

Aatola, Heikki *

Koskinen, Lasse **

Voutilainen, Raimo ***

Biological age derived from cardiorespiratory fitness assessment and its impact on life assurance business

*MD, PhD, Researcher, Department of Clinical Physiology, University of Tampere

**PhD, Professor, Insurance Science, University of Tampere

***PhD, Adjunct Professor, Insurance Science, University of Tampere

Biological age derived from cardiorespiratory fitness assessment and its impact on life assurance business

Abstract

In this paper we show that aortic aging is an important factor explaining biological age. Therefore, it is advisable to use cardiorespiratory fitness assessment (CFA) to obtain an estimate of a patient's biological age. We conclude that CFA is a potential substitute for previously widely used biological age assessment methods. We also discuss the importance of biological age in risk management.

We also study the significance of biological age to insurance business. Cohort life expectancy is the single most influential factor that insurance companies use to determine life insurance premiums. Biological age can represent a person's aging status more appropriately compared to chronological age because biological age is associated with health status. CFA is an accurate estimator of biological age. We propose to test its use in the calculation of premiums and technical reserves and underwriting processes instead of cohort life expectancy. This can be operationalized by an easy to use assessment method like CFA and standard age transfer formula.

Keywords

Biological age, aortic aging, cardiorespiratory fitness, life assurance, risk management

1 INTRODUCTION

Chronological age is the number of years a person has been alive, while **biological age** refers to how old a person seems. Biological age, also referred to as physiological age, takes many lifestyle factors into consideration, including diet, exercise and sleeping habits, to name a few. Not surprisingly, many studies have been conducted to quantitatively estimate biological age using measurable biomarkers (see e.g. Arging, 1991, Ingram et al. 2001, Simm et al. 2008, Parkes et al. 2008, and McClelland et al. 2009).

Recent research suggests that telomeres and DNA methylation play big parts in the aging process and they can be used to determine biological age (Herrmann et al. 2018, Chen et al. 2019). It is evident that each individual can affect his/her biological age by adopting healthy living habits. This is an essential part of personal risk management.

There is a direct correlation between cohort life expectancy and how much one is charged for a life insurance policy. Biological age is a concept used to describe a difference between a population cohort life expectancy and the life expectancy of an individual of the same age. Hence, an accurate estimate of biological age would be of utmost importance to actuarial calculations.

Cardiovascular diseases are the number 1 cause of death globally, and because of that, physicians and cardiologists in the life insurance industry have been particularly interested in the examination of the circulatory system. They first accepted the importance of brachial cuff blood pressure as a measure of risk already in 1917, and they have used palpation, radial artery tonometry and pulse waveform analysis to reject applicants for life insurance even earlier (Postel and Vinay, 1996).

Chronological age has been used in different risk calculators and risk assessments to provide patients with detailed information about their personal risks of cardiovascular, or overall, morbidity and mortality. However, it is well known that risk for death varies significantly among individuals of the same age. There should be a composite measure that would replace chronologic age in risk calculations to improve these

predictions. This biological age should be determined by physiology, not only by specific pathology.

Aforementioned biological age measures are made at rest, although human circulatory system appears to be designed for optimal function during physical exercise (Nichols et al. 2011). Exercise capacity is also a strong and independent predictor of morbidity and mortality in many patient populations, and it is a stronger prognostic indicator than many traditional and non-traditional cardiovascular risk factors (Blair et al. 1989, Laukkanen et al. 2004, Sui et al. 2007).

2 FITNESS AS A BIOLOGICAL AGE: PHYSIOLOGIC BACKGROUND

The aorta and large arteries have two distinct functions. They deliver blood pumped by the heart to the capillaries of bodily organs and tissues according to their need and dampen the pulsation caused by the beating heart in the circulating blood. Hence, these elastic arteries are working simultaneously and efficiently as a conduit and as a cushion. By age thirty, repetitive expansion stretches the non-living aortic elastic lamellae around billion times (about 30 million times per year at an average heart rate of 70 bpm). The cumulating stretch load wears out elastic fibers, and they begin to tear. The wall's connective tissue layer thickens, and the aorta starts to stiffen. The stiffening increases with age. It takes more effort for the heart (left ventricle) to stretch the aorta. This leads to an inevitable decrease in physical performance in middle age (Lakatta and Levy, 2003, O'Rourke and Hashimoto, 2007, Nichols et al. 2011).

