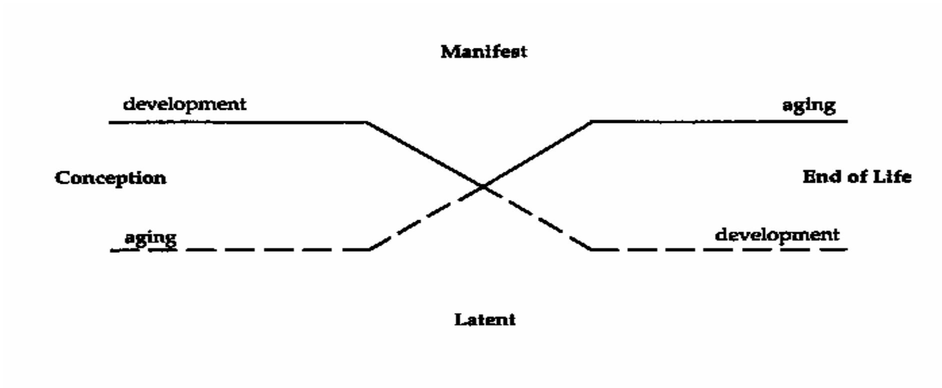

Figure 1. Butterfly diagram of human ontogenesis (Schroots, 1982; Birren & Schroots, 1984).

Figure 1 illustrates clearly the modern conception of development and aging as two simultaneous processes of change, from conception to death, which manifest themselves successively in the form of a unitary life trajectory (solid line). As such, the butterfly diagram functions as a metaphor for a dynamic life-course model.

Basic model

The terms model and metaphor are often used interchangeably in the research literature. Their relative significance, however, is expressed by the phrase that models are more general, extended and systematic metaphors (Schroots, 1991). A recent trend is for systematic metaphors to be formalized in mathematical terms. The question then becomes how the butterfly metaphor can be turned into a mathematical model fit for the computer simulation of life-course dynamics.

First of all, the widely interpretable terms and processes of 'development' and 'aging' should be reduced to the more elementary form of one-dimensional growth, which – based on the mathematical principle of iteration – follows an S-shaped power curve in which there is a limit to growth, *i.e.* the logistic or limited growth curve (van Geert, 1994; Verhulst, 1838). Mathematically, the logistic curve can be expressed in a differential equation in terms of either negative (1) or positive (2) growth, where the limit C is constant. Following the butterfly diagram, ‘development’ and ‘aging’ are then reduced to respectively negative (x) and positive (y) growth.

$$(1) \qquad \frac{dx}{dt} = - \frac{x\,(C - x)}{C} \qquad\qquad (2) \qquad \frac{dy}{dt} = \frac{y\,(C - y)}{C}$$

Second, the representation of ‘development’ as negative growth seems paradoxical, but harks back to Minot’s (1908) concept that the growth rate is highest at birth (or conception) and steadily declines thereafter (see Medawar, 1957). Following the same line of reasoning, ‘aging’ is conversely conceived as the process with the lowest growth rate at birth and the highest rate at the end of life. As such, the logistic x and y curves represent two complementary processes of change over the course of life, in respect of which the formula $x + y = C$ applies.

Third, from a dynamic systems perspective of the butterfly metaphor, the two lifespan processes of negative (x) and positive (y) growth are not only complements of each other, but are also united in the same living system V (Miller, 1978). Starting from the complementary equations (1) and (2), the next step in the development of a mathematical model should be the construction of the differential equation for V, which describes the vitality (V) of a dynamic system over the course of life.

Fourth, in 1825 the German philosopher Johann Friedrich Herbart introduced the concept of mental dynamics. He pointed out that the human mind includes all sorts of counteractive forces (ideas, concepts or *Vorstellungen*) involved in a permanent struggle for self-preservation. Herbart regarded this tendency as the fundamental principle of mental dynamics, taking into account that each of the forces’ movements is confined between two fixed points, *i.e.* their state of complete inhibition and their state of complete liberty. Therefore the interaction of two opposite forces, x and y, in respect of which $x + y = C$ applies, shows that the living system V has a systematic course in accordance with Herbart’s formula $xy / (x + y)$, or xy / C (see Boring, 1950, pp. 250 – 260). By means of this formula, equations (1) and (2) can now be rewritten in terms of variable V. Mathematical coupling of the two equations eventually results in the desired differential equation (3), which describes the life trajectory or life-course dynamics of a living system:

$$\frac{dV}{dt} = \frac{V(-x+y)}{x+y} = \left[\frac{-x+y}{C}\right] V \tag{3}$$

Equations (1), (2) and (3) form the basis of a dynamic life-course model, which can be simulated on the computer with Stella software (2006). Computer simulation of this *basic model* over time shows that (a) the relative distributions of *x* and *y* follow two crossing patterns of a negative (1) and positive (2) growth curve, and that (b) the distribution of *V* follows a bell-shaped curve (3) representing the vitality of the dynamic system under study (Figure 2).

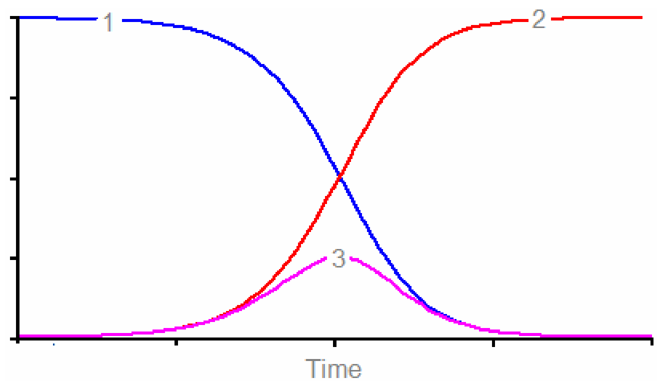

Figure 2. Computer simulation of the *Basic* model:
curve 1: negative growth curve *x*
curve 2: positive growth curve *y*
curve 3: bell-shaped curve *V*

Fifth, generally speaking, the terms ‘positive’ and ‘negative’ growth are respectively associated with development and aging. In the basic model, these terms have to be used in the reverse order. To avoid semantic confusion, the term ‘force of senescence’ will be used for processes of ‘positive growth’ (*y*) and the term ‘force of growth’ for processes of ‘negative growth’ (*x*). According to this new terminology, figure 2 clearly illustrates that the two simultaneous forces of growth (*x*) and senescence (*y*) produce a unitary life trajectory (*V*), traditionally interpreted as the succession of development and aging, with the apex at the maximum of the system's life-course.

