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Pace and Shape of Causes of Death

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Abstract

Within the last two centuries, humans have experienced remarkable mortality changes, paralleled by a shift in leading causes of death from infectious and parasitic diseases towards neoplasms and cardiovascular diseases. As recently shown, this development resulted in exceptional high levels of senescence. The question arises: how do specific causes of death shape the human aging pattern? To evaluate the relation between causes of death and the pattern of aging, we present a new method applying the recently developed framework of the "pace and shape of aging". This approach disentangles the pace of life from the qualitative, pace-standardized pattern (or shape) of aging. Based on French data from 1950 to 1999, we define three criteria to quantify 1) whether specific causes of death are more or less senescence related, 2) whether they have an accelerating or decelerating effect on aging, and 3) whether the causes are more or less alterable. We utilize the "pace-shape space" as a novel tool to summarize complex demographic information without need for parametric modeling, visualizing results along only two axes.

1 Introduction

The main drivers of longevity extension have changed over time. Declining infant and early adult mortality drove progress until the late 1940s, whereas older and old age mortality have been fueling progress since the 1950s. Old and very old ages have begun to make major contributions (Rau et al., 2008). This shift in age-contributions to longevity progess has been paralleled by unprecedented changes in the human pattern of senescence, where senescence is defined as an increase in mortality over age.

Figure 1 illustrates this development for French females. The right panel reveals the increasing

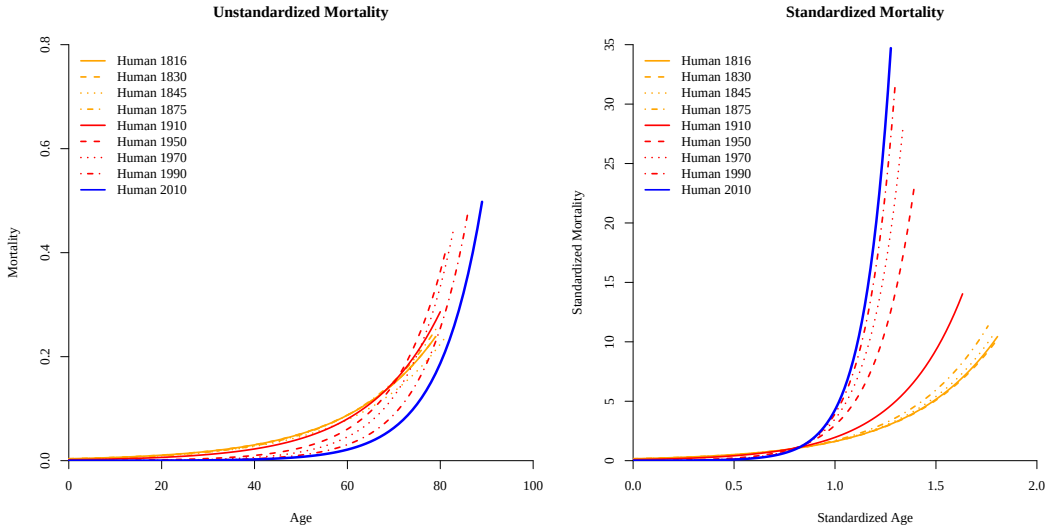


Figure 1: **Gompertzian Mortality - Unstandardized vs. Pace Standardized Perspective, French Females 1816-2010: The curves end when only 1% of the population is still alive.**

steepness of mortality change over adult ages, starting with a 14 fold rise over the life course in 1914 and developing towards a 35 fold rise in 2010.

The right panel of Figure 1 views the life course from a time-standardized perspective, as suggested by Baudisch (2011). Standardized age is calculated as

$$x_s = \frac{x}{e_0},$$

where e_0 denotes life-expectancy, and standardized mortality derives from

$$\mu_s(x_s) = e_0 \cdot \mu(x) = \frac{\mu(x)}{\overline{\mu}},$$

where $\overline{\mu}$ denotes average mortality. Hence, the right panel of Figure 1 depicts age in units of life-expectancy, and mortality as a multiple of its average level. By contrast, depicting the same data in an unstandardized way, the left panel of Figure 1 may leave the impression that French females in 2010 suffer less senescence than those a century earlier, since mortality is much lower.

In fact, the development of senescence is difficult to judge from the left panel, because it entangles two aspects of aging that need to be distinguished. On the one hand, populations experienced a prolongation of life, which mainly reflects a reduction in the overall level of mortality, or, as Baudisch (2011) argues, a change in the *pace* of life. On the other hand, populations experienced an exceeding concentration of deaths and a shift of the death hump towards higher ages (Bongaarts, 2005; Canudas-Romo, 2008; Kannisto, 2000), which implies an increasing steepness of mortality change over the life-course (see right panel of Figure 1), or, as Baudisch (2011) argues, an increasing steepness of the *shape* of aging. The shape of aging captures how mortality changes over the life-course, standardized by its average length, i.e. pace. It is hence independent of units of time and describes whether mortality changes markedly or mildly, thereby measuring the strength of senescence. Pace, by contrast, measures longevity and captures how fast the death clock ticks away. Note that strong, mild or nil senescence could come with a fast or slow pace of life.

Recent research has shown that conclusions about the strength of aging can change significantly and even reverse when pace and shape are disentangled(Baudisch, 2011; Jones et al., 2014). Separating pace from shape allows acknowledging that the meaning of time and age are not fixed but plastic over generations, and hinge on current conditions. For example, whereas in historical populations life-expectancy was about 40, and a year thus comprised 2.5% of the life course, nowadays with life-expectancies around 80, a year comprises only half as much, i.e. 1.25%. Likewise, the slowing of our life-course has altered the meaning of age. Burger et al. (2012) demonstrate that the colloquial expression "40 is the new 30" is more than just a mere saying. It reflects solid empicially observed truth. In fact, Japanese people around age 70 today could be considered just as young as hunter gatherers were in their late teenage years, at least judging from ages of similar mortality experience ("equivalent age"). What means "old" and what means "young" is relative and only makes sense given a certain context.

Previous approaches to judge whether deaths are premature or "normal" is an old question in demography. Already Lexis (1877) made an attempt to distinguish between juvenile, premature and normal deaths by separating the human death distribution in three parts. Also based on the distribution of death, Zhang and Vaupel (2009) suggest a more technical approach. They separate early from late deaths by means of a threshold age. They observe that, before this age, mortality improvements result in compression of ages at death, and thereafter in an expansion. Though both approaches reveal that early deaths today correspond to ages which have been considered late 10 years ago, they do not separte the pace from the shape of aging, as age-distributions are given over an absolute time scale. A similar reservation holds for the approach by Horiuchi and Wilmoth (1997) and Horiuchi (2006), who distinguish causes of death based on cause-specific aging rates, which are given along an absolute age axis. Notably, this is also true for judging full life histories as senescent or not on the basis of aging rates, as commonly done in gerontology (Finch, 1990). Alternatively, Bongaarts (2009) suggests to classify deaths in senescent and non-senescent causes depending on their avoidability and their biological origin, where unavoidable, internal

changes are classified as senescent. This approach resembles a classification typically used by biologists (e.g. Abrams (2004)), who distinguish between extrinsic and intrinsic mortality, the former being prevalent due to the environment, the latter capturing senescent causes of death. It is questionable, however, whether this distinction is meaningful and even well defined. Death inevitably comes about by an interplay of internal state and external environment. Death can be more or less successfully avoided depending on internal state, and a certain internal state may only lead to death given a certain environment, or the lack thereof (Wensink et al., 2014).

Our approach is purely demographic, i.e. based on the pattern of mortality. We utilize the pace-shape framework to classify causes of death and their influence on the changing human mortality profile, thereby accounting for the relative meaning of age and time. Our approach provides a novel tool to concisely summarize complex demographic information without need for parametric modeling, visualizing results along only two axes. We classify causes by pace and by shape separately based on explicit criteria and provide a new view on the relativity of senescence.

2 Material and Methods

2.1 Isolating Causes of Death and Using the Mixture Distribution as Benchmark

Ordinary life table methods neglect the fact that populations are heterogeneous. The effects of heterogeneity as well as their contribution to population analyses have been extensively studied (eg. Vaupel et al., 1979; Vaupel and Yashin, 1985; Wienke, 2011). Modeling strategies usually depend on whether sources of heterogeneity are observed or not. In our context, causes of death could be interpreted as indicators of observed heterogeneity within the dead, considered retrospectively.