It is important to notice that aortic aging is a different process than atherosclerosis. Atherosclerosis has its predisposing risk factors, the process begins in youth, and clinical manifestations (coronary artery disease, myocardial infarction, and stroke) may occur even decades later. Aortic aging is an independent process which could advance without atherosclerosis, even without any risk factors (Avolio et al. 1985, Lakatta and Levy, 2003, Dzau et al. 2006, Nichols et al. 2011). However, there are often coexistent progressions of aortic aging and atherosclerosis as introduced in The Cardiovascular Aging Continuum

(O'Rourke et al. 2010), and it is difficult to separate age-related changes from disease-related changes.

Blaha et al. (2016) reported previously that fitness-associated biological age was a stronger predictor of mortality and myocardial infarction than chronological age. These results are easy to understand when combined with decreasing physical performance caused by aortic aging mentioned above. Fitness based biological age may be comprehensive clinical tool for facilitating patient discussions regarding the impact of exercise capacity on long-term risk and compliance with important lifestyle changes. It should also be used in risk assessment rather than chronologic age.

American Heart Association released its scientific statement in 2017 (Ross et al.) and recommends annual CFA for all adults during routine clinical visits. The measured or estimated result obtained should be interpreted by using appropriate reference values (eg. Blaha et al . 2016), or by using standard reference values and the age at which that fitness level is normal, could be used as a biological age.

3 INTENSITIES IN PERSONAL RISK INSURANCE

If x is the age of the insured, the related risk intensity can be defined as

$$(1) \quad h(x) = \alpha e^{\beta x} + \lambda$$

This is called a hazard function or an intensity function. This old and widely used function is called Gompertz-Makeham function and it was presented by Makeham (1860). The formula (1) was originally used for mortality modelling, but it has been noticed that it is suitable for modelling of other personal risk insurance like critical illness, health and permanent disability as well.

Recently it has been shown that the Gompertz-Makeham function (1) is slightly too steep for old ages (75-80 years and up) compared to many observed deaths, and it requires compensation, see Gavrilov and Nosov (1985). The Gompertz-Makeham function can be used to generate a probability distribution, see e.g. Jodra (2009).

An insurance premium is obtained by multiplying the intensity function by suitable parameters. The intensity function is a part of the actuarial rules of an insurance company, and the parameters in the formula (1) are defined by performing a periodical curve fitting between the function (1) and real mortality data.

Recently cohort mortality models have grown popular, see e.g. Sanders (2017). Mortality is also an important factor in statistical demography research, see Alho and Spencer (2005). Alho (2016) shows that female and male mortality have converged in Europe.

4 AGE TRANSFER IN PREMIUM RATING

It has been noticed that the mortality of a certain group A of insureds can sometimes be obtained by adding a parameter to the mortality of another group B of insureds. Referring to the formula (1), the intensity function for group A can be defined as

$$h_A(x) = h(x + \tau),$$

where x is the age of a member of group A, and τ is an age transfer. The only difference from the basic formula (1) is that the age x in the exponent is replaced by $x + \tau$.

A practical example is the mortality model used by many Finnish life insurance companies, where the intensity of a woman is the same as the intensity of a seven years younger man. If the group of women is denoted by A, we get

$$h_A(x) = h(x - 7),$$

where the age transfer is -7 . This kind of mortality modelling results in different premiums for men and women, and it was forbidden by EU Court in 2012. It is allowed, however, in the calculation of technical reserves.