Summarizing, the basic model explains the transition from development into aging. From a theoretical perspective, this is called the principle of *transition*.

Extended model

Symmetrical, bell-shaped curves as described by the basic model are seldom found in nature. Growth and senescence are, indeed, simultaneous and complementary forces, but the rate of these isochronous forces may vary mutually, depending on the function or system in question. Differing rates of growth and senescence will produce more natural, asymmetric bell curves with the mode moving accordingly. Equation (3) therefore needs to be extended with two parameters, p and q, according to the formula $pxqy /(px + qy)$, in which p represents the rate of growth and q the rate of senescence (see Schroots & Yates, 1999). Computer simulation (Stella, 2006) of the *extended model* for differing values of p and q results in equally differing (a)symmetric distributions of V over the course of the system's lifespan (Figure 3). Note that for easy reference, the lifespan is set at 100 years and that the negative and positive growth curves are omitted in the figures.

In order to demonstrate the effect of p and q apart and in combination, one parameter was fixed and the other varied. Figure 3 shows that if q is fixed (curves 1 and 2, as well as curves 3 and 4), the maximum level of V varies with the growth rate p, while the (symmetric) distribution of V becomes more or less negatively skewed. However, if p is fixed (curves 1 and 3), then both the maximum and distribution of V vary with the rate of senescence q. Generalizing, parameter p therefore primarily controls the *level* of maximum vitality (i.e. the peak capacity of the system), while parameter q determines the *time* of maximum vitality (i.e. the chronological age of the system's vitality peak). Note that if p equals q (curve 1), the distribution of V is similar to the symmetric, bell-shaped curve (3) as presented in figure 2.
The connection between the extended model and the sequential two-phase model of development and aging is also illustrated in figure 3. The four curves or life trajectories are traditionally interpreted in terms of two sequential processes of change, development and aging, with the

apex as point of transition. Following the extended model, the question could be asked how development and aging relate to each other if the rates of growth and/or senescing change.

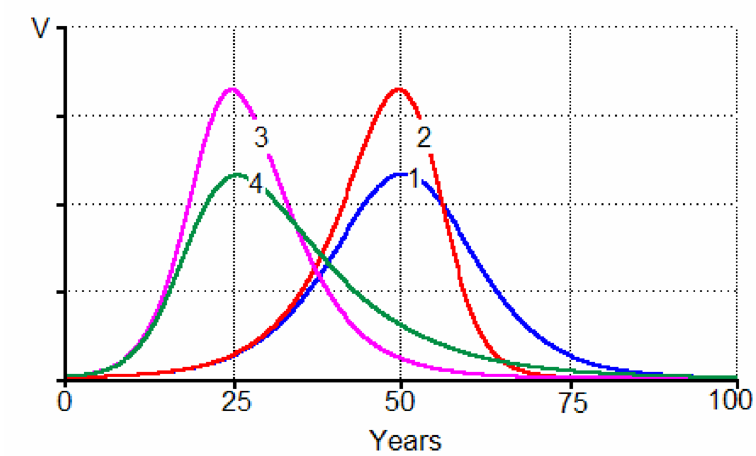

Figure 3. Computer simulation of the *Extended* model:
curve 1: $p = 0.14$; $q = 0.14$
curve 2: $p = 0.30$; $q = 0.14$
curve 3: $p = 0.14$; $q = 0.30$
curve 4: $p = 0.08$; $q = 0.30$

The symmetric, bell-shaped curve (1) that is based on the principle of *transition* serves as a reference for curves (2), (3), and (4) and nicely illustrates that the two phases of development and aging cover equal time spans for equal rates of growth and senescence. Curves (1) and (2), as well as curves (3) and (4) demonstrate the second principle of *peak capacity*: the growth rate determines the system's maximum capacity at the end of its development. A comparison of curves (1) and (2) with curves (3) and (4) demonstrates the third principle of *peak time*: the rate of senescence controls the age at which the system reaches its maximum capacity, that is, at the end of development and the beginning of aging.

The second and third principle have an important corollary in accordance with the well-known sayings *soon ripe, soon rotten* and *live fast, die young*. The second principle of peak capacity relates to the phenomenon that rapid growth in the development phase leads to rapid decline (soon ripe, soon rotten), with the result that *V* reaches its critical capacity for survival at a younger age than it would have with slow growth, if the rate of senescence is constant. Note, for example, the rapid growth and steep right tail of curves 2 and 3 in figure 3 compared with the slower growth and flatter right tails of curves 1 and 4, respectively. The effect of rapid senescence (third principle of peak time) is even more dramatic: higher rates of senescence with constant growth rate mean that the system reaches its peak and critical threshold at an increasingly early age (live fast, die young) (see curves 3 and 4 vs. curves 1 and 2 in figure 3).

Summarizing, the extended model explains (a) the transition of development into aging (1st principle), and (b) the transition point in terms of peak capacity and peak time (2nd and 3rd principle, respectively).

Validity of the extended model

The proof of the pudding is in the eating. In the foregoing it has been shown that the synchronic forces of growth and senescence, formalized in the extended model, offer a satisfactory account of the life-course dynamics of living systems. The model's validity for empirical data can, however, be questioned. One way to test the validity is by defining the optimal fit between model and data.

Since Gompertz and Quetelet, the life trajectories of numerous bodily functions have been studied empirically. In 1993, Kemper and Binkhorst presented the idealized life trajectory of general physiological performance (after Smith & Serfass, 1981). They noted that, in general, peak performance is reached at about age 30. After that time, functional capacity declines gradually at varying rates, depending on the individual and the organ system. In figure 4, both the (idealized) empirical curve of functional capacity (dotted line) and the simulated curve (bold line) are presented for ages 10–90. The simulation was executed with the Madonna program by Macey, Oster, and Zahnley (2000), which finds those values in the model that minimize the deviation between the model's output and functional capacity's empirical dataset, *i.e.*, the root mean square of the differences between individual data points in the dataset and the corresponding points of the model as run by the program.

Figure 4 shows the graphical outcome of the fitting procedure, resulting in an almost perfect fit between the functional capacity data (dotted line) and the model's output (bold line). In this context, it should be noted that the scale of the functional capacity plot (X-axis = 0-100 yrs; Y-axis = 0-120%) defines the initial values of the forces of growth (*x*) and senescence (*y*), as selected by the program for an optimal model and data fit.