Suppose a population can die from k different causes of death at any time. Each of the causes constitutes an age-specific proportion, $\pi_i(x)$, of all deaths occurring at age x, given by

$$\pi_i(x) = \frac{D_i(x)}{D(x)} \tag{1}$$

Assuming that all-cause death rate at age x is given by

$$m(x) = \sum_{i=1}^{k} m_i(x)$$

$\pi_i(x)$ can serve to separate total death by their respective causes into k subgroups. We then define a life table for every cause of death via

$$d_i(x) = d(x)\pi_i(x) \tag{2}$$

with $d(x)$ being the life table deaths at age x and $d_i(x)$ denoting the life table deaths of cause i at age x. In each sup-population, individuals can only die from a single characteristic cause of death. Though this is generally similar to a multiple-decrement life table approach (see Preston et al. (2001)), our approach differs because we treat each sub-population separately, normalizing death such that each life table of a sub-group starts at one. Hence, we normalize cause-specific deaths via

$$d_i^n(x) = \frac{d_i(x)}{\sum_{x=0}^{\omega} d_i(x)} \tag{3}$$

where $d_i^n(x)$ denotes the normalized cause-specific life table deaths. With this step, causes of death are isolated. The associated normalized cause-specific life-table is given by

$$l_i^n(x) = \sum_{x}^{\omega} d_i^n(x). \tag{4}$$

Life expectancy for individuals dying from cause i can then be calculated as

$$e_i(x) = \frac{\sum_{x}^{\omega} L_i^n(x)}{l_i^n(x)}. \tag{5}$$

$L_i^n(x)$ indicates the lived person years of the normalized life table population in the x age-interval. With this approach, the difference between total and cause-specific life expectancy hinges merely

on the age- and cause-specific proportion of all deaths, $\pi_i(x)$. This enables us to analyze the influence of isolated causes of death on the pace and shape values of the total population (for an illustration see appendix). Though separating causes of death as above is hypothetical and indeed unrealistic, it serves to study contributions of causes of death to total mortality in isolation, which might be considered similarly unrealistic as studying age-specific deriviatives of mortality assuming no effect at all other ages. Isolating causes of death, we do not account for competing causes of death, which has been intensively discussed elsewhere (Hougaard (1984) and Prentice et al. (1978)). Instead, we consider hypothetical sub-populations where individuals can only die from one specified cause of death, and determine the associated cause-specific pace and shape values as described below. The total population represents a mixture distribution of all subpopulations. Its pace and shape values represent the benchmark against which all causes of death are evaluated.

2.2 The Measurement of Pace

Pace captures the timing of death. We measure pace by the cause-specific average age at death, $e_{i,0}$. Other quantities, like the median age at death or maximum life span, similarly qualify, as they fullfil desired mathematical properties of pace measures (cf. Wrycza and Baudisch, 2014). We selected our pace measure, because $e_{i,0}$ fullfils the additional property that the pace value of a mixture distribution, here the total life span distribution, is the weighted average of the pace values of its component distributions, here cause-specific life span distributions (Wrycza and Baudisch, 2014). Furthermore, $e_{i,0}$ can be interpreted as an indicator of average mortality, which appeals to intuition. Low (high) average mortality results in a slow (fast) pace, which is associated with high (low) values of the pace measure. Since we investigate aging as a property of the adult life course, life expectancy is calculate from maturity onwards.

2.3 The Measurement of Shape

Shape captures the time-standardized change in mortality. It reveals whether mortality (on average) increases or decreases over age, and whether these changes are more or less pronounced. Thereby, the shape of aging shows whether and to what extent organisms get better at concurring death, sustain a certain state or get worse over their life course, independent of their length of life. Wrycza et al. (2014) derive desired mathematical properties of shape measures and evaluate a number of potential candidates. Following that study, well-known measures in demography that account for lifespan disparity justify as good indicators of shape, such as the Gini-coefficient, Keyfitz's entropy, or the coefficient of variation. Intuitively this can be understood recognizing that a high shape value is analog to low variability in the age at death. Hence, if everyone would die at the same time, variability is zero, the age-pattern of mortality shows maximum steepness, rising from zero to infinity at the unique age at death. In contrast, constant or falling mortality patterns would lead to high inequality in the age at death, many dying early in the life course while a few

would experience exceptionally long lifespans.

In this study, we use a shape measure S, given in equation 6, that is based on standard deviation σ around the mean age at death,

$$S = 1 - \frac{\sigma}{e_0} \quad \text{with} \quad \sigma = \sqrt{\int_0^\omega (x - e_0)^2 d(x)\, dx}. \tag{6}$$

Note that this measure does not depend on units of time. The ratio of standard deviation and life-expectancy can be interpreted as pace-standardizing lifespan disparity and can be recognized as the coefficient of variation. The additional scaling ("1-") is technically motivated to ensure that the boundary case of constant mortality is given by a shape value of zero, whereas a value of one would indicate a perfectly rectangular survival function. In general, a high (low) shape value indicates pronounced (weak) changes of mortality over age and strong (weak) age-dependency.

2.4 Data

The analysis is based on data provided by the "Institut national d'études démographiques (INED) (Vallin and Meslé, 2013). The database contains death counts on causes of death from 1925 to 1999. During this period, the ICD revision has changed several times. Thus, the provided data is a reconstructed time series based on the 9th Revision of the ICD. Table 1 gives an overview on the coding of specific causes.

36 different causes of death as well as all-cause death counts for females and males separatly are selected to calculate cause- and age-specific proportions. However, the freely available data is only provided in 5-year age groups, ending at the open age group 100+. The groups comprise approximately 97% of all deaths in the respective years. HIV is available from 1982 onward.

To increase the level of detail of our analysis, the cause-specific proportions are spline-smoothed to create single-year proportions. The choice of spline-smoothing is a result of a pretest on grouped population from the Human Mortality Database (2014).

The cause-specific proportions are applied to life tables calculated with death counts and exposures from the Human Mortality Database (2014). Due to irregularities between the all-cause death counts from INED-database and the Human Mortality Database (2014) in the years from 1925 to 1949, the analysis is conducted from 1950 onward.

