



Universiteit
Leiden
The Netherlands

When longevity meets vitality

Westendorp, R.G.J.; Schalkwijk, F.H.

Citation

Westendorp, R. G. J., & Schalkwijk, F. H. (2014). When longevity meets vitality, 73, 407-412. doi:10.1017/S0029665114000573

Version: Not Applicable (or Unknown)

License: [Leiden University Non-exclusive license](#)

Downloaded from: <https://hdl.handle.net/1887/117465>

Note: To cite this publication please use the final published version (if applicable).



Conference on ‘Nutrition and healthy ageing’ Plenary Lecture I: The John Waterlow Lecture

When longevity meets vitality

Rudi G. J. Westendorp^{1,2*} and Frank H. Schalkwijk^{1,2}

¹Leyden Academy on Vitality and Ageing, Poortgebouw LUMC, Rijnsburgerweg 10, 2333 AA Leiden, The Netherlands

²Department of Gerontology and Geriatrics, Leiden University Medical Center, PO Box 9600, 2300 RC Leiden, The Netherlands

Alarmed by the sustainability of our health and social security systems, longevity has become a great societal challenge. In line with evolutionary logic we see a continuous increase of average life expectancy and maximal lifespan. Striving for a healthy old age, however, is an infelicitous expression as for human subjects the ageing process cannot be ultimately postponed. Not disregarding the huge variation in health trajectories, in old age we will all suffer from frailty and infirmity. As yet efforts of the biomedical arena are almost exclusively focused on stalling the ageing process and preventing dysfunction. Too little effort is spent on how to inspire and coach the great majority of people who still feel relatively well notwithstanding the presence of multiple age-related disorders. There is a strong rationale to separate the quest to live in good health for longer from actively and effectively negotiating the challenge of functional decline in old age. In particular, we emphasise a focus on adjusting the environment in order to correct the gene–environment mismatch that contributes to ill health. An additional strategy is to empower people to set ambitions and to realise appropriate goals, in spite of infirmity. Striving for vitality presents a striking opportunity to achieve subjective feelings of life satisfaction when ageing.

Ageing: Healthy ageing: Environmental interventions: Vitality

John Conrad Waterlow, physiologist, (13 June 1916–19 October 2010) was a highly esteemed and well-known physiologist and nutritional scientist. From 1954 to 1970, he was Director of the Medical Research Council Tropical Metabolism Research Unit at the University of the West Indies. He became Professor of Human Nutrition, London School of Hygiene and Tropical Medicine in 1970. In 1982, he was elected Fellow of the Royal Society of London for Improving Natural Knowledge. He is widely appreciated for his work on malnutrition in children. Additionally, in 1975, he was one of the first to recognise the consequences of obesity for public health, as chair of a government committee in the United Kingdom⁽¹⁾.

The root cause of ageing

In 1891, August Weismann wrote one of the first evolutionary theories on ageing, in which he hypothesised

the existence of an intrinsic death mechanism that would affect only the old and weak subjects of a population. In this way it was argued that sufficient space and resources would be guaranteed for the younger, reproducing generations to prevent the species from going extinct⁽²⁾. Although this reasoning seems logical, the existence of a ‘programmed’ death mechanisms is highly unlikely from an evolutionary perspective. There is no sound mechanism by which death genes might have a beneficial effect on the individual and would thus be selected for in the population genome⁽³⁾. Moreover, a bird’s-eye view across the tree of life shows that there is a great variation in the course of mortality with age, among the various species’ constant, decreasing, humped and bowed mortality trajectories that can be observed⁽⁴⁾. The generalised idea that life inevitably leads to ageing can thus be falsified. The sobering truth, however, is that *Homo sapiens* is a prime example of those species that exhibits an ageing phenotype with the consequence that we will suffer from functional decline, disability and handicaps when growing older.