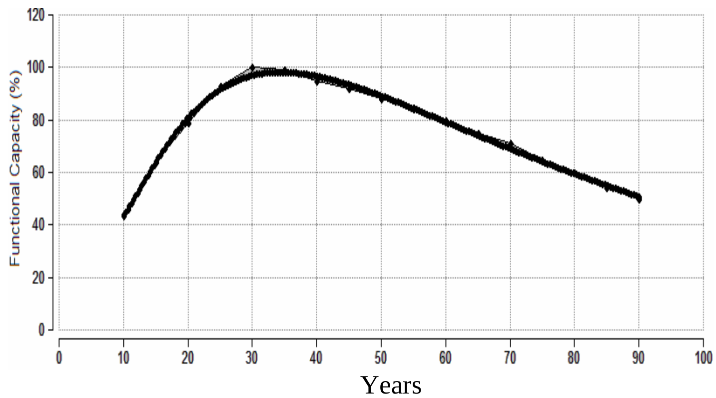

Figure 4. Optimal fit of functional capacity (dotted line) and extended model (bold line) for parameter values p = 0.018 and q = 0.179 (initial values: x = 9229 and y = 329).

Since the Gompertz curve can be regarded as the inverse of a growth and decline curve, the simulation of mortality data may also be used to support the extended model's validity. In 1988, Schroots presented the idealized age-specific mortality curve for the 1940 US population (adapted from Fries & Capro, 1981; see figure 5). Initially, the simulation of the full mortality data curve with the Madonna program failed due to the extreme range of values in the age-specific mortality plot: (a) logarithmic Y-axis (0.1-1000) with mortality rates varying from ca. 0.4-400 deaths per year per 1000 individuals entering each age, and (b) lowest mortality rates in the lower age range (ca. 10-30 yrs) of the mortality plot. In preliminary trials, it was found that the extended model is more or less capable of covering the first section of the mortality curve, *i.e.* the developmental phase of decreasing mortality, but can barely cover the second section of increasing mortality, *i.e.* the aging phase.

However, if the semi-logarithmic mortality data are transformed into a linear scale before they are imported into Madonna, the modeling problem is solved. In figure 5, both the mortality curve (dotted line) and simulated curve (bold line) are presented for the ages 0-90. The left Y-axis (0-100), labeled Mortality (T), refers to the linear scale of the transformed mortality data; the right Y-axis, labeled Mortality Rate, reflects the original, logarithmic scale. It is important to note that the transformation of mortality data does not affect the form and dynamic structure of the mortality plot (see D'Arcy Thompson, 1917).

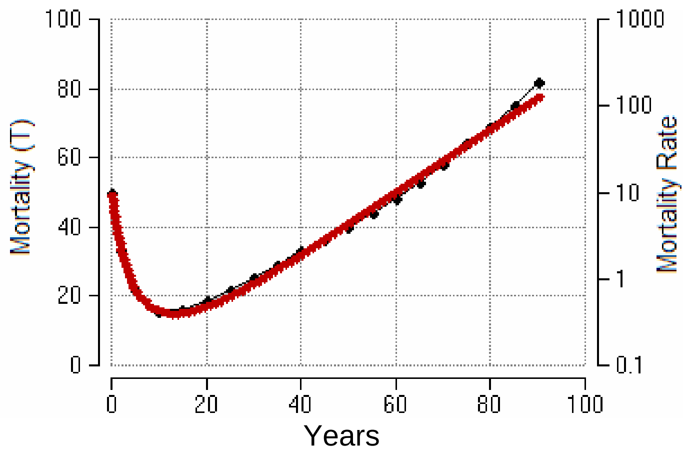

Figure 5. Optimal fit of mortality (dotted line) and extended model (bold line) for parameter values p = 0.003 and q = 1.306 (initial values: x = 145873 and y = 1691).

Figure 5 shows an almost perfect fit between mortality data (dotted) and the model's output (bold). As such, the outcome of the fitting procedure supports the view that the idealized mortality curve for population data from birth to death should be conceived as (the inverse of) a simple growth and decline curve, subject to the hypothetical lifespan forces of growth (x) and senescence (y). The high initial values of these forces suggest, however, that the extended model has reached its limits regarding extreme data points.

Summarizing, the extended model offers a satisfactory account of the dynamics of relatively simple growth and decline functions, both at the biological and demographic level.

Janus model

Simple growth and decline curves show two phases and one transition; complex curves, however, come in all shapes and sizes. The mortality curve in figure 5, for example, is a simple, idealized curve, of which the age period between 10 and 30 years has been smoothed for the sake of convenience. In reality, however, there is an upward sloping of the mortality rate from age 10 (lowest probability of dying), eventually reaching a steady slope some time after 30 years of age (see Fries & Crapo, 1981). From the perspective of life-course dynamics, this means that the human mortality curve has three instead of two phases (0–10 yrs, 10–30 yrs, 30–90 yrs) and two minima or transitions instead of one at 10 and 30 years, respectively. Consequently, a sequential three-phase model of growth, relative stability, and decline would be more appropriate for the simulation of complex curves than the two-phase extended model. The life-course dynamics of general intelligence, which is rooted in the central nervous system (CNS), may serve as an example for the construction of such a model.

Traditionally, general intelligence is divided into two factors (abilities): fluid and crystallized intelligence (Cattell, 1963). The ontogenetic patterns of both abilities show a rapid rise until early adulthood followed by a period of relative stability for the crystallized abilities until the age of about 60-70, but a distinct decline in the fluid abilities after early adulthood (Garlick, 2002). The question is therefore how the differential course of these abilities over the lifespan can be explained. The answer can be argued as explained in the following paragraphs.

From a dynamic systems perspective, general intelligence is conceived as a living system divided in two subsystems: fluid and crystallized intelligence. Both subsystems are localized in the CNS and subject to forces of growth and senescence. In principle, the extended model accounts for each individual (sub)system. The subsystem of fluid intelligence relates primarily to the *speed* of information processing, whereas the subsystem of crystallized intelligence relates to the *storage* of information. Information processing precedes storage of information. From this it follows that crystallized intelligence is composed of both information processing and storage of information. In other words, crystallized intelligence rides piggyback on fluid intelligence, which explains the period of the crystallized abilities' relative stability after early adulthood.