Table 1: Overview on CoD Coding and Frequencies

	Causes of death	ICD 9 codes	1950 Absolute/Relative Frequency		1999 Absolute/Relative Frequency	
			Female	Male	Female	Male
	Infectious and parasitc diseases					
1	Septicemia	038	756 (0.29%)	621 (0.23%)	765 (0.29%)	676 (0.25%)
2	HIV	042	0 (0.00%)	0 (0.00%)	202 (0.08%)	741 (0.27%)
3	Other infectious and parasitic diseases	001-037; 039-041; 043-139	12099 (4.61%)	19596 (7.20%)	2875 (1.09%)	2574 (0.94%)
	Neoplasms					
4	Malignant neoplasm of colon and rectum	153-154	4564 (1.74%)	3788 (1.39%)	7810 (2.97%)	8748 (3.18%)
5	Malignant neoplasm of liver and intrahepatic bile ducts	155	2849 (1.08%)	2245 (0.82%)	1621 (0.62%)	5279 (1.92%)
6	Malignant neoplasm of trachea, bronchus and lung	162	929 (0.35%)	2252 (0.94%)	4329 (1.65%)	20866 (7.59%)
7	Malignant neoplasm of breast (women only)	174	4332 (1.65%)		11281 (4.29%)	-
	Malignant neoplasm of prostrate (men only)	185	-	2291 (0.84%)	-	9476 (3.45%)
8	Leukemia	204-208	651 (0.25%)	858 (0.32%)	2223 (0.85%)	2568 (0.93%)
9	Other neoplasm	140-152; 156-161; 163-173; 175-184; 186-203; 209-239	25716 (9.81%)	25513 (9.37%)	32178 (12.25%)	42204 (15.36%)
	Endocrine, nutritional and metabolic diseases and immunity disorders					
10	Diabetes mellitus	250	2369 (0.90%)	1279 (0.47%)	5466 (2.08%)	4330 (1.58%)
11	Other endocrine, nutritional and metabolic diseases and immunity disorders	240-249; 251-279	3059 (1.17%)	1972 (0.72%)	4799 (1.83%)	2662 (0.97%)
12	**Diseases of the blood and blood-forming organs**	280-289	625 (0.24%)	561 (0.21%)	1480 (0.56%)	1356 (0.49%)
	Mental disorders					
13	Dementias	290	370 (0.14%)	266 (0.10%)	5135 (1.95%)	1935 (0.70%)
14	Other mental disorders	291-319	962 (0.37%)	1686 (0.62%)	4519 (1.72%)	4305 (1.57%)
	Diseases of the nervous system and sense organs					
15	Parkinson's disease	332	703 (0.27%)	701 (0.26%)	1720 (0.65%)	1841(0.67%)
16	Alzheimer's disease	331.0	5 (0.00%)	6 (0.00%)	4824 (1.84%)	2081 (0.76%)
17	Other diseases of the nervous system and sense organs	320-330; 331.1-331.9; 333-389	6242 (2.38%)	5964 (2.19%)	3077 (1.17%)	3325 (1.21%)
	Diseases of the circulatory system					
18	Hypertensive diseases	401-405	1692 (0.65%)	1318 (0.48%)	5302 (2.02%)	2930 (1.07%)
19	Ischemic heart diseases	410-414	1684 (0.64%)	2011(0.74%)	9217 (3.51%)	10476 (3.81%)
20	Heart failure	428	29630 (11.30%)	22983 (8.44%)	14962 (5.70%)	9192 (3.35%)
21	Cerebrovascular diseases	430-438	32587 (12.43%)	27446 (10.08%)	22607 (8.61%)	16011 (5.83%)
22	Other diseases of the circulatory system	390-400; 406-409; 415-427; 429; 439-459	27020 (10.30%)	30550 (11.22%)	36756 (13.99%)	37466 (13.64%)
	Diseases of the respiratory system					
23	Pneumonia and influenza	480-488	13573 (5.18%)	12755 (4.69%)	9885 (3.76%)	8343 (3.04%)
24	Chronic obtrusive pulmonary diseases and allied conditions	490-496	2873 (1.10%)	4565 (1.68%)	5403 (2.06%)	8608 (3.13%)
25	Other diseases of the respiratory system	460-479; 489; 496-519	10131 (3.86%)	10180 (3.74%)	6122 (2.33%)	5465 (1.99%)
	Diseases of the digestive system					
26	Chronic liver diseases and cirrhosis	571	2505 (0.96%)	4306 (1.58%)	2849 (1.08%)	6405 (2.33%)
27	Other diseases of the digestive system	520-570; 572-579	7011 (2.67%)	7734 (2.84%)	9092 (3.46%)	7165 (2.61%)
	Diseases of the genitourinary system					
28	Nephritis, nephrotic diseases and nephrosis	580-589	9135 (3.48%)	11412 (4.19%)	2387 (0.91%)	2541 (0.92%)
29	Other diseases of the genitourinary system	590-629	390 (0.15%)	2198 (0.81%)	1635 (0.62%)	1213 (0.44%)
30	**Diseases of the skin and subcutaneous tissue**	680-709	458 (0.17%)	391 (0.14%)	1733 (0.66%)	758 (0.28%)
31	**Diseases of the musculoskeletal system and connective tissue**	710-739	855 (0.33%)	554 (0.20%)	1959 (0.75%)	1013 (0.37%)
32	**Ill-defined causes**	780-799	39791 (15.17%)	33954 (12.47%)	19176 (7.30%)	14710 (5.35%)
	External causes of injury and poisoning					
33	Transport accidents	E800-E848	427 (0.16%)	1965 (0.72%)	2192 (0.83%)	5930 (2.16%)
34	Accidental falls	E880-E888	1485 (0.57%)	1527 (0.56%)	6646 (2.53%)	3874 (1.41%)
35	Suicide and self-inflicted injury	E950-E959	1529 (0.58%)	4714 (1.73%)	2841 (1.08%)	7427 (2.70%)
36	Other external causes	E850-E879; E890-E949; E960-E999	5496 (2.10%)	12128 (4.46%)	6185 (2.35%)	8688 (3.16%)

3 Criteria to Classify Causes o Death

Each sub-population can be classified assigning two scalar values for pace and shape respectively. This enables depicting populations in a scatter plot. The plot concisely summarizes cause-specific characteristics with respect to the average level of mortality (i.e. pace of death) and the relation of mortality to age (i.e. shape).

To illustrate our appoach, Figure 2 exemplifies such a "Pace-Shape Space" for an artificial population with artificial causes of death that resemble typical components of the human life span distribution. The graph depicts the mixture distribution of causes, representing the total population marked in orange, along with its associated sub-population, each representing a certain cause of death. All distributions are located at the coordinates given by their respective pace and shape values.

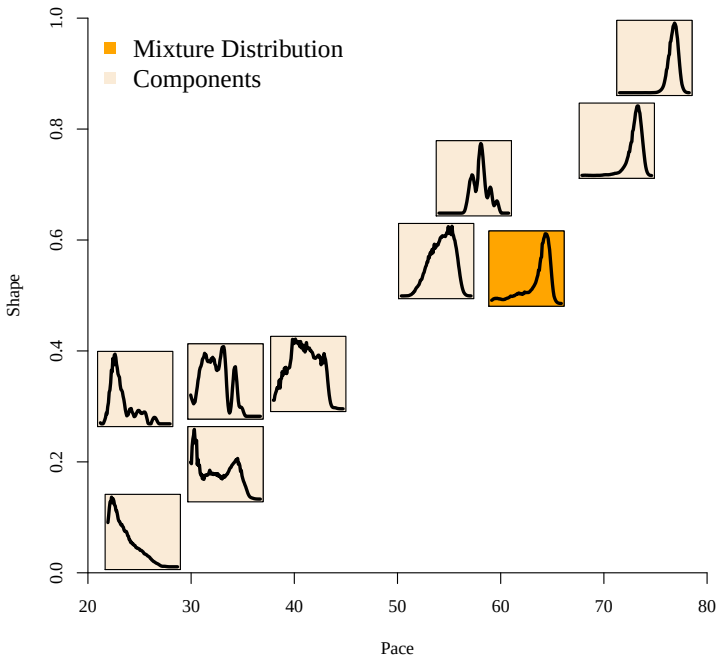


Figure 2: **Pace-Shape Space with Artifical Life Span Distributions: All distributions are plotted along the same time axis; each distribution is normalized, such that the area under the curve equals one.**

The distributions of death in Figure 2 reveal three clusters, which fundamentally differ in their combination of pace and shape values. The first cluster, located in the lower left corner, is generally characterized by a high density of deaths at or close after the age of maturity, corresponding to low pace and low shape values. The second cluster concentrates around the mixture distribution, thus sharing similar characteristics. All components are uni-modally distributed with the mode located in the second half of the life span. They exhibit narrower and later death humps corresponding to

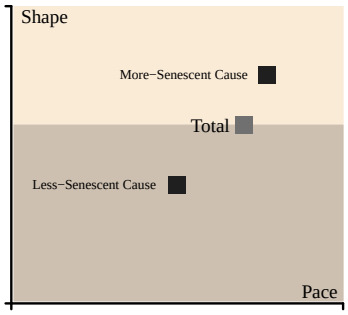
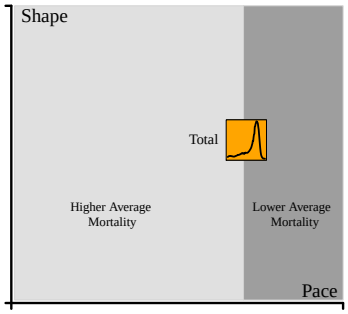
higher shape values. The third cluster in the upper right corner contains components displaying mortality concentrated at the end of life, associated with sharply rising mortality at late ages. Pace values are high due to long stretches of life with low mortality.