*Corresponding author: Rudi G. J. Westendorp, fax +31 71 5248159, email westendorp@leydenacademy.nl

Ageing comes about because of the accumulation of permanent damage and the rate of occurrence is determined by the impact of intrinsic and extrinsic stressors counteracted by endogenous and exogenous repair. It is the probability of repair function that is the likely explanation why ageing can be avoided⁽⁴⁾. As we grow old, like all other mammals, the accumulated damage will result in typical ageing phenotypes and we will start to 'look old'. Damage accumulation, in cells and tissues also explains the incidence of age-related diseases, such as CVD, cancer or dementia. This is why ageing is the most important single risk factor for chronic degenerative diseases to occur. This theory on damage accumulation, first proposed by Medawar provides an evolutionary background to why ageing occurs⁽⁵⁾. He was the first to put forward that in a natural environment, under adverse environmental conditions, people used to die young. Consequently, selection pressure was high for mutations with an effect at a young rather than old age. Medawar further suggested that mutations that have a deleterious effect in old age only, will not be selected against and therefore can accumulate in the gene pool. Although there is as yet little evidence for the idea that late-acting deleterious mutations have accumulated in the population genome, this does not exclude that deleterious mutations accumulate in the DNA of individual cells over one's lifetime and may give rise to cellular dysfunction, senescence or cancer⁽⁶⁾. Old age is a period in life that has little or no influence on the fitness of the individual, with little consequences on evolutionary development of the species and is therefore also referred to as the 'selection shadow'.

The absence of a genetic programme for death, and its inverse a healthy longevity, does not impede the differences in the way that people age having a genetic underpinning. Twin studies showed that approximately a quarter of lifespan variation can be attributed to genetic factors^(7–10). Furthermore, several studies showed clustering of longevity in families^(11–13). In our own research, we have found that life histories of offspring of these long-lived families clearly differed from their spouses with whom they had shared a great deal of their lifetime and environmental exposures. The offspring had lower mortality risk, less diabetes, hypertension and CVD⁽¹⁴⁾. Furthermore, they had better lipid profiles⁽¹⁵⁾, higher insulin sensitivity⁽¹⁶⁾, slightly less circulating thyroid hormone⁽¹⁷⁾, slightly lower circulating cortisol⁽¹⁸⁾ and positive outcome expectations⁽¹⁹⁾.

Over the last two decades, studies in experimental animals have identified several cellular signalling pathways that partially explain the influence of genetic differences on longevity. In these pathways, components of stress and nutrient sensing pathways including, insulin, insulin-like growth factor 1 and mammalian target of rapamycin were identified as important players⁽²⁰⁾. Since these pathways are linked to metabolism, energy expenditure and energy allocation, the effect of dietary interventions on human longevity gained obvious attention and has led to the idea that dietary restriction may lead to a lengthening of our lifespan. The effectiveness of these interventions in human subjects is still highly

controversial⁽²¹⁾. Especially so as two long-running intervention studies on great apes have shown evidence for preventing age-related diseases but have not been conclusive on the eagerly expected effect on life span extension^(22,23). Several excellently written reviews provide a more elaborate background on the complex and intriguing nature of the biology of ageing^(20,24).

The quest for healthy ageing

The evolutionary approach when studying the biology of ageing has led to a more thorough understanding of why and how ageing occurs, but it is questionable if the concomitant focus on extending longevity will suffice to address the challenges of our ageing societies. With the present longevity revolution, the great majority of new borns will become old and once old will live longer, and most people reach an age far into the selection shadow at which chronic degenerative diseases will occur. This raises important questions on the quality of these extra years gained. For this purpose, the quest for 'healthy ageing' seems more appropriate and is presently central to the agenda of many research programmes, conferences and political reports.

The idea of healthy ageing emerged in 1980, when Fries outlined his views on the compression of mortality and morbidity⁽²⁵⁾. He argued that with the incremental emphasis on prevention and successful treatment of disease we would all survive in good health up to old age and then die in a relatively short period of time due to the supposed death mechanism. However, the existence of a genetically determined death mechanism is, as mentioned earlier, highly unlikely and population data are in line with this reasoning; average and maximal lifespan have increased relentlessly⁽²⁶⁾. Moreover, we have recently reported on Dutch data showing that the average period of disability at the end of one's life has remained the same but is delayed until older age⁽²⁷⁾. In other words, the age at which disabilities are reported has increased to the same degree as life expectancy. Some of us are rather puzzled as the number of years lived with morbidity has even increased, but the trend of earlier disease diagnosis and prevention complicates the interpretation.