The above argumentation can be easily generalized to other growth and decline curves like the mortality curve. The point is that the differential equation (3) of the extended model needs to be enlarged with an extra mathematical term that is equal to the original equation. Mathematical coupling of the two differential equations will then result in differential equation (4) of what has been called the *Janus* model after the Roman god with two faces – one face looking into the future and one into the past.

$$\frac{dJ_V}{dt} = \frac{V_1(-x+y)}{x+y} + \frac{V_2(-v+w)}{v+w} \tag{4}$$

Analogous to the extended model formula, the *Janus model* formula for two living (sub)systems is as follows:

$$J_V = V_1 + V_2 \tag{5}$$

in which $V_1 = pxqy / (px+qy)$ and $V_2 = rvsw / (rv+sw)$, and parameters p and r represent the rate of growth, and parameters q and s the rate of senescence. Taking the Janus model one step further, differential equation (4) and formula (5) can be respectively generalized to equation (6) and formula (7) of the Janus model of n living systems:

$$\frac{dJ_V}{dt} = \sum_{k=1}^{n} \frac{V_1(-x+y)}{x+y} + \frac{V_2(-v+w)}{v+w} \tag{6}$$

$$J_V = \sum_{k=1}^{n} V_k \tag{7}$$

Summarizing, in principle, the Janus model explains the life-course dynamics of living systems with n transitions.

Validity of the Janus model

In order to test the validity of the Janus model of two coupled systems ($V_1 + V_2$), two empirical intelligence and mortality data sets are respectively simulated with the Madonna program (Macey, Oster & Zahnley, 2000). Note that both data sets have primarily been selected for their potential to demonstrate the Janus model's validity.

Intelligence

The first data set is extracted from the Li et al. (2004) cross-sectional study on intellectual abilities across the lifespan. Li and colleagues collected intelligence data on a stratified (age, sex) German sample of 356 participants between 6 and 88 years old, and presented the crystallized (Gc) and fluid (Gf) intelligence's group means in a graph (p. 158). The graph data copied from this study were imported into Madonna and the fit between the Janus model and the intelligence data defined. In figure 6, the empirical data of Gc and Gf, as well as the simulated Janus curves Jf and Jc are presented for the ages 6-88. The data are plotted as T-scores for comparison purposes.

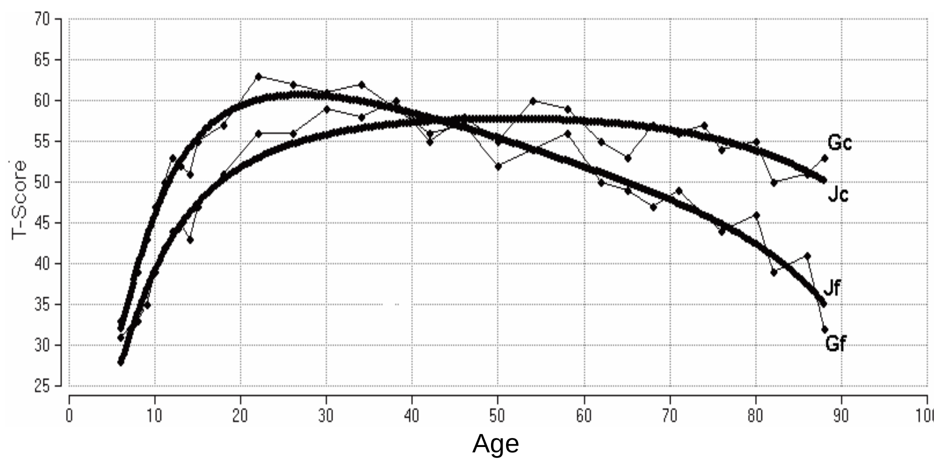

Figure 6. Computer simulation of fluid and crystallized intelligence (dotted lines) with the Janus model (bold lines): Fit between Janus curves (Jf, Jc) and intelligence plots (Gf, Gc).

Figure 6 shows a satisfactory fit between the Janus curves and the Gf and Gc data plots. In view of the complexity of the curves, the interpretation is not simple, but could be as follows: the first step concerns the traditional interpretation in terms of development and aging with the peak of intellectual abilities at the point of transition, *i.e.* the extended model. The fluid (Jf) intelligence curve then shows a peak at ca. 27 years, while the crystallized (Jc) curve peaks at ca. 50 years. It should be noted that fluid intelligence follows the traditional pattern of development (6-27 years) and aging (27-88 years), but that in contrast with traditional conceptions (see Garlick, 2002), crystallized intelligence continues to develop from the age of 27 to the peak age of ca. 50, after which the aging phase (50-88 years) starts.

The second step follows an analytical approach to the Janus model of two coupled systems, in which the composite Janus curves are resolved into system curves V_1 and V_2. Figure 7 presents both the Janus curves (Jf, Jc), the V_1 curves (V1f, V1c), and the V_2 curves (V2f, V2c) of the Gf and Gc data plots. For convenience, the data plots are omitted from the figure. Note that due to graphical limitations, the exact bifurcation points of the Janus and system curves only become visible after enlargement of the figure.

Starting from the two system curves V_1 and V_2 (formula 5), the Janus curves of fluid and crystallized intelligence consist of three phases and two transitions. Visual inspection of the enlarged figure shows that the first transition of the fluid Janus (Jf) curve coincides with the transition of the V1f curve at about 27 years. The peak age of both the fluid curve and V1f curve is controlled by the product of parameter *q* and initial value *y* (see the legend of figure 7). Generally, the V1f curve corresponds to the simulated curves of simple growth and decline functions as produced by the extended model (see figure 4). The second transition of Jf, controlled by s*w, coincides with the transition of the V2f curve at the age of ca. 50. The second Jf transition reflects the onset and impact of the second system V2f, of which the T-score is practically zero until the age of 50 and then becomes negative for the rest of its life trajectory. This second system makes the fluid curve bend down for a second time. The theoretically unexpected discrepancy between the Janus curve of fluid intelligence and the V1f curve at the older age comes as a surprise. Not only does the Li et al. (2004) study report an apparently good fit between statistical model and data, comparable to fluid data's extended model (V1f), but the Janus model reveals a second, to date unknown, system's mid to late-life impact on fluid abilities that apparently accelerates the aging process.