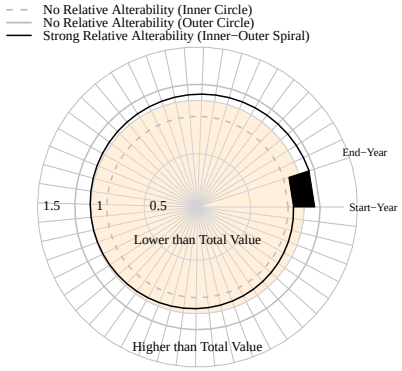
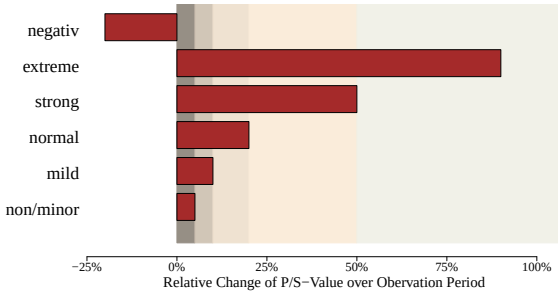
The location of sub-populations relative to the total enables us to define criteria that classify causes of death 1) with respect to shape and 2) with respect to pace. By the definition of shape, higher (lower) shape values mark stronger (weaker) changes of mortality over age. Our first criterion therefore defines a cause of death as more senescence-related, if the cause-specific shape value is larger than the all-cause shape value. We define a cause of death as less related to senescence, if the cause-specific shape value is smaller than the all-cause shape value. By the definition of pace, higher (lower) pace values mark lower (higher) levels of average mortality and thus a slower (faster) life course. If components have a faster than average pace, we define them to exert an accelerating effect on total pace. If components have a slower than average pace, we define them to exert a decelerating effect on total pace. Notably, these definitions do not specify the magninute of the effect, i.e. whether the contribution of a specific cause of death is large or small.

We emphasize that our criteria hinge on a given benchmark population; they are only valid relative to a reference group given by the mixture distribution of all-cause mortality. Thereby, our classification of causes of death accounts for the relativity of senescence.

Together, these criteria help define a further criterion that can be used to interpret time-series of cause of death data. Plotting pace and shape values in PS-space for successive time points, trajectories emerge that reveal the *alterability* of causes of death. On the one hand, we can identify whether causes of death are generally alterable, i.e. whether progress can be made at all for a certain cause of death (*absolute alterability*). On the other hand, we can identify causes that are more or less alterable relative to total mortality improvements (*relative alterability*). We define causes as ”normally” alterable, if cause-specific changes are similar to the total, i.e. if changes fall no more than about 5% above or below the benchmark. Causes are ”non-alterable”, if absolute changes do not exceed about 5%. Causes are ”mildly” alterable, if changes fall between the range of non- and normally alterable. Causes are ”strongly” alterable, if changes exceed the range of normally alterable. We will distinguish between pace-alterability and shape-alterability. Table 2 summarizes the criteria defined above.

Table 2: Criteria Description

Category	Description	Example
Criterion I – More-Senescent vs. Less-Senescent – Shape Level		
More-Senescent	If cause-isolated shape values lie *above* the total, then these changes are *more*-senescence related, *more* related to changes over age	
Less-Senescent	If cause-isolated shape values lie *below* the total, then these changes are *less*-senescence related, *less* related to changes over age	
Criterion II – Pace Level – Average Mortality		
Accelerating Cause	If the isolate cause has a *faster pace*, the cause has a *higher* average mortality then the total and exerts an *accelerating* effect on total pace	
Decelerating Cause	If the isolate cause has a *lower* pace, the cause has a *lower* average mortality then the total and exerts a *decelerating* effect on total pace	

Criterion III – Alterability		
Description		
We investigate whether causes of death are generally alterable (*absolute alterability*) i.e. whether progress can be made for a certain cause of death. We further investigate whether causes are more or less alterable relative to total mortality improvements (*relative alterability*).		
Category	Relative Alterability: $\frac{V_t^i}{V_t}$	Absolute Alterability: $\frac{V_{t+n}^i - V_t^i}{V_t^i}$
Pace-Alterable		
Shape-Alterable		
V = Pace or Shape Value t = Start Year $t+n$ = End Year i = Specific Cause of Death		

4 Preliminary Results

4.1 Criteria I and II: Dynamics of Cause-Specific Shape and Pace Values over Time

Figure 3 shows a ranking of cause-specific pace and shape values for females (see appendix for males), exemplarily highlighting three groups of causes of death: cardiovascular diseases, infectious diseases, and external causes. Figure 4 illustrates a male-female comparision of shape values for malignant neoplasms. Many more results could be zoomed into; we give a full overview of PS-spaces for both males and females for all causes of death in the appendix. The results below are selected to reveal general findings and serve to demonstrate the utility of the method in analyzing causes of death.

Figure 3: **Shape and Pace Ranking, Females, France, 1959-1999 (ten-year intervals): Causes of death are ranked relative to the total population, which is highlighted in black. Red marks cardiovascular diseases, blue external causes, and yellow infectious and parasitic diseases.**

Regarding shape, Figure 3 shows that cardiovascular diseases unambiguously classify as more senescence-related causes for females, ranking in the upper third of cause-specific shape values and staying consistently above the total. Though not among the groups highlighted in Figure 3, note that dementia is the single cause of death that is most senescence-related at all times. By contrast, external causes (accidents, suicide) consistently rank as least senescence-related, with one exception. Accidental falls clearly show a relation to senescence, though with a downward trend

in rank position over time.[1] Infectious diseases reveal marked change in their shape ranking across time. Especially septicemia has undergone a major shift from low-senescence to high-senescence.

Regarding pace, Figure 3 shows that cardiovascular diseases generally exert a decelerating effect on total pace, with little rank changes over time. By contrast, infectious diseases, as for shape, show remarkable changes with respect to pace. From an initially accelerating effect in 1959, septicemia developed into a strongly decelerating component of death in 1999. Other infectious and parasitic diseases also start with a fast pace in the late sixties, eventually converging with the total at the end of the century. Notably, HIV (included in the data since 1983) has the fastest pace of all causes. Further fast causes of death are external causes, except for accidential falls, which instead rank among the slowest. This highlights that dying from physiological frailty only happens very late in the life course, whereas accidents mainly hit younger people.

Figure 4: **Shape Ranking, Males and Females, France, 1959 - 1999 (ten-year intervals): Causes of death are ranked relative to the total population, which is highlighted in black. Blue marks malignant neoplasms.**

Shape values can differ significantly among the sexes. Figure 4 shows that, for males, neoplasms (except leukemia) are more senescence-related (have shape values above the total), prostate cancer even ranking second highest among all causes of death. Only leukemia ranks below the total, though with a strong upward tendency over time. By contrast, all female neoplasms rank as

[1]This illustrates the importance of accounting for the interaction of (internal) state and origin of cause of death (external) when classifying causes of death on the physiological level.

less senescence-related in recent years, which again highlights the relativity of senescence. Over time, all cancers experienced a trend towards less senescence for both sexes, despite Leukemia, which shows the opposite trend.

In general we find that total shape for female ranks higher than for males, i.e. females experience on average more senescence than males. For both sexes, the majority of causes of death rank above the total, hence causes below the total need to exert a certain level of influence to balance the majority of causes above. Over time, causes of death tend to become more senescence-related and tend to have a decelerating effect on pace. People today thus die more and more due to senescence rather than anything else, and the human life-course is slowing down, at least with respect to death, which is consistent with previous findings. As a general pattern we see that higher pace values come along with higher shape values.

4.2 Criterion III: Alterability of Causes of Death

Changes in overall mortality have been brought about by changes in causes of death, which have been diverse. To shed light on the underlying details of overall mortality change, Figure 5 reveals whether and how much the pace and shape of specific causes of death have changed over time, whereas Figure 6 reveals whether these changes are alterable relative to the total mortality trend.

