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# Age-Related Dynamics and Gender Differences Reveal that Senescent Phase of Human Ontogeny can be Subdivided According to Morbidity and Mortality Caused by Various Disorders


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## Abstract

The epidemiologic data are presented for morbidity and mortality from cardiometabolic disorders and Alzheimer disease during chronologic period 2008-2023 in three Brazilian states of Southern region. The evidence obtained suggests that senescent phase of human ontogeny can be subdivided due to peculiarities of age-related dynamics and gender differences in morbidity and mortality.

**Keywords: Morbidity; Mortality; Ontogeny; Metabolic Syndrome; Dementia**

## Introduction

This story began in the first decade of current century at the University of Ijui (Unijui) in the South of Brazil. At that time the author of present article worked as a Professor of Pharmacology, trying to use scarce opportunities for studying the influence of glucocorticoids on somatic and organ growth on experimental models in rats. When these opportunities were in general exhausted, our attention was fortunately attracted to epidemiologic data freely available in Brazilian national database called DataSus on the website of Brazilian Ministry of Health.

Since our first trial based on DataSus in 2005 on cardiometabolic disorders was quite successful [1], we decided to continue these investigations in larger scale. A great part of them was presented at the World Congress of International Society for DOHaD in Santiago, Chile in 2009. It was possible already at that time to affirm that the evidence in favour of unique general scheme of aging does not exist [2]. In fact, if to accept the definition of aging as biological process that provokes an increase in morbidity and mortality with age, then substantial heterogeneity of epidemiologic parameters should be taken into account.

Later on, we tried to use this heterogeneity to justify the periodization of postnatal ontogeny in humans to at least 3 phases: early, middle and late one, dividing the whole age scale roughly to 3 parts: 0-29, 30-59 and 69-79 and more than 80 y [3]. The early phase included childhood (0-9 y), adolescence (10-19 y) and young adulthood (20-29 y), and it was possible to perform further periodization of the first two decades, according to linearization of growth curves in mono- and bilogarithmic coordinates [4], whereas middle and late phases of ontogeny could be distinguished by specific association of morbidity and mortality from various disorders. As a matter of fact, cardiometabolic disorders were characteristic to late phase of senescence, whereas some neuropsychiatric and gastro-intestinal diseases were more prominent in the middle phase [5, 6]. Moreover, there were clear gender differences in at least some of disorders evaluated.

All these data were obtained mainly for chronologic period 1998-2007, therefore just recently, since 2025 we decided to amplify our investigations to more recent chronologic period of 2008-2023. Here we show and discuss our data for cardiometabolic disorders and Alzheimer disease.

## Methodology

The whole procedure used since 2005 can be divided to 2 steps: a) data extraction and recalculation; b) statistics and visualization. During the first step the raw data were extracted from DataSus and recalculated in per cent of total (in the whole life-scale) as relative or proportional morbidity and mortality for both genders together, for each decade of age in every chronologic year, separately for each of 3 states (provinces) in Southern region of Brazil: Rio Grande do Sul

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(RS), Santa Catarina (SC) and Parana (PR). It should be clarified that morbidity was assessed in accord to the number of hospitalizations.

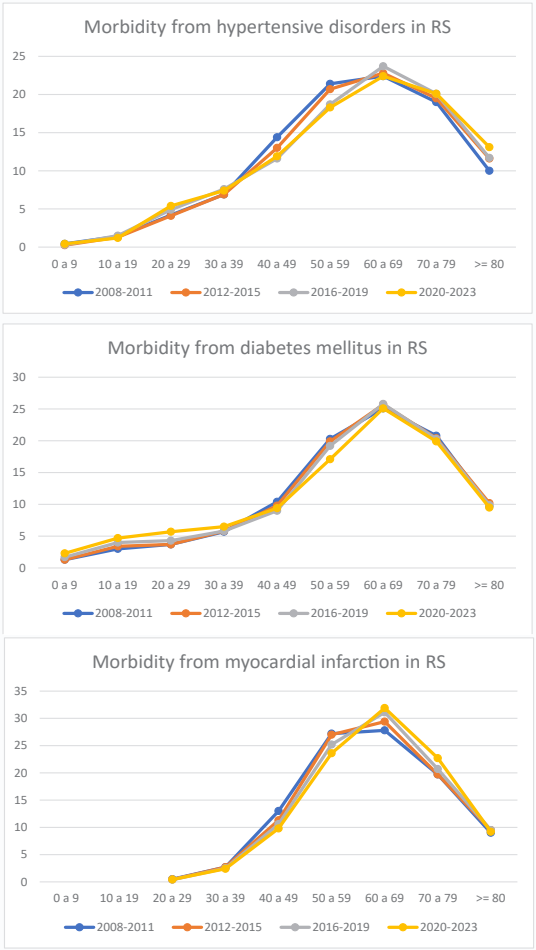
Moreover, feminine fraction in percent of total (for both genders together) was also calculated. Finally, descriptive statistics were performed in each of 4 equal parts of the whole chronologic period evaluated, generating the data in tables that were transformed to plots by means of Excel software for better visualization.