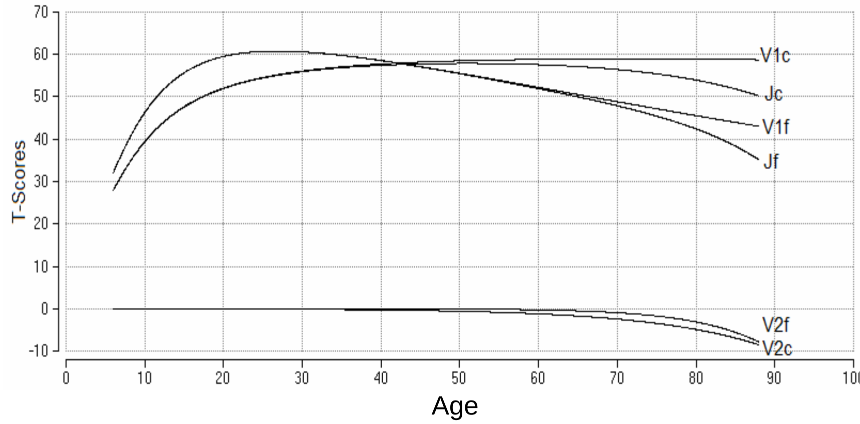

Figure 7. Janus curves (Jf, Jc), V_1 curves (V1f, V1c) and V_2 curves (V2f, V2c) of fluid and crystallized intelligence. Legend:

Janus curve	V_1 parameter	V_1 initial value	V_2 parameter	V_2 initial value
Jf	p = 0.008	x = 9988	r = -0.201	v = 1176
	q = 0.390	y = 139.7	s = 0.120	w = -0.004
Jc	p = 0.001	x = 87359	r = -0.163	v = 0.073
	q = 0.390	y = 127.47	s = 0.120	w = -1.552

The Janus curve of crystallized intelligence (Jc) also displays two transitions, as expected from the Janus model. The first transition of Jc coincides with the transition of V1c at the age of ca. 27 and is controlled by q*y. Expectations are that the second transition of Jc, controlled by s*w, will coincide with the transition of V2c and the peak of Jc at the age of about 50. However, visual inspection of the enlarged figure shows that the onset of second system V2c starts at ca. 27 years, but that the negative impact of the system on the crystallized Janus curve only becomes manifest after 50. The onset in early adulthood and the midlife impact of the second system V2c on crystallized intelligence should not come as a surprise, because crystallized intelligence rides piggyback on fluid intelligence. The latter goes downhill after the peak age of 27 and accelerates in midlife from about 50 years. In other words, the downward change of crystallized abilities after the 50-year peak can be explained by the second transition of fluid abilities at about the same age.

As discussed before, the traditional view of fluid intelligence is primarily based on the neurobiological property of information processing speed (Birren & Fisher, 1995). As such, the dynamics of the fluid system generally corresponds with the life trajectories of many other biological systems that reach their peak performance in early adulthood and decline afterwards (Sehl & Yates, 2001; see also fig. 4). The Janus model of two coupled systems has shown that there must be a second system, emerging in young to middle adulthood, which accelerates the slowing of fluid intelligence and initiates the decline of crystallized intelligence. The latter relates primarily to the storage of information, but must process the information before it can be stored or retrieved.

Mortality

The second empirical data set relates to mortality and is borrowed from Fries and Crapo (1981, p. 29) who presented a graph of vital statistics from the United States for 1910 and 1970. For scale-technical reasons, the semi-logarithmic mortality data were transformed linearly before the copied graph data were imported into the Madonna simulation program. Figure 8 presents the empirical mortality data for the United States in the years 1910 (M_{10}) and 1970 (M_{70}), as well as the simulated Janus curves J_{10} and J_{70}. Note that the transformed (T) linear scale of the left Y-axis (0-100) is labeled 'Mortality (T)' and that the original, logarithmic scale of the right Y-axis (0.1-1000) is labeled 'Mortality Rate.'

Figure 8 shows an almost complete overlap between the Janus curves (bold) and the mortality data (dotted). As such, the fit between the Janus curves (J_{10}, J_{70}) and data plots (M_{10}, M_{70}) is more than satisfactory. Both Janus curves can be characterized in terms of development (decreasing mortality) and aging (increasing mortality), with the lowest probability of death (minimum) at the age of about 10. However, there are also differences between the J_{10} and J_{70} curves. First, the minimum mortality was much higher in 1910 than in 1970. This huge drop in mortality is generally attributed to the improvement in the overall health of western populations. Second, the Janus curves, particularly the J_{70} curve, are not smooth over the ages 10–30 (J_{70}) and 10–45 (J_{10}); this irregularity or 'bump' in the mortality curve represents traumatic deaths (accidents), which peak during these ages.

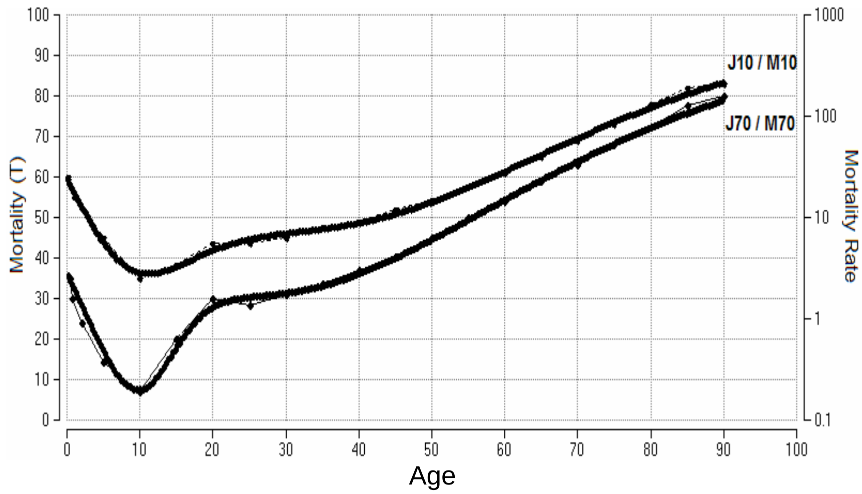

Figure 8. Computer simulation of the US mortality in the years 1910 and 1970 (dotted) with the Janus model (bold): Fit between Janus curves (J_{10}, J_{70}) and mortality (M_{10}, M_{70}) plots.

In this context, Fries and Capro (1981) maintained that since 1910 "the relative importance of trauma has increased greatly; such deaths made up nearly 75% of all deaths between ages 15 and 25" (p. 28). After the 'bump' period, the mortality rate of both curves displays an upward slope until the age of ca. 90.