Notwithstanding our personal wish and political desire, data on the evolution of our life course under affluent conditions show that 'healthy ageing' is an infelicitous expression, as it implies that the deterioration of the human body due to the accumulation of damage can be avoided. The good part of the story is that the ageing process in human subjects can increasingly be slowed down and the occurrence of degenerative disease successfully postponed. At the end of life however, the great majority of people have to cope with several co-morbid conditions, disabilities and handicaps⁽²⁸⁾ and a sole focus on physical or mental health may thus be inappropriate for improving well-being.

Both the issues of health in later life and the well-being of old people are fundamental when taking up new personal and societal responsibilities in our rapidly greying societies. Here, we will explore two approaches that we

deem necessary when addressing these issues. First, we will take a closer look at the traditional strategy of improving health in our present affluent environment in order to postpone age-related disease and disability. Then, we will argue that efforts should be made to better inspire and support the majority of elderly that will ultimately suffer from chronic disease and disability, by applying the concept of vitality.

Ageing in today's environment

With the start of the industrial revolution and more specifically, the increased economic prosperity over the last century, average life expectancy has increased from about 40 to 80 years. Underlying the longevity revolution is an incremental decrease in frailty during young and middle age. Whereas in the beginning it was the disappearance of child mortality that was driving the increase of life expectancy, over the last five decades the rapid decline in mortality figures of the older generation has fuelled the increase of life expectancy from 70 to 80 years. There are no signs that this trend is coming to an end⁽²⁶⁾.

There is general agreement that the close interactions of genes and environmental factors determine the rate and the nature of the ageing process. In contrast, the contribution of chance as a third 'factor' to explain for individual life-course trajectories is undervalued. All individuals from the same species, even experimental animals from the same inbred lines that are reared under similar environmental conditions, age markedly differently because interactions between genes and the environment will always be dissimilar in time and place⁽²⁹⁾. The revolutionised genomic techniques have allowed us to study the impact of genetic variance on the length of lifespan in animal studies and human populations. Whereas induced or spontaneously occurring mutations can have a major effect on the course of the lifespan, there is hardly explanatory power of genomics to explain the existing variation in age of death in (human) populations⁽³⁰⁾. Moreover, it is unlikely that within the small timeframe that our life expectancy has doubled, natural selection has significantly and causally changed our population genome. The rapid increase in life expectancy that has taken place must thus be attributable to the improvements in the environment in which we live. Phrased otherwise, we now better exploit the original potential of the ancient lay out for body and mind that results in a longer and healthier lifespan.

Not all recent changes in our present environment have had a beneficial effect on our health. Consider the sharp increase of many age-related diseases, such as CVD and diabetes that are best explained by the slow accumulation of permanent damage to heart, vessels and pancreas. It can also be explained as the outcome of a mismatch of our genome and our present environmental conditions. Under the adverse environmental conditions that our genome was shaped, food was scarce and fierce physical labour was necessary to gather sufficient means. Day and night we were craving for food and felt perfectly

comfortable with that; satiation and exercise-induced endorphin production being perfect examples of our past evolutionary development. The present affluent environment could not have been more different. Food is abundantly available at low costs in Western households and physical activity is no longer necessary to survive. On an evolutionary scale this complete environmental turnover has occurred instantaneously, the human genome is expressing an energy saving behaviour as if nothing has changed. This gene–environment disparity undoubtedly expedites the epidemic of age-related cardiovascular and metabolic diseases in our rapidly developing world. To find further evidence for this reasoning, we have studied health and survival of a human population in a rural area in Ghana where people had lived, and are still living in an environment that is very similar to the environmental conditions in which our genome was shaped. In this remote place there is only a relatively small chance that new-borns make it into old age as hygiene, healthcare and affluence are virtually absent. When the young make it to a high age however, they are not likely to suffer from the typical age-related diseases that are so abundant in the developed world. We found that only very few older individuals had signs or symptoms of CVD and none of them suffered from diabetes, the main cause of death in old age being infectious diseases^(31,32).