Here we shall demonstrate the evidence only for the state of RS, since the data for SC and PR were quite similar. Moreover, for the sake of clarity and better analysis, only arithmetic means will be shown on the plots, because usually the values of standard errors were less than 1/10 of the mean.

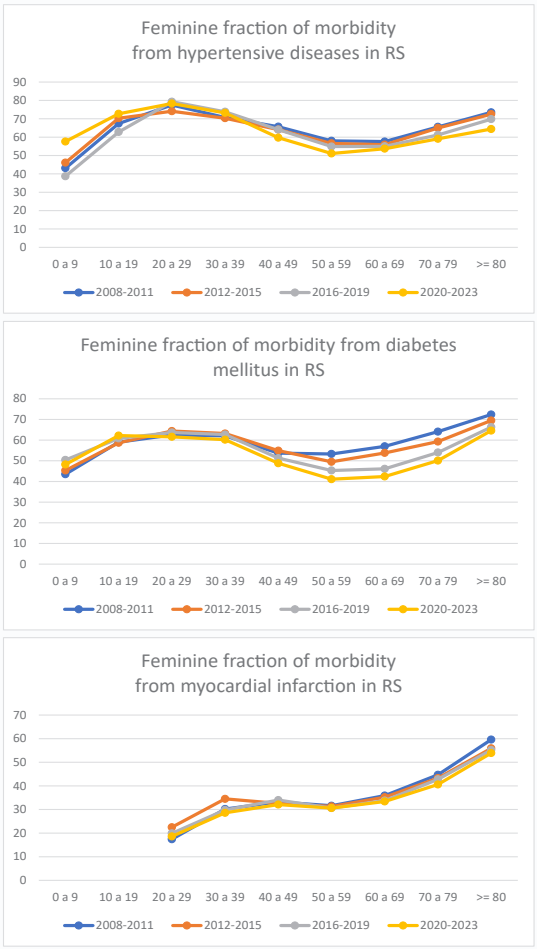
## Results

First of all, our present data (Figures 1-6 and 9-14) largely confirm the conclusions already made previously for cardiometabolic disorders: hypertensive diseases, diabetes mellitus and acute myocardial infarction (as a consequence of atherogenic dyslipidaemia) [5]. In fact, morbidity and mortality from these disorders are characteristic mainly for senescent, late phase of human ontogeny.

However, although morbidity and mortality for Alzheimer disease (Figures 7, 8 and 15, 16) were concentrated also in senescent phase, their age-related patterns occurred somewhat later, as compared with cardiometabolic disorders. Moreover, if feminine fraction for cardiometabolic disorders began to increase already since the age of menopause (~50 y), for Alzheimer disease feminine predominance occurred later on, in the decade 70-79 y and more than 80 y.




**Figures 1-3:** Morbidity from cardiometabolic disorders in the state of RS. Here and on other figures the scale of age in years is on the absciss (axis X), and relative (or proportional) parameter in per cent is on the ordinate (axis Y). Letter "a" on the scale of age indicates an interval.



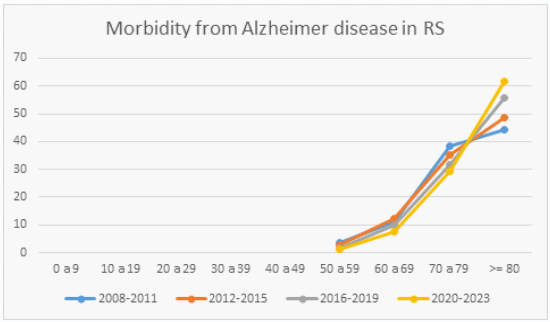

**Figures 4-6:** Feminine fraction of morbidity from cardiometabolic disorders in the state of RS.

All these data allow to suggest that present-day justification of elderly (>60 y old) and oldest old (> 80 y) subpopulations may be based on specific age-related dynamics and gender differences of morbidity and mortality.