From an analytical perspective, the composite Janus curves can be resolved into system curves V_1 and V_2. In figure 9, the Janus curves, as well as the V_1 and V_2 curves are presented for the years 1910 (top panel) and 1970 (bottom panel). The data plots have been omitted from the figure for convenience and visibility. Note that due to graphical limitations, the exact bifurcation points of the Janus and system curves only become visible after enlargement of the figure.

Figure 9 indicates that the minimum mortality of both the Janus curves practically coincides with the transition (minimum) of the V_1 curves at the age of ca. 10. The V_1 minimum mortality in both years is controlled by q*y (see legend of figure 9). After visual inspection of the V_2 curves in the enlarged figure, it was determined that the V_2 mini-

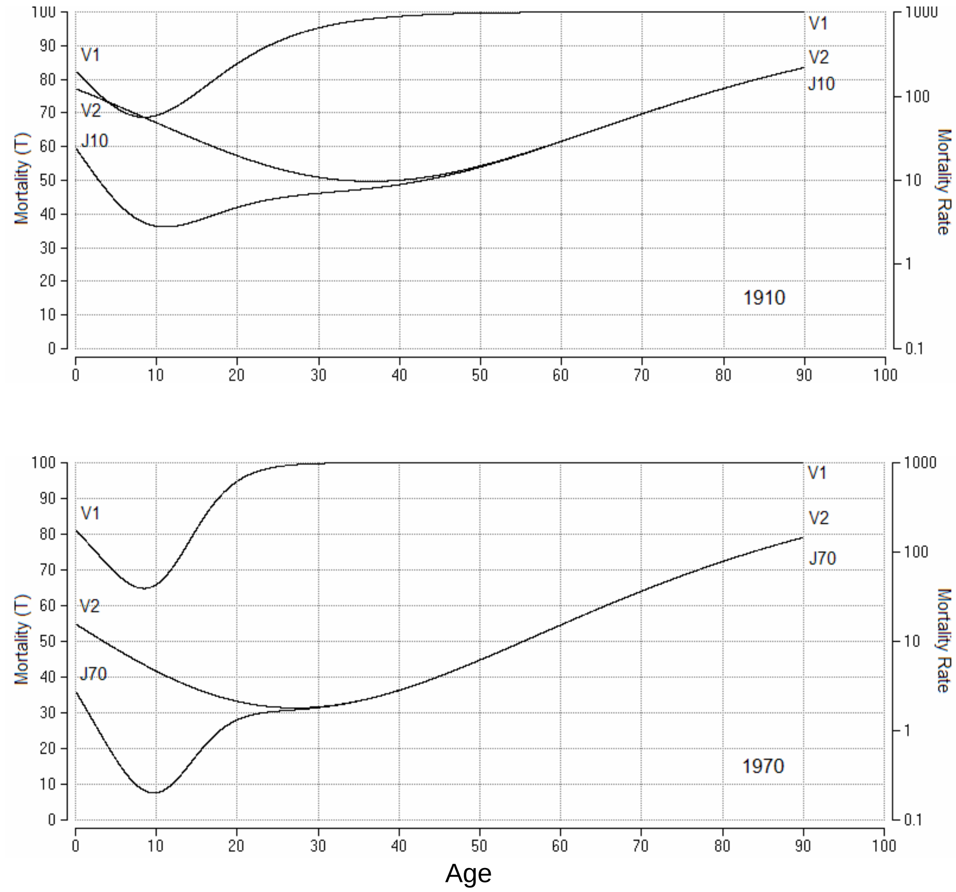

Figure 9. Top panel: Janus curve (J_{10}), V_1 and V_2 curves of US 1910 mortality.
Bottom panel: Janus curve (J_{70}), V_1 and V_2 curves of US 1970 mortality. Legend:

Janus curve	V_1 parameter	V_1 initial value	V_2 parameter	V_2 initial value
J_{10} (1910)	p = 0.134 q = 0.223	x = 633 y = 101	r = 0.039 s = 0.059	v = 3783 w = 459
J_{70} (1970)	p = 0.327 q = 0.160	x = 500 y = 135	r = 0.033 s = 0.062	v = 5230 w = 990

mum for 1970 (bottom panel) coincides with the Janus curve at the end of the 'bump' period (ca. 27-30 years), and that the V_2 minimum for 1910 (top panel) is reached at the age of ca. 37. The latter is well before the end of the 'bump' period (ca. 45-50 years). Note that the minima of the V_2 systems is controlled by s*w (see the legend of figure 9). The question arises how the first (V_1) and second (V_2) system in the Janus model framework should be interpreted.

With regard to the first system, the interpretation should not be too difficult, *i.e.* the V_1 curve reflects the impact of environmental and pathological conditions, as well as of accidents on individual lives. The reasoning is as follows: Given negative prenatal, perinatal, and postnatal conditions (*e.g.* congenital defects, infectious diseases), infant mortality will be high and, consequently, the impact of accidents in adolescence and young adulthood will be relatively low, as is shown for the year 1910 in which the 'bump' is hardly visible. However, if the overall health conditions are improved, as in 1970 western society with its public services (*e.g.* potable drinking water, community health programs, sewers, etc.), infant and childhood mortality will be low. Consequently, the 'bump' mortality of adolescence and young adulthood emerges relatively distinct from what is presumably the natural, intrinsic mortality of human beings as reflected by the V_2 curve (see bottom panel of figure 9). In other words, the first system of 'extrinsic' mortality dominates the second system of 'intrinsic' mortality, but in combination they produce the full Gompertz curve as simulated by the Janus model.

On the whole, the V1 curve primarily reflects the shift in the mortality dynamics of an increasingly healthy (or unhealthy) population (see Finch & Crimmins, 2004). The V_2 curve, on the other hand, expresses the basic mortality dynamics in the form of an inverse growth and decline function that is intrinsic to the human organism, and relatively independent of the environment.

Summarizing, the Janus model of two coupled systems offers a quite satisfactory account of the life-course dynamics of complex growth and decline functions as demonstrated in respect of empirical data sets of intelligence and mortality.

Discussion

In Roman mythology, Janus was the two-faced god of gates and doorways, beginnings and endings. He was frequently used to symbolize change and transitions such as the progression of the future to the past or the transition of development into aging. Janus was also known as the figure representing time because he could see into the past with one face and into the future with the other. Hence, it seemed appropriate to name the mathematical model of life-course dynamics after the Roman god Janus.