The notion that environmental conditions are essential to influence the occurrence of age-related disease may not seem a remarkable finding. However, the detrimental interaction of our ancient genome with our modern environment does represent an essential insight on how to improve health over our life course. As can be inferred from the Ghanaian example, these preventive strategies can be extremely powerful but are notoriously under exploited. Almost by default, present preventive strategies are aimed at improving health through individual behavioural change, such as diets and exercise programmes. As we have pointed out, not only our biology but also our behaviours are anchored in our genome, which explains the poor outcomes of attempts to convince people to make healthier choices. It is not that we should stop teaching health literacy, but just hammering on individual habits to provoke behavioural change may not be the way forward. Here we contend that there is an urgent need to get rid of the extraordinary cues in the present Western environment that trigger our ancient, genetically engrained behaviours with adverse outcomes. Next to a public ban on smoking, we should reconsider the massive presence of fast-food and the ample opportunities for passive movement as promising public interventions to overcome the gene–environment mismatch that underlies the incidence of age-related disease⁽³³⁾.

Our living environment can be adjusted in various ways and it does not need draconian measures to better maintain our health. People themselves can make relatively small adjustments to their personal environment, which stimulate healthy behaviours, preferably by tricking our genetic predispositions. For example, marketing research and dietary studies show that visibility and availability of food strongly influence our intake^(34,35).

Numerous studies in fast-food restaurants, cafeterias and lunchrooms in schools show that people more often choose a healthier option when visibility and availability of healthy options is improved^(36,37). Furthermore, recent studies on the use of small tableware and serving sizes have shown encouraging results^(38,39). There is a rapidly increasing amount of literature on how to tweak the environmental signals to influence our food intake⁽⁴⁰⁾. In the same vein, physical activity can be stimulated by 'smart' triggers in the environment. A recent Australian study among 222 497 adults has shown that sitting time is a risk factor for all-cause mortality, independent of physical activity⁽⁴¹⁾, the remarkable conclusion being that 'sitting is the new smoking'. Installing a standing desk at work is an example how to reduce the amount of sitting hours per day.

Earlier we have highlighted only a few examples of how to stimulate healthy behaviours by changing the environment, instead of trying to change individual behaviour directly. Many more can be listed, but most important is the thorough understanding of the human mind when designing and introducing strategies to elicit a healthy lifestyle. It is the scientific knowledge of human behaviour that should be structurally and consciously incorporated by strong-minded health politicians to make the right things happen.

Vitality in old age

Older people without major disease, disability and handicap consider themselves as lucky. From a biomedical perspective those individuals are considered as slow agers and represent an elite at the positive extreme of growing old⁽⁴²⁾. They do age however, and at later age most of this elite will suffer from disease and infirmity, as the ageing process cannot ultimately be postponed. Most are less lucky and in late age have worn out their bodies with the result that the overwhelming majority of older persons has to cope with several co-morbid conditions, disabilities and handicaps and are considered unhealthy⁽²⁸⁾. What follows is an adapted and shortened version of a text in which we recently pointed out that the judgement of the biomedical arena is a juxtaposition of the mind set of older people themselves, as the overwhelming majority of older people judge themselves as healthy⁽⁴³⁾.