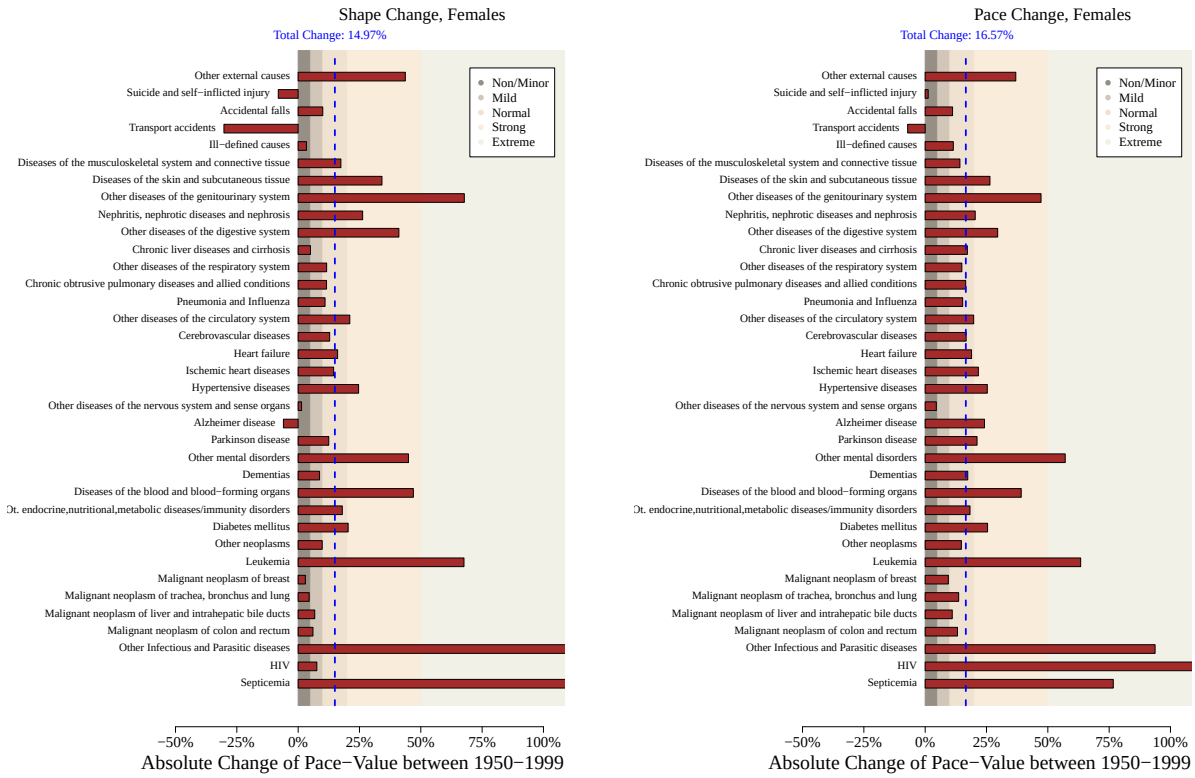


Figure 5: **Absolute Cause Alterability: Changes in cause-specific shape and pace values between 1950 and 1999, relative to their starting values in 1950 for French Females**

Figure 5 shows that infectious diseases are remarkably alterable for females (see appendix for males) in both pace and shape. This finding was already indicated in the figures above by the markedly shifting rank positions. But different to the pure ranking shown there, Figure 5 reveals the actual magnitude of changes, which exceeds more than 100% for septicemia and other infectious or parasitic diseases. At the opposite extreme, some causes barely made any progress, despite overall mortality improvements, which is particularly true for neoplasms. Only leukemia increased in shape value, which implies that young people have been successfully saved from death for this cancer, while progress at higher ages is lacking behind. Some causes show negative alterability, meaning that shape values have declined over time. This is true for suicide and transport accidents. Note that most cases (for both pace and shape) that markedly exceed average progress (given by total shape and pace improvements as indicated by the dashed vertical line) correpond to causes that are classified as "other" in the various groups of diseases. This suggest that the conspiciously large values for these groups might possibly be an artefact of misclassification.

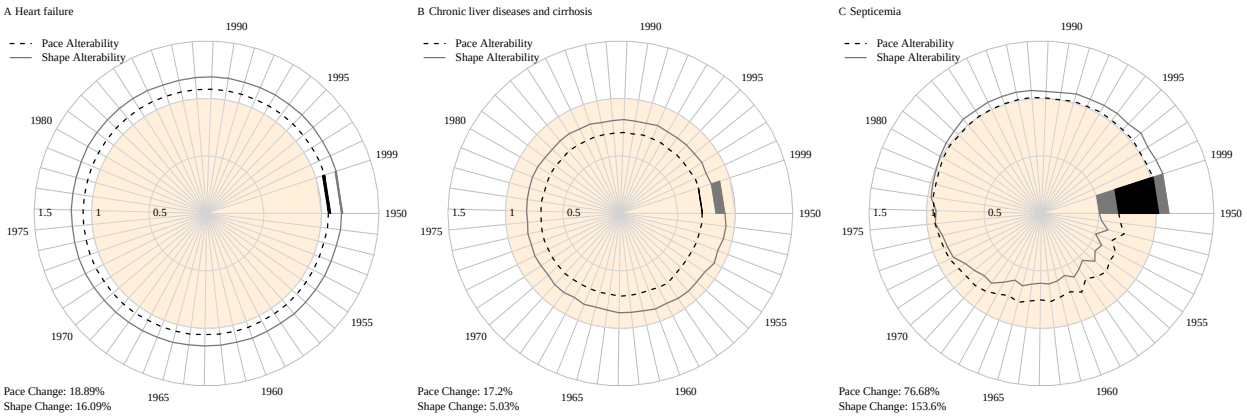


Figure 6: **Relative Cause Alterability: Ratio of the cause-specific pace and shape values to the total at each time point between 1950 and 1999 for French Females. The outer edge of the colored area marks the reference line given by the total, where the ratio equals one. The time trend can be read clockwise starting at three o'clock. Absolute alterability for each cause (as shown in Figure 5) is denoted below each circle. All-cause alterability between 1950 and 1999 was 16.57% for pace and 14.97% for shape.**

Figure 6 shows changes in female pace and shape values for three selected example causes of death relative to the total. It allows to zoom into the development of the specific cause of death over time in relation to the general trend. As general patterns we either find circles or spirals.

Graphs A and B exemplify outer and inner circles. Outer (inner) circles represent causes that over time keep a constant distance above (below) the total. These causes are bound to closely follow the general trend of mortality improvement. They are alterable in the absolute sense, but non-alterable relative to the total. We interpret circular patterns outside the total as characteristic

for causes of death that can be postponed, and circles inside the total as characteristic for ”lagging behind”, in example B most likely due to unhealthy behavioral habits.

Graphs B and C exemplify inward and outward spirals. The outward spiral in C depicts the develpment of pace and shape values for septicemia, which is particularly pronounced. Such outward spirals characterize causes whose development over time have exceeded overall progress. The graph illustrates the detailed changes over time. Progress in septicemia has been unimpeeded over decades, shifting the cause from less to more senescence-related in the mid-seventies and continuing to be on the rise. This development tells the success story of saving lifes from septicemia, which nowadays only pose a probelm to exceptionally frail individuals at relatively old ages. This cause of death is strongly alterable, both in absolute and in relative terms

The inward spiral of graph B for shape exemplifies a completely different process. For such cases, we observe a negative relative alterabiltiy. However, this is an indirect development, as the cause in itself is non-alterable in absolute terms. Due to the overall improvements in total mortality, however, such causes diverge (passively) from the average trend in the population.