5 USE OF BIOLOGICAL AGE IN PREMIUM RATING AND UNDERWRITING

Insurance needs to be to a certain degree collective to be insurance. However, moderate individualizing can bring mutual benefits for an

insured and an insurer, as Voutilainen and Koskinen (2017) show. Biological age gives an interesting method of individualizing premium rating and underwriting procedures. What is important, a customer can influence his/her premium and underwriting results by lowering his/her biological age by healthy living habits. In that sense, biological age driven insurance is incentive based, cf. Voutilainen and Koskinen (2017).

Let CHRON and BIO be an individual's chronological and biological ages, respectively. If BIO is directly available, the intensity function is simply $h(\text{BIO})$, which determines the corresponding premium. The difference $\text{BIO} - \text{CHRON}$ can also be used as an age transfer. If we denote it by AT, we get the intensity function $h(\text{CHRON} + \text{AT})$, which determines the corresponding premium.

The use of biological age in underwriting is straightforward: simply replace chronological age by biological age in the underwriting procedure. Thus, denial and extra premiums are now the result of biological age instead of chronological age.

In Finland the majority of personal insurance lines use age-dependent tariffs and can therefore benefit from the use of biological age as a tariff factor. These lines are at least the following:

- term life insurance
- critical illness insurance
- health insurance
- permanent disability insurance
- mandatory pension insurance (TyEL), disability and old age pension

Exponential risk models of type (1) in Ch. 3 are widely used and therefore significant. In most of the above insurance lines the risk model is exponential, but this does not limit the use of biological age. To achieve risk-based tariffs biological age should be used in premium rating, but it has also significance in technical reserves. For example, in mandatory pension insurance, motor third party liability insurance and workers' compensation insurance pensions are reserved in claims reserves. The biological age of an insured gives much better picture than the chronological age about how long the pension will be payable.

6 BIOLOGICAL AGE AND RISK MANAGEMENT

Most risk management articles in the literature refer to risk management as a company, organization or industry related topic. For example, the recent handbooks of Hopkin (2018) and Hillson (2016) define risk management as a corporate concept. So do the articles of Dionne (2013) and Haimes (2009) who discusses “risks to systems”.

Boyle (2015) and Kavalier and Alexander (2014) cover risk management in health sector.

Insurance company risk management is governed in EU by the Solvency II regime. It requires among other things that insurance companies reserve funds for different age-related underwriting risks. They include changes caused by e.g. mortality, longevity, morbidity, disability and lapse in the risk position of an insurance company. See e.g. Christiansen and Niemeier (2014). For example, longevity and disability risks are discussed by Levantes and Menziatti (2012) and Jarner and Möller (2015).

Knowledge of individual customers’ biological age gives an insurance company a much better way to assess the underwriting risk according to Solvency II than chronological age. Even good estimates give this advantage. After all, biological age is the “real” age taking into account diet, exercise and sleeping habits et al. The procedure sketched at the end of Ch. 2 is a promising effective way to accomplish this.

So far, we have discussed corporate risk management. The risk management for individual persons found in the literature can be classified into wealth risk management and health risk management. Wealth risk management is discussed by e.g. Michaud and Michaud (2008). According to our knowledge, wealth risk management is not related to biological age, even if this could be possible.

Biological age and patient health risk management are discussed by the following authors:

Soriano-Tarraga et al. (2018) discuss biological age as a predictor of mortality in ischemic stroke. Age and stroke severity are the main mortality predictors after ischemic stroke. However, chronological age and biological age are not exactly concordant. As estimated by DNA

methylation, biological age is an independent predictor of 3-month mortality in ischemic stroke.

Farquharson et al. (2001) study the importance of biological age in surgical decisions in the elderly. These results indicate that decisions on surgical management are strongly influenced by the patient's star rating or biological age. Here a simple biological age assessment was used.

Hospers et al. (2015) discuss the relation between blood pressure and mortality risk in an older population and there the role of chronological and biological age. In a large population-based cohort of older adults, low diastolic blood pressure was associated with an increased all-cause mortality risk, especially in the oldest old and in biologically old individuals.