Some years ago, we set out for a combined qualitative and quantitative study on successful ageing amongst older people from the general population to understand the difference between the biomedical 'outsider' perspective and the psychological 'insider' perspective. According to the WHO definition of health, we found that at age 85 years only one out of ten had minor physical disabilities, good mental function, regular social activities and high feelings of well-being⁽⁴⁴⁾. Most of those aged 85 years had to be categorised as unhealthy because of suboptimal physical and mental function, but only a small minority rated their own health as poor or very poor. Almost half of these older people scored an optimal state of well-being in response to the question: 'Are

you satisfied with your life?' indicative for the 'disability paradox', i.e. that people can feel good despite disease, disability and handicap.⁽⁴⁵⁾ More generalised, 85 year olds on average rated the quality of their lives with eight points out of ten. The qualitative part of the study showed that older people considered being successful not as a matter of good physical and mental functioning, but above all as a satisfactory adaptation to functional limitations in old age⁽⁴⁶⁾. The clear message is that a sole focus on healthy ageing is not sufficient as it is only attainable for the lucky few, whereas good self-rated health and feelings of well-being in old age appear to be the norm and dependent on yet not clearly identified determinants.

In the social sciences it has been emphasised that vitality is an important characteristic to reach well-being in old age^(47,48). It is an essential competence to make use of one's functional abilities. It consists of having the motivation to take up responsibilities, the knowledge and skills to do so and ultimately the gift to enjoy the results. Without that there is no appreciation of well-being. Among the attributes of vitality are introspection, positive affect, energy, engagement, resilience, self-esteem, coping, autonomy, sense of purpose, and these may all be essential to reach a satisfactory life. Of course, it requires the constant balancing of the possible with the impossible and of the available with the non-available. This is especially so for older people who have developed disabilities and handicaps. It is for this reason that we propose to operationalise vitality as 'the ability of a person to set ambitions appropriate for one's life situation and being able to realise these goals'.

Triggered by the economic crisis of 2008 and pressure on the sustainability of our health and social security systems, healthy ageing has become a 'hot topic'. Foremost it prevails in the narrow, functional definition of health, the outsider perspective, as medical professionals use it. The attention for vitality, the insider perspective, is lagging far behind. The medical arena considers it to be a psychological concept that is primarily part of a distinct social domain. However, the two concepts are not independent of each other. For example, people who are able to maintain a positive attitude, even in the face of disability and handicap display overall better outcomes of disease and lower mortality⁽⁴⁹⁾. Those who feel better, do better. Also, offspring of long-lived families display a more optimistic outlook on life and have better health outcomes, further emphasising the close interplay between the two notions⁽¹⁹⁾. It is tempting to conclude that when health in the narrow definition is deteriorating, vitality plays a pivotal role in assuring well-being despite functional limitations.

Conclusion

We contend that the debate in developed countries on how to accommodate the rapidly increasing lifespan of their citizens is too much focused on addressing the infirmities of frail elderly, whereas too little effort is spent on how to inspire and coach the great majority of people

who still function relatively well notwithstanding the presence of multiple age-related morbidities. The rationale is to separate the quest for a healthy longevity from actively and effectively negotiating the challenges of ageing. Empowering citizens to set ambitions and achieving appropriate goals, in spite of age, functional decline or morbidity presents a striking opportunity to achieve subjective feelings of satisfaction over the life course from which society at large could benefit.

Acknowledgements

This manuscript is a written report of a tribute to John Waterlow addressed at the Nutrition Society Annual Summer Meeting, 15–18 July 2013, Newcastle, UK.

Financial support

This work is supported by the Leyden Academy on Vitality and Ageing and the Leiden University Medical Center.

Conflicts of interest

None.

Authorship

R. W. and F. S. drafted and finalised this manuscript based on the lecture given by R. W. at the Nutritional Society Annual Summer meeting 2013.