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A Pace-Shape Spaces

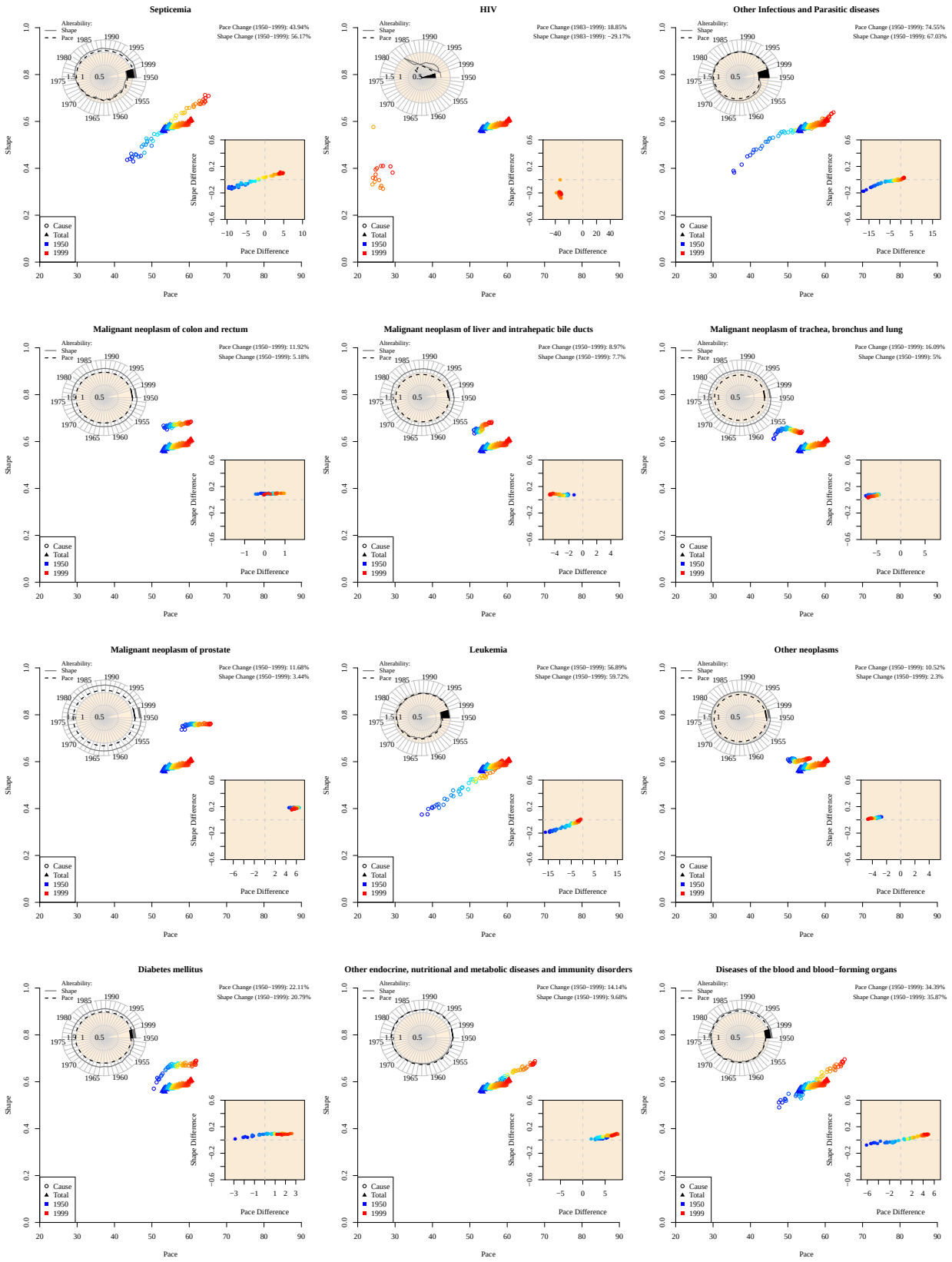

Figure 7: Pace-Shape Spaces Males, France, 1950-1999

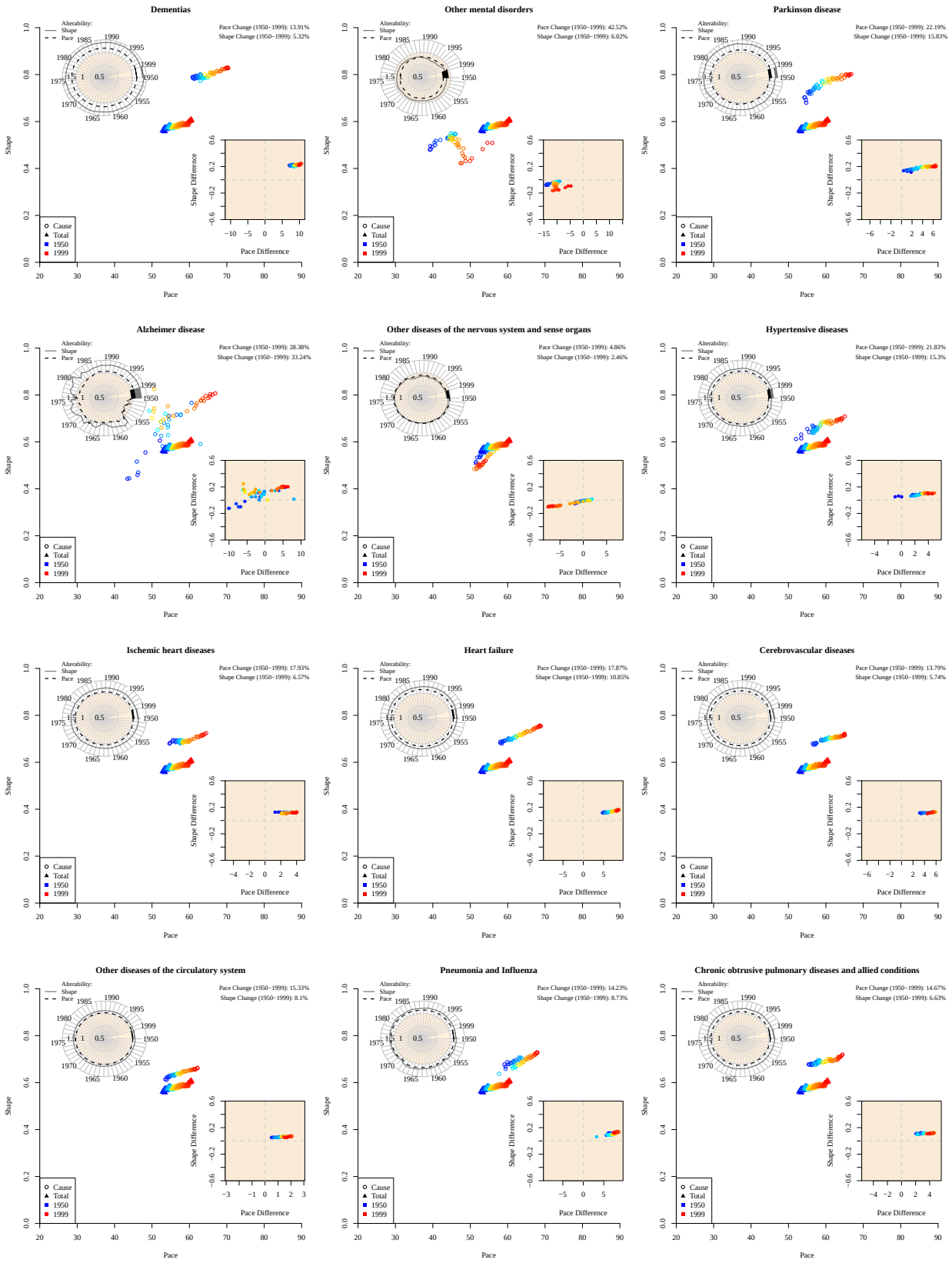

Figure 8: Pace-Shape Spaces Males, France, 1950-1999, continued

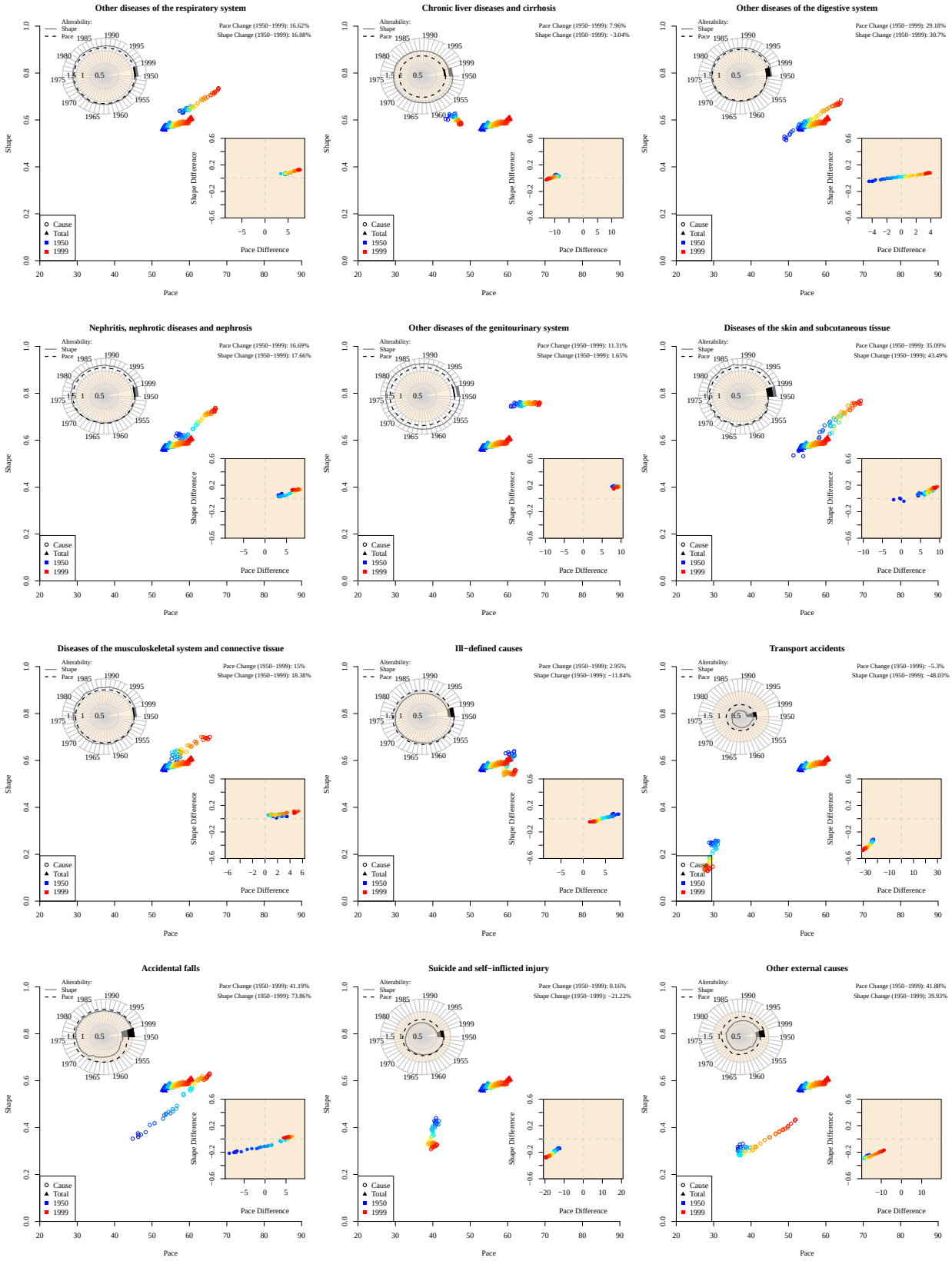