Postulates

The simple version of the Janus model (two phases, one transition) is based on the simultaneous and complementary action of two coupled forces, growth and senescence, which determine the dynamics of living systems, or – to put it differently – define the life trajectories of dynamic systems. Note that the forces of growth and senescence should be conceived as postulates of the same order as the physical force of gravity, which does not as yet have an explanation. The term ‘living system’ is extracted from Miller’s (1978) systems theory, which states that humans are primarily regarded as living systems, hierarchically organized from many subsystems such as cells, cell tissues, organs, etc., according to their complexity levels. As a system, humans can be conceived as part of an even more complex, larger system – for example, the social and physical environment. From the latter point of view, it depends on the system level whether the term ‘living system’ or ‘dynamic system’ is used. Whatever term is selected, the Janus model is primarily a mathematical, ‘empty’ model that fits the growth and decline curves of widely divergent systems from biological and psychological systems to social and demographic systems. As such, the Janus model has proved its validity.

Principles

The construction of the Janus model revealed approximately three principles. The first principle of *transition* solved the traditional problem of the age at which development ends and the process of aging starts. This principle stated that the apparent unitary lifespan trajectory of development and aging is in fact the product of two complementary forces, growth and senescence, which are effective from conception until death. It is assumed that with more expertise based on the Janus model, it would be possible to refute ‘ageist’ opinions of older people’s growth potential or, conversely, of young people’s senescence risk. The second and third principles of *peak capacity* and *peak time* refer respectively to the impact of growth rate (peak capacity) and rate of senescence (peak time) on the life-course of dynamic systems and of human beings in particular. Different growth rates with a constant rate of senescence have implications for the peak capacity and the residual lifespan after the transition point. Rapid growth, for example, leads to a higher peak at a certain age, but also to rapid decline and a shorter residual lifespan than slow growth, which results in a lower peak, slower decline, and a longer residual life trajectory after the point of transition. On the other hand, different rates of senescence with a constant growth rate mainly have implications for peak time (age), peak capacity, and the total lifespan. Rapid senescence, for example, results in a higher peak at a younger age, but also in a shorter lifespan than slow senescence with a lower peak at an older age and a longer total lifespan. Generally, growth rate therefore defines the system’s maximum capacity (2^{nd} principle) and the rate of senescence defines the age at which the system reaches its maximum capacity (3^{rd} principle).

Limitations

The latter statement deserves a *caveat* because the forces of growth and senescence are confounded unless one of the two parameters is kept constant, for example, in some quasi-experimental design in which the growth parameter is manipulated while the system’s peak age is fixed. In this respect, it should be noted that the parameters, as well as the initial values of the forces of growth and senescence determine the Janus model’s output in terms of fit between the empirical and simulated curve. So far, no direct solution has been found for the initial values and parameters’ confounding effects on the modeling procedure outcome, but it is assumed that the introduction of standard scaling methods will solve the calibration problem of finding and interpreting the different parameter combinations (for an overview, see Kirkwood et al., 2006). A final remark concerns the Janus model’s limited capacity to fit wide ranging data plots like skewed, semi-logarithmic distributions. The transformation and back-transformation of data plots in the same graph were necessary to achieve a satisfactory model fit.

New findings

Validation of the Janus model of two coupled systems led to some surprising discoveries. First, by simulating *intellectual abilities*, the Janus model revealed a second, to date unknown, system's mid to late-life impact on the fluid (and crystallized) abilities. This second system accelerates the fluid decline in the second half of life. In view of the close connection between neurobiological substrate and fluid intelligence, it is hypothesized that this unknown system reflects the disorganization, disintegration or dedifferentiation of the human organism from middle age, which might eventually result in the terminal drop in intellectual abilities at the end of life (Berg, 1996). It would be interesting to learn whether the Janus model could also demonstrate the existence of this second system in respect of biological functions and organ systems.

Second, by simulating US *mortality* for 1910 and 1970, the Janus model of two coupled systems could resolve overall mortality in two components, tentatively labeled intrinsic and extrinsic mortality. The first component of extrinsic mortality (1^{st} system) reflects the fatal impact of environmental and pathological conditions, as well as accidents on individual lives. The extrinsic mortality curve indicates a minimum in respect of the age of ca. 10 and extends from birth to about the age of 50 with regard to the 1910 data and to about age 30 with regard to the 1970 data. According to the third principle of peak time, this means that the rate of senescence in the first system is constant for both 1910 and 1970. The second principle of peak capacity (growth rate) thus explains the differences between the first system's 1910 and 1970 curves with regard to the minimum and residual trajectories. In other words, the rapid decrease in negative conditions in 1970 leads to a lower minimum mortality and shorter residual trajectory (ca. 10-30 years) than the slow decrease in 1910 with its higher minimum mortality and longer residual trajectory (ca. 10-50 years). From this perspective, the 1910 and 1970 extrinsic mortality curves serve as example of a quasi-experimental, demographic design with a variable peak capacity and constant peak time.

The second component of intrinsic mortality reflects the human organism's inherent, natural capacity to adapt to life. The intrinsic mortality curve (2^{nd} system) extends over the full lifespan (0-90 years) for both 1910 and 1970, with minimum mortality from about age 37 in 1910 to the age of 27 in 1970. Note that the age shift of minimum intrinsic mortality from 37 to 27 years is coupled with an improvement in living conditions and lower extrinsic mortality. Note, moreover, that the minimum intrinsic mortality in 1970 (ca. 27 years) corresponds to the peak of general physiological performance at about the age of 30 (see Smith & Serfass, 1981). It is therefore not unreasonable to assume that intrinsic mortality's inverse growth and decline curve reflects some basic survival mechanism, also called adaptability or functional fitness, which reaches its maximum strength at the age of ca. 30. Generally, therefore, the environment's impact on mortality dominates in the first 30 to 50 years of life and from then onwards our mortal nature becomes manifest. It would be interesting to learn whether the Janus model of two coupled systems could clarify other nature-nurture problems as well.

From model to theory

There is nothing as practical as a good theory for the development of an empirical science (Lewin, 1951). A theoretical framework helps the scientist to accumulate and integrate data into a solid body of knowledge, as well as to provide directions for new research. Unfortunately, Birren's (1995) dictum on gerontology still contains an essential truth: "the study of aging has become a field of knowledge that is data rich and theory poor, a vast collection of unintegrated pieces of information" (p. 1).