## Discussion

Of course, since the very beginning of these epidemiologic studies we were really preoccupied with proper interpretation of the results obtained. Here we should explain what is our updated opinion about the mechanisms of age-related dynamics and gender differences.

As a matter of fact, shortly after seminal series of studies of David J.P. Barker and his colleagues (see discussion in [6]), the search began for mediators of the phenomena of perinatal programming/imprinting, and it appears that glucocorticoids may be good candidates for such role [7]. These hormones may be involved also in the phenomena of embedding occurring in later ontogeny in cumulative mode, generating allostatic load and overload, according to Bruce S. McEwen [8]. However, in order to explain the periodization of later ontogeny to two other phases (middle and late), we have to focus



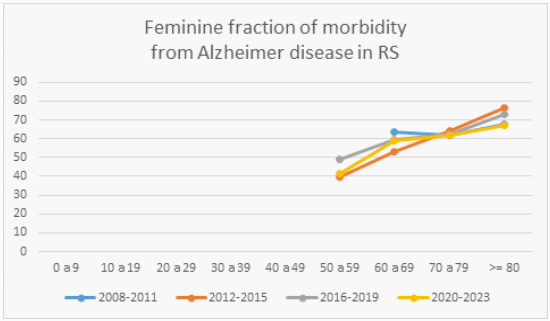
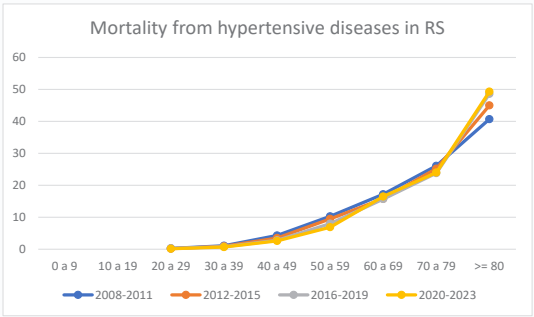
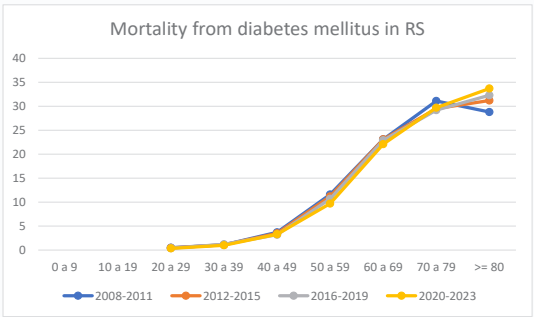
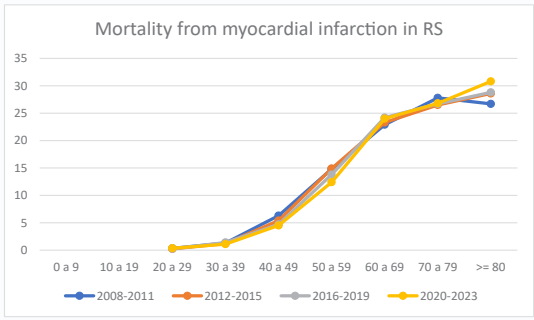