Chen et al. (2016) present a meta-analysis on DNA methylation-based measures of biological age. Estimates of biological age based on DNA methylation patterns have been shown to be robust biomarkers of age in humans. Overall, the study of Chen et al. strengthens the evidence that biological age predicts all-cause mortality above and beyond chronological age and traditional risk factors, and demonstrates that biological age estimates that incorporate information on blood cell counts lead to highly significant associations with all-cause mortality.

Kang et al. (2017) propose a metabolic syndrome biological age model, through which overall evaluation and management of the health status and aging state in metabolic syndrome can be done easily.

Meisel et al. (2019) show that biological age combines various measures into a single score and allows identifying individuals at increased risk of tooth loss.

Liang et al. (2016) study effects of biological age on the associations of blood pressure with cardiovascular and non-cardiovascular mortality in old age.

The CFA procedure described at the end of Ch. 2 is an alternative to produce biological age estimates for the above-mentioned health risk management cases.

CONCLUSIONS

In this paper we have shown that aortic aging is an important factor explaining biological age. Therefore, it is advisable to use CFA to obtain an estimate of a person's biological age.

It would be interesting to compare the performance of the CFA for biological age to the previously widely used biological age assessment methods like those using telomeres and DNA methylation.

Furthermore, the procedure described at the end of Ch. 2 would be an alternative to produce biological age estimates for the health risk management cases referred in Ch. 6 and also other cases reported in the literature.

We have also discussed the significance of biological age to the risk management of insurance companies. Biological age can be taken into account in actuarial functions by simple age transfer. We have discussed the potential significance of biological age for premium rating, underwriting and technical reserves. Inclusion of biological age in these areas would require an easy-to-use assessment method, maybe something like the CFA discussed in this paper.

REFERENCES

Alho J (2016), Descriptive Findings on the Convergence of Female and Male Mortality in Europe, *ETLA (The Research Institute of the Finnish Economy) working paper 40/2016*, 301 pp.

Alho J and Spencer B (2005), *Statistical Demography and Forecasting*, Springer, ISBN 978-0-387-23530-1, 412 pp.

Arking R (1991), *Biology of Aging: Observations and Principles*. Prentice Hall, Inc: Englewood Cliffs.

Avolio AP, Deng FQ, Li WQ, Luo YF, Huang ZD, Xing LF and O'Rourke MF (1985), Effects of aging on arterial distensibility in populations with high and low prevalence of hypertension: comparison between urban and rural communities in China, *Circulation* 71:202-210.

Blaaha MJ, Hung RK, Dardari Z, Feldman DI, Whelton SP, Nasir K, Blumenthal RS, Brawner CA, Ehrman JK, Keteyian SJ and Al-Mallah MH

(2016), Age-dependent prognostic value of exercise capacity and derivation of fitness-associated biologic age, *Heart* 102:431–437.

Blair SN, Kohl HW 3rd, Paffenbarger RS Jr, Clark DG, Cooper KH and Gibbons LW (1989), Physical fitness and all-cause mortality: a prospective study of healthy men and women, *Journal of the American Medical Association* 262:2395–2401.

Boyle T (2015), *Health and Safety: Risk Management*, Routledge, 556 pp.

Chen B, Marioni RE, Colicino E, Peters MJ, Ward-Caviness CK, Tsai PC, Roetker NS, Just AC, Demerath EW, Guan W, Bressler J, Fornage M, Studenski S, Vandiver AR, Moore AZ, Tanaka T, Kiel DP, Liang L, Vokonas P, Schwartz J, Lunetta KL, Murabito JM, Bandinelli S, Hernandez DG, Melzer D, Nalls M, Pilling LC, Price TR, Singleton AB, Gieger C, Holle R, Kretschmer A, Kronenberg F, Kunze S, Linseisen J, Meisinger C, Rathmann W, Waldenberger M, Visscher PM, Shah S, Wray NR, McRae AF, Franco OH, Hofman A, Uitterlinden AG, Abscher D, Assimes T, Levine ME, Lu AT, Tsao PS, Hou L, Manson JE, Carty CL, LaCroix AZ, Reiner AP, Spector TD, Feinberg AP, Levy D, Baccarelli A, van Meurs J, Bell JT, Peters A, Deary IJ, Pankow JS, Ferrucci L and Horwath S (2016), DNA methylation-based measures of biological age: meta-analysis predicting time to death, *Aging* 8(9): 1844–1859.