Figure 9: Pace-Shape Spaces Males, France, 1950-1999, continued

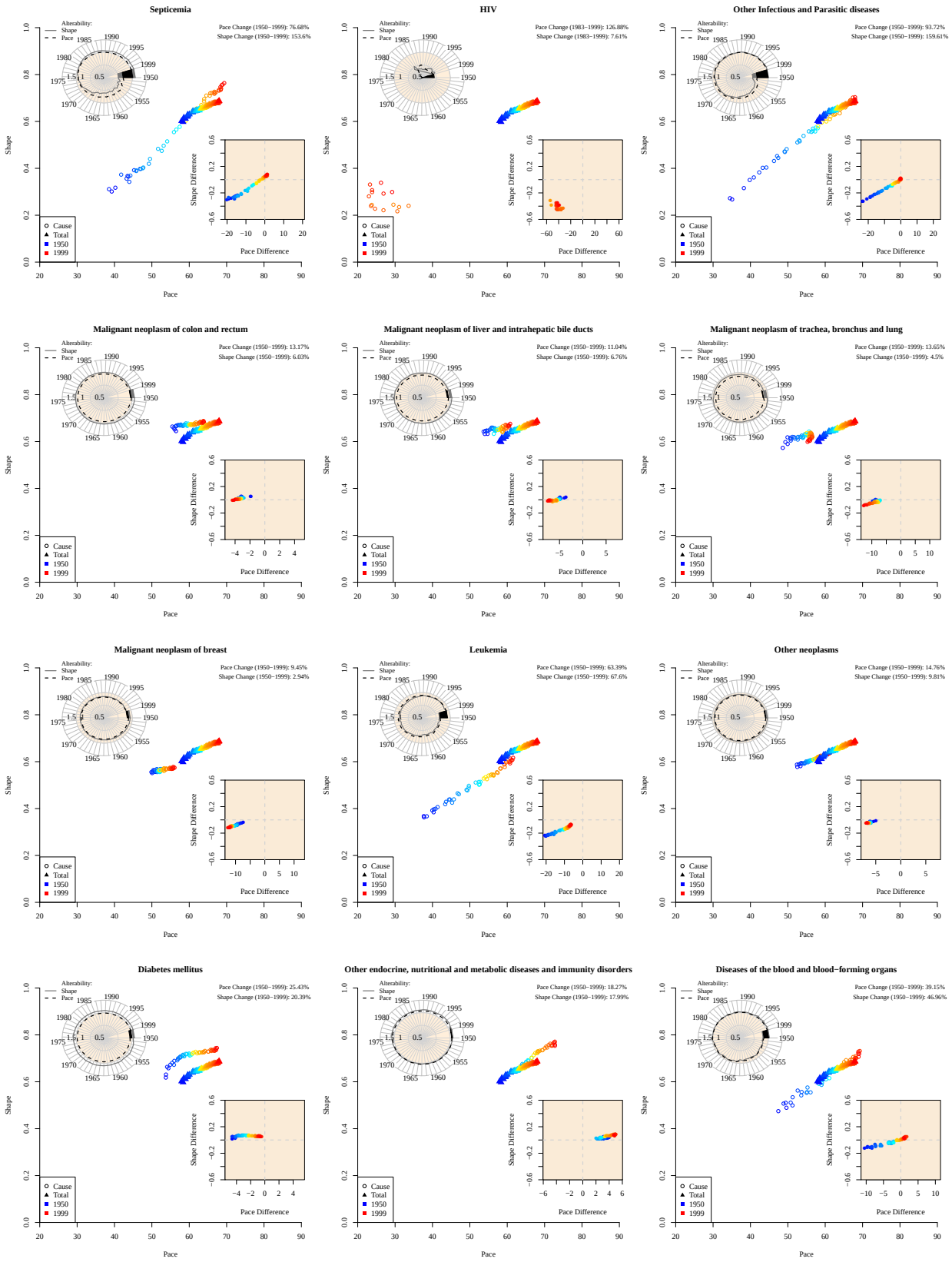

Figure 10: Pace-Shape Spaces Females, France, 1950-1999

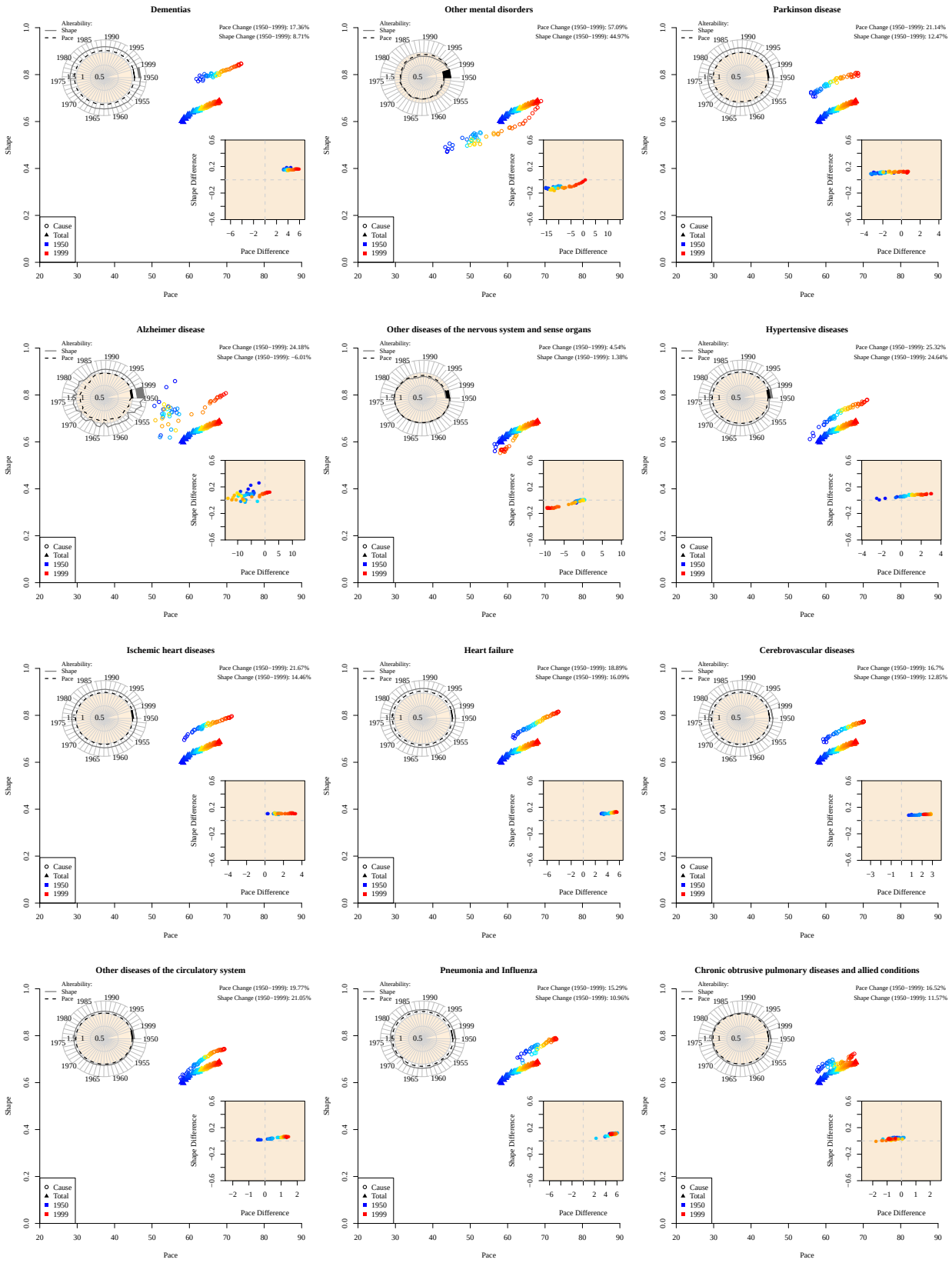

Figure 11: Pace-Shape Spaces Females, France, 1950-1999, continued

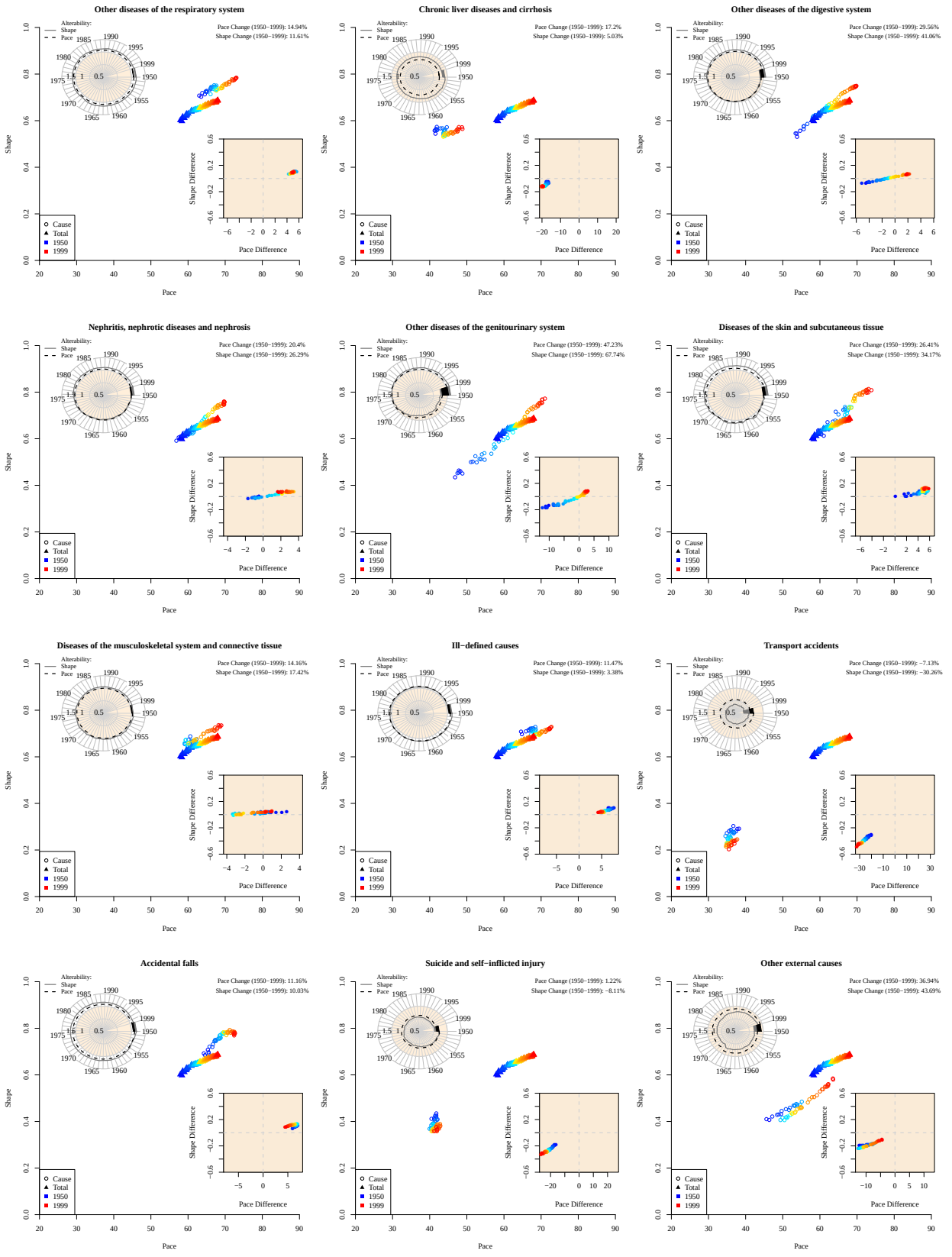

Figure 12: Pace-Shape Spaces Females, France, 1950-1999, continued