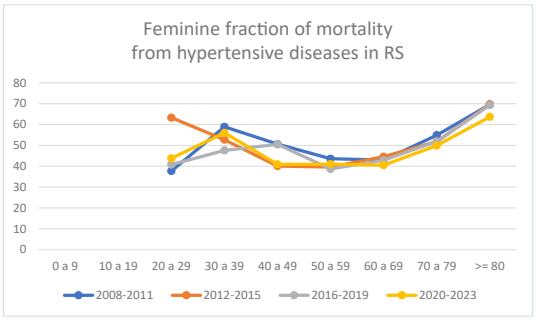

**Figure 7 and 8:** Morbidity and feminine fraction of morbidity from Alzheimer disease in the state of RS.

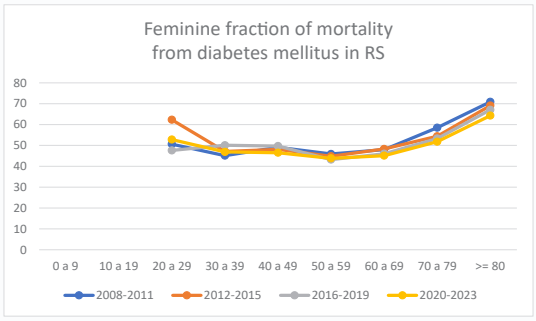


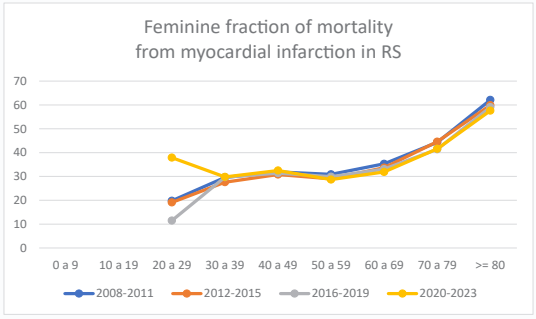



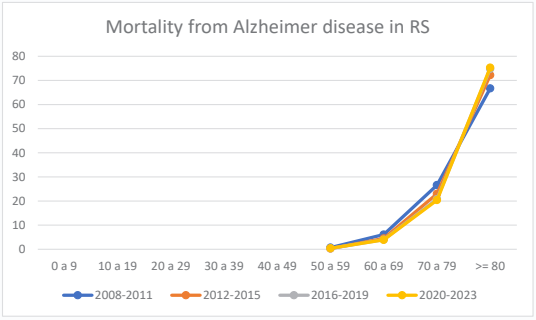

**Figures 9-11:** Mortality from cardiometabolic disorders in the state of RS.

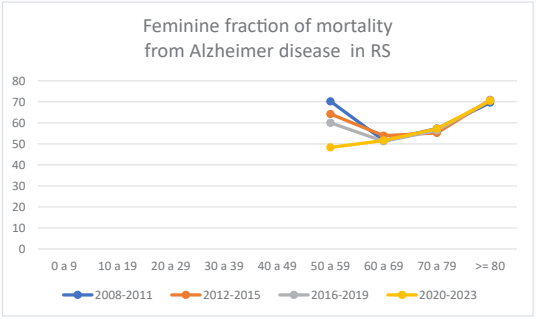



**Figures 12-14:** Feminine fraction of mortality from cardiometabolic disorders in the state of RS.




**Figures 15 and 16:** Mortality and feminine fraction of mortality from Alzheimer disease in the state of RS.

our attention also on interactions of glucocorticoids with antistress hormones, first of all, melatonin and growth hormone [9].

On the other hand, the increase in feminine fraction with the onset of menopause was interpreted by us as accelerated aging, due to rapid decrease in the production of oestrogens possessing antistress and neuroprotective properties [6, 10]. It means that age-related dynamics and gender differences for Alzheimer disease, as compared to cardiometabolic disorders should look for some other explanations.

Earlier we have noticed that from two ophthalmic disorders, only cataract followed the age-related dynamics similar to Alzheimer disease, whereas glaucoma seemed to follow better cardiometabolic disorders [11], together with cerebrovascular diseases and renal insufficiency. Probably, chronic stress and glucocorticoids in excess may result in higher peroxidation [12], and the relative lack of protection by stress proteins (heat shock proteins and others) [13] in senescent phase provokes late-onset Alzheimer disease and cataract.

## Conclusion

Of course, the investigations of age-related dynamics and gender differences for morbidity and mortality should continue for other groups of diseases (pulmonary, gastro-intestinal, etc.). Moreover, we are aware of some problems and disadvantages, as referred to the use of relative (or proportional) parameters, so we are planning at least to use other epidemiologic approaches and databases. However, the possibility to perform the periodization of human life-course is rather attractive, since it combines with the main goals of DOHaD concept, as well as with our appeal to consider gerontology as a science of whole ontogeny [14].

Nevertheless, we ought to continue also further elaboration of expanded endocrinology as a science of bioregulation by including atypical release of extracellular vesicles (exosomes) [15], probably with non-coding RNAs as principal bioregulators.

## Acknowledgement

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