Chen M, Wong E, Nguyen T, Dite G, Stone J, Dugue P, Giles G, Southy M, Milne R, Hopper J, Li S (2019), DNA methylation-based biological age, genome-wide average DNA methylation, and conventional breast cancer risk factors, *Scientific Reports* 9, 15055, [nature.com](https://doi.org/10.1038/s41598-019-50555-2).

Christiansen M and Niemeyer A (2014), Fundamental definition of the solvency capital requirement in Solvency II, *ASTIN Bulletin*, 44(3), 501-533.

Dionne G (2013), Risk management: History, definition, and critique, *Risk Management and Insurance Review* 16(2), 147-166.

Dzau VJ, Antman EM, Black HR, Hayes DL, Manson JE, Plutzky J, Popma JJ and Stevenson W (2006), The cardiovascular disease continuum validated: clinical evidence of improved patient outcomes: part I: Pathophysiology and clinical trial evidence (risk factors through stable coronary artery disease), *Circulation* 114:2850-2870.

Farquharson SM, Gupta R, Heald RJ and Moran BJ (2001), Surgical Decisions in the Elderly: The Importance of Biological Age, *Journal of the Royal Society of Medicine*, 94(5), 232–235.

Gavrilov L and Nosov V (1985), A new trend in human mortality decline: derectangularization of the survival curve, *Age* 8(3): 93.

Haimes Y (2009), On the complex definition of risk: A systems-based approach, *Risk Analysis* 29(12), 1647-1654.

Herrmann M, Pusceddu I, März W, Herrmann W (2018), Telomere biology and age-related diseases, *Clinical Chemistry and Laboratory Medicine* 56(8), 1210-1222.

Hillson, D. (2016), *The Risk Management Handbook: A Practical Guide to Managing the Multiple Dimensions of Risk*, Kogan Page Publishers, 336 pp.

Hopkin, P. (2018), *Fundamentals of Risk Management: Understanding, Evaluating and Implementing Effective Risk Management*, Kogan Page Publishers, 480 pp.

Hospers GP, Smulders Y, Maier A, Deeg D, Muller M (2015), Relation between blood pressure and mortality risk in an older population: role of chronological and biological age, *Journal of Internal Medicine* 227(4), 488-497.

Ingram DK, Nakamura E, Smucny D, Roth GS, Lane MA (2001), Strategy for identifying biomarkers of aging in long-lived species, *Experimental Gerontology* 36:1025–1034.

Jarner S & Möller T (2015), A partial internal model for longevity risk, *Scandinavian Actuarial Journal*, 2015:4, 352-382.

Jodra P (2009), A closed-form expression for the quantile function of the Compertz-Makeham distribution. *Mathematics and Computers in Simulation* 79(10), 3069-3075.

Kang YG, Suh EK, Chun HJ, Kim SH, Kim DK and Bae CY (2017), Models for estimating the metabolic syndrome biological age as the new index for

evaluation and management of metabolic syndrome, *Clinical Interventions in Aging* 12, 253-261.

Kavaler F and Alexander R (2014), *Risk Management in Healthcare Institutions: Limiting Liability and Enhancing Care*, Jones & Bartlett Publishers, 530 pp.

Lakatta EG and Levy D (2003), Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: Part I: aging arteries: a "set up" for vascular disease, *Circulation* 107:139-146.

Laukkanen JA, Kurl S, Salonen R, Rauramaa R and Salonen JT (2004), The predictive value of cardiorespiratory fitness for cardiovascular events in men with various risk profiles: a prospective population-based cohort study, *European Heart Journal* 25:1428–1437.