In this article, an attempt has been made to integrate data and theory on the basis of a mathematical model that connects theory with empirical research. It should be noted that in themselves, the differential equations of the Janus model are not the model or theory. The equations are only a model because they represent the life-course or life trajectories of dynamic systems as defined in the respective disciplines of biology, psychology, demography, etc. Once interpretation and context are added, the mathematical model loses its separate identity, and the scientist finds him or herself in the process of theory development.

Schroots and van Dijkum (2004) took the first step in theory development by simulating the A(utobiographical) M(emory) bump with a precursor of the Janus model. Autobiographical memory generally obeys the classic principles of remembering and forgetting, in which the distribution of memories, also called the 'forgetting curve', follows a power function. Contrary to this is the AM bump phenomenon of a disproportionally higher recall of memories in respect of the age period 10 to 30, as observed in middle-aged and older adults. After the successful simulation of the autobiographical memory curve, Schroots and van Dijkum concluded that the lifespan forces of growth and decline (senescence) offered the first satisfactory account of the dynamics of the AM bump, due to the relatively more intensive, neural encoding and storage of information between 10 and 30 years of age. Using this conclusion as their point of departure, they proposed a dual process theory of ontogenesis, stating that the lifespan forces of growth and senescence could also explain the differential course of other neurobehavioral functions.

Development of a Janus prototheory

The Janus model has so far produced two important constructs simply labeled 'peak' and 'bump.' A peak age of ca. 30 has been found for all sorts of organismic growth and decline curves, which has been denoted by means of terms like maximum functional capacity (physiology), maximum speed of information processing (psychology), or minimum intrinsic mortality (demography). The peak age of ca. 30 across widely different systems suggests the existence of a fundamental, non-specific, vital system that is typical of human life and reaches its full power around 30 years (vitality peak), thereafter declining. It is assumed that this non-specific, vital system reflects the life-course dynamics of numerous biological and psychological functions (*e.g.*, functional capacity, fluid intelligence), as well as - indirectly - the dynamics of intrinsic mortality over the lifespan. The question then arises how the peak age of this vital system relates to the bump of 10-30 years as observed, for instance, in the curves of mortality and autobiographical memory (AM). According to the Janus model of two coupled systems, the answer is easy: in addition to the primary system, there must be a second system that emerges with puberty at age 10 and reflects neurohormonal changes. The interaction of this system with the primary system explains both the increased mortality rate between 10 and 30 years and middle-aged and older adults' relatively higher recall of memories from adolescence and young adulthood. Generally, the bump therefore reflects the dynamics of two interacting systems.

Steinberg (2004) produced evidence from developmental neuroscience to explain the mortality bump of accidents (10-30 years) as due to risk-taking behavior in adolescence. He states that neurohormonal changes at puberty cause a temporal disjunction of two neurobehavioral systems: the socioemotional and the cognitive-control system. The cognitive control system subserves executive functions such as planning, thinking ahead, and self-regulation and is located in the outer regions of the brain (including the lateral prefrontal and parietal cortices and parts of the anterior cingulate cortex). The brain's socioemotional system, however, is located in the interior regions of the brain (including the limbic and paralimbic areas, amygdala, ventral striatum, orbitofrontal cortex, medial prefrontal cortex, and superior temporal sulcus), which are especially sensitive to social and emotional stimuli from a person's environment (*e.g.* peers, arousal) and to reward processing (*e.g.* gratification). Maturation of the socioemotional system appears to be driven by puberty, whereas maturation of the cognitive-control system does not. Different rates of maturation of the socioemotional and cognitive-control system, which cause temporal friction between a person's rational and emotional behavior, would therefore explain the bump of fatal accidents in adolescence and young adulthood.

Recapitulating the most important findings of the Janus model, the construction and validation of the model yielded a set of postulates, principles and constructs, which almost unnoticed constitute a frame of reference for a prototheory of life-course dynamics (see Schroots, 2003). As it turns out, this Janus prototheory *in statu nascendi* explains the mechanisms of the bump phenomenon in three different domains of research: mortality, autobiographical memory, and developmental neuroscience. Expectations are that the Janus theory *in statu nascendi* will explain the dynamics of other life-course phenomena as well. A promising candidate for study is the paradox of aging: How can people suffer significant loss with age and yet experience life more positively? Carstensen, Mikels and Mather (2006) explain this so-called 'positivity' effect by means of the socioemotional selectivity theory, which "… considers the effects of a continually changing temporal horizon on human development. The theory holds that when time is perceived as open ended, as it typically is in youth, people are strongly motivated to pursue information (…) In contrast, when time is perceived as constrained, as it typically is in later life, people are motivated to pursue emotional satisfaction." (p. 347). In other words, Carstensen and colleagues' theory predicts an age-related shift from primarily information processing and storage at an early age to a focus on the regulation of emotion states and emotional aspects later in life. It would be interesting to learn whether the Janus prototheory could explain the paradox of aging on the basis of the life-course dynamics of cognition and emotion.

Conclusion

If the metaphor drives the theory, then the model functions as a formalized kind of metaphor that connects theory with empirical research. Starting with the butterfly diagram as metaphor of human ontogenesis, the Janus model has been developed for the computer simulation of dynamic systems with *n* transitions. As demonstrated in respect of various empirical data sets, the Janus model of two coupled systems not only offers a satisfactory explanation of the dynamics of complex growth and decline curves with two transitions, but also provides new theoretical insights into the connection of systems from different scientific disciplines.

In conclusion, the interdisciplinary Janus model has proved its validity regarding the integration of data and theory in life-course dynamics. Adolphe Quetelet would have been satisfied.

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Summary

In this article, the classic problem of the human life-course, “how can the transition(s) of development into aging be explained?” has been solved. A theoretical model was constructed for the computer simulation of dynamic systems with one or more transitions in biology, psychology, and demography.

In constructing the Janus model, it was found that the hypothetical forces of growth and senescence determine the life trajectories of dynamic systems of development and aging in terms of peak time and peak capacity. In validating the model in respect of empirical data, it was found that the Janus model of two coupled systems offers a quite satisfactory explanation for the life-course dynamics of simple and more complex growth and decline functions like general physiological performance, fluid and crystallized intelligence, and US mortality in 1910 and 1970.

In conclusion, the interdisciplinary Janus model has therefore proved its validity in respect of the simulation of widely divergent life trajectories in biology, psychology, and demography. As such, the Janus model is fit par excellence for the integration of data and theory in developmental and aging research.

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