References

- Alleyne G, Picou D, Forrester T *et al.* (2011) John conrad waterlow CMG, FRS, FRCP, DSc obituary. *Br J Nutr* **106**, 1–5.
- Weismann A (1891) *Essays upon Heredity and Kindred Biological Problems*, 2 ed., vol. 1. Oxford: Clarendon Press.
- Kirkwood TB & Cremer T (1982) Cytogerontology since 1881: a reappraisal of August Weismann and a review of modern progress. *Hum Genet* **60**, 101–121.
- Jones OR, Scheuerlein A, Salguero-Gomez R *et al.* (2014) Diversity of ageing across the tree of life. *Nature* **505**, 169–173.
- Medawar PB (1952) An Unsolved Problem of Biology: An Inaugural Lecture Delivered at University College, London, 6 December, 1951: H.K. Lewis and Company.
- Hoeijmakers JH (2009) DNA damage, aging, and cancer. *N Engl J Med* **361**, 1475–1485.
- Finch CE & Tanzi RE (1997) Genetics of aging. *Science* **278**, 407–411.
- Herskind AM, McGue M, Holm NV *et al.* (1996) The heritability of human longevity: a population-based study of 2872 Danish twin pairs born 1870–1900. *Hum Genet* **97**, 319–323.
- Iachine IA, Holm NV, Harris JR *et al.* (1998) How heritable is individual susceptibility to death? The results of an analysis of survival data on Danish, Swedish and Finnish twins. *Twin Res* **1**, 196–205.
- Ljungquist B, Berg S, Lanke J *et al.* (1998) The effect of genetic factors for longevity: a comparison of identical

- and fraternal twins in the Swedish Twin Registry. *J Gerontol A Biol Sci Med Sci* **53**, M441–M446.
- Perls TT, Bubrick E, Wager CG *et al.* (1998) Siblings of centenarians live longer. *Lancet* **351**, 1560.
- Schoenmaker M, de Craen AJ, de Meijer PH *et al.* (2006) Evidence of genetic enrichment for exceptional survival using a family approach: the Leiden Longevity Study. *Eur J Hum Genet* **14**, 79–84.
- Kerber RA, O'Brien E, Smith KR *et al.* (2001) Familial excess longevity in Utah genealogies. *J Gerontol A Biol Sci Med Sci* **56**, B130–B139.
- Westendorp RG, van HD, Rozing MP *et al.* (2009) Nonagenarian siblings and their offspring display lower risk of mortality and morbidity than sporadic nonagenarians: the Leiden Longevity Study. *J Am Geriatr Soc* **57**, 1634–1637.
- Heijmans BT, Beekman M, Houwing-Duistermaat JJ *et al.* (2006) Lipoprotein particle profiles mark familial and sporadic human longevity. *PLoS Med* **3**, e495.
- Wijman CA, Rozing MP, Streefland TC *et al.* (2011) Familial longevity is marked by enhanced insulin sensitivity. *Aging Cell* **10**, 114–121.
- Rozing MP, Westendorp RG, de Craen AJ *et al.* (2010) Low serum free triiodothyronine levels mark familial longevity: the Leiden Longevity Study. *J Gerontol A Biol Sci Med Sci* **65**, 365–368.
- Noordam R, Jansen SW, Akintola AA *et al.* (2012) Familial longevity is marked by lower diurnal salivary cortisol levels: the Leiden Longevity Study. *PLoS ONE* **7**, e31166.
- Rius-Ottenheim N, Kromhout D, de Craen AJ *et al.* (2012) Parental longevity correlates with offspring's optimism in two cohorts of community-dwelling older subjects. *Age (Dordr)* **34**, 461–468.
- Partridge L & Gems D (2002) Mechanisms of ageing: public or private? *Nat Rev Genet* **3**, 165–175.
- Cava E & Fontana L (2013) Will calorie restriction work in humans? *Aging (Albany NY)* **5**, 507–514.
- Colman RJ, Anderson RM, Johnson SC *et al.* (2009) Caloric restriction delays disease onset and mortality in rhesus monkeys. *Science* **325**, 201–204.
- Mattison JA, Roth GS, Beasley TM *et al.* (2012) Impact of caloric restriction on health and survival in rhesus monkeys from the NIA study. *Nature* **489**, 318–321.
- Kirkwood TB (2005) Understanding the odd science of aging. *Cell* **120**, 437–447.
- Fries JF (1980) Aging, natural death, and the compression of morbidity. *N Engl J Med* **303**, 130–135.
- Vaupel JW (2010) Biodemography of human ageing. *Nature* **464**, 536–542.
- Engelaer FM, Van Bodegom D & Westendorp RG (2013) Sex differences in healthy life expectancy in the Netherlands. *Annu Rev Gerontol Geriatr* **33**, 361–371.
- World Health Organization (2005) *Preventing Chronic Disease. A Vital Investment: WHO Global Report*. Geneva.
- Finch CE & Kirkwood TBL (2000) *Chance, Development, and Aging*. New York: Oxford University Press.
- Beekman M, Blanche H, Perola M *et al.* (2013) Genome-wide linkage analysis for human longevity: genetics of Healthy Aging Study. *Aging Cell* **12**, 184–193.
- Koopman JJ, Van Bodegom D, Jukema JW *et al.* (2012) Risk of cardiovascular disease in a traditional African population with a high infectious load: a population-based study. *PLoS ONE* **7**, e46855.
- Koopman JJ, Van Bodegom D, Beenakker KG *et al.* (2012) Hypertension in developing countries. *Lancet* **380**, 1471–1472.