B Additional Ranking Graphs

Figure 13: Pace Ranking, French Males and Females, Neoplasms highlighted

Figure 14: Shape and Pace Ranking, French Males, heart, infectious and external highlighted

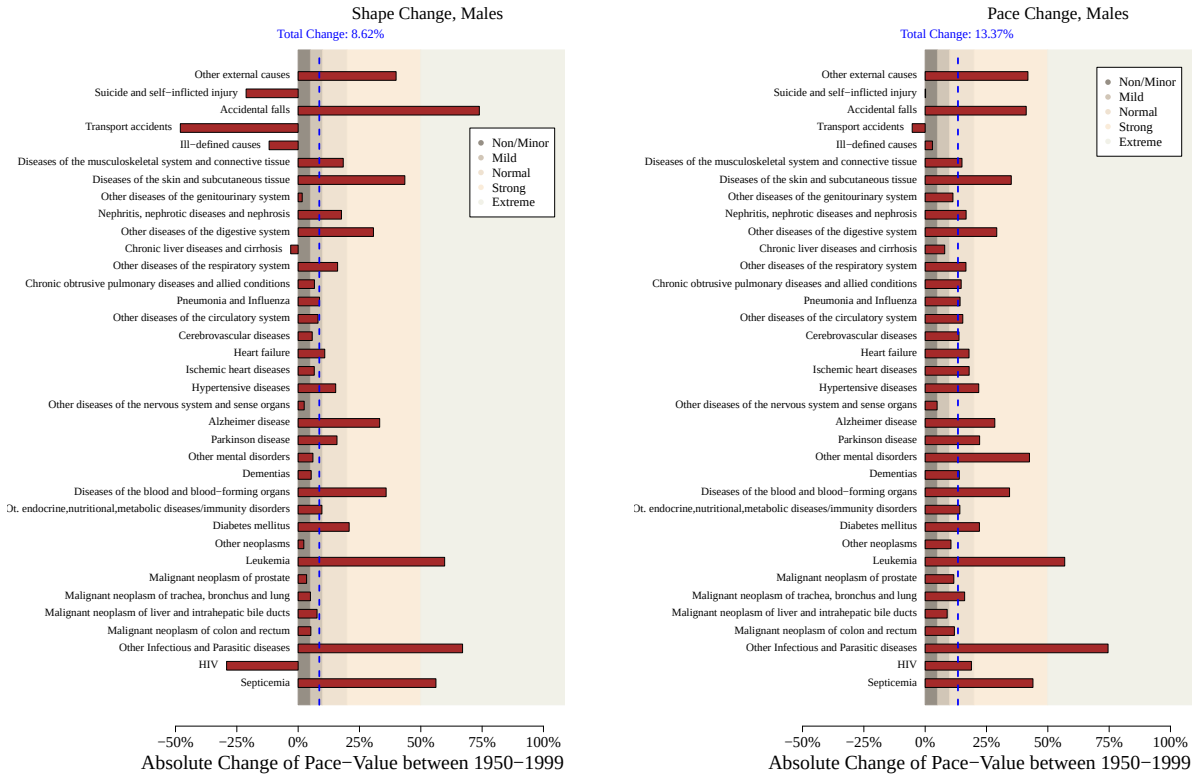

Figure 15: Relative Shape and Pace Change between 1950 and 1999, French Males