Levantes S and Menziatti M (2012), Managing longevity and disability risks in life annuities with long term care, *Insurance Mathematics and Economics* 50(3), 391-401.

Liang Y, Fratiglioni L, Wang R, Santoni G, Welmer AK, Qiu C (2016), Effects of biological age on the associations of blood pressure with cardiovascular and non-cardiovascular mortality in old age: A population-based study, *International Journal of Cardiology* 220, 508-513.

Makeham W (1860), On the Law of Mortality and the Construction of Annuity Tables, *Journal of the Institute of Actuaries and Assurance Magazine* 8, 301-310.

McClelland RF, Nasir K, Budoff M, Blumenthal RS and Kronmal RA (2009), Arterial Age as a Function of Coronary Artery Calcium (From the Multi-Ethnic Study of Atherosclerosis [MESA]), *American Journal of Cardiology* 103: 59–63.

Meisel P, Pink C, Nauck M, Völzke H and Kocher T (2019), Construction of a Biological Age Score to Predict Tooth Loss over 10 Years, *Journal of Dental Research* 98(10), 1096–1102.

Michaud R and Michaud R (2008), *Efficient Asset Management, A Practical Guide to Stock Portfolio Optimization and Asset Allocation*, 2. ed, Oxford University Press

Nichols WW, O'Rourke MF and Vlachopoulos C (2011), *McDonald's Blood Flow in Arteries: Theoretical, Experimental and Clinical Principles, 6th edition*. London: Hodder Arnold.

O'Rourke MF and Hashimoto J (2007), Mechanical factors in arterial aging: a clinical perspective, *Journal of the American College of Cardiology* 50:1-13.

O'Rourke MF, Safar ME and Dzau V (2010), The Cardiovascular Continuum extended: Aging effects on the aorta and microvasculature, *Vascular Medicine* 15:461-468.

Parkes G, Greenhalgh T, Griffin M and Dent R (2008), Effect on smoking quit rate of telling patients their lung age: the Step2quit randomised controlled trial, *British Medical Journal* 336:598–600.

Postel-Vinay N (1996), *A Century of Arterial Hypertension 1896–1996*, Chichester: John Wiley

Ross R, Blair SN, Arena R, Church TS, Després J-P, Franklin BA, Haskell WL, Kaminsky LA, Levine BD, Lavie CJ, Myers J, Niebauer J, Sallis R, Sawada SS, Sui X, Wisløff U (2016), On behalf of the American Heart Association Physical Activity Committee of the Council on Lifestyle and Cardiometabolic Health; Council on Clinical Cardiology; Council on Epidemiology and Prevention; Council on Cardiovascular and Stroke Nursing; Council on Functional Genomics and Translational Biology; and Stroke Council. Importance of assessing cardiorespiratory fitness in clinical practice: a case for fitness as a clinical vital sign: a scientific statement from the American Heart Association, *Circulation*, 2016;134: e653–e699.

Sanders S (2017), *Period and cohort life expectancy explained. Guide to the two types of life table – period and cohort, used to calculate past and projected life expectancy*, Office for National Statistics, UK, December 2017.

Simm A, Nass N, Bartling B, Hofmann B, Silber RE, Navarrete SA (2008), Potential biomarkers of ageing, *Journal of Biological Chemistry*, 389:257–65.

Soriano-Tárraga C, Mola-Caminal M, Ois A, Rodriguez-Campello A, Cuadrado-Godia E, Fernandez-Cadenas I, Cullell N, Roquer J, Jimenez-

Conde J (2018), Biological Age is a predictor of mortality in Ischemic Stroke, *Scientific Reports* 8, nr. 4148.

Sui X, LaMonte MJ and Blair SN (2007), Cardiorespiratory fitness as a predictor of nonfatal cardiovascular events in asymptomatic women and men, *American Journal of Epidemiology* 165:1413–1423.

Voutilainen R and Koskinen L (2017). Customers' opinions on incentive based insurance, *Journal of Insurance and Financial Management*, 3(1), 30-52.