33. Matheson GO, Klugl M, Engebretsen L *et al.* (2013) Prevention and management of non-communicable disease: the IOC consensus statement, Lausanne 2013. *Br J Sports Med* **47**, 1003–1011.
34. Chandon P, Hutchinson JW, Bradlow ET *et al.* (2009) Does in-store marketing work? Effects of the number and position of shelf facings on brand attention and evaluation at the point of purchase. *J Mark* **73**, 1–17.
35. Wansink B, Painter JE & Lee YK (2006) The office candy dish: proximity's influence on estimated and actual consumption. *Int J Obes (Lond)* **30**, 871–875.
36. Downs JS, Loewenstein G & Wisdom J (2009) Strategies for promoting healthier food choices. *Am Econ Rev* **99**, 159–164.
37. Thorndike AN, Sonnenberg L, Riis J *et al.* (2012) A 2-phase labeling and choice architecture intervention to improve healthy food and beverage choices. *Am J Public Health* **102**, 527–533.
38. Wansink B, van Ittersum K & Painter JE (2006) Ice cream illusions bowls, spoons, and self-served portion sizes. *Am J Prev Med* **31**, 240–243.
39. van Ittersum K & Wansink B (2012) Plate size and color suggestibility: the Delboeuf Illusion's bias on serving and eating behavior. *J Consum Res* **39**, 215–228.
40. Larson N & Story M (2009) A review of environmental influences on food choices. *Ann Behav Med* **38**(Suppl 1), S56–S73.
41. van der Ploeg HP, Chey T, Korda RJ *et al.* (2012) Sitting time and all-cause mortality risk in 222 497 Australian adults. *Arch Intern Med* **172**, 494–500.
42. Rowe JW & Kahn RL (1997) Successful aging. *Gerontologist* **37**, 433–440.
43. Westendorp RG, Mulder B, Van der Does W *et al.* (2014) When vitality meets longevity. New strategies for health in later life. In *Wellbeing: A Complete Reference Guide. Volume IV: Wellbeing in Later Life* [TB Kirkwood and CL Cooper, editors]. London: John Wiley & Sons.
44. von Faber M, Bootsma-van der Wiel A, van Exel E *et al.* (2001) Successful aging in the oldest old: who can be characterized as successfully aged? *Arch Intern Med* **161**, 2694–2700.
45. Pavot W & Diener EV (1993) Review of the satisfaction with life scale. *Psychol Assess* **5**, 164–172.
46. Baltes PB & Baltes MM (1993) Psychological perspectives on successful ageing: the model of selective optimization with compensation. In *Successful Aging: Perspectives from the Behavioral Sciences*, pp. 1–34 [PB Baltes and MM Baltes, editors]. Cambridge: Cambridge University Press.
47. Ryan RM & Frederick C (1997) On energy, personality, and health: subjective vitality as a dynamic reflection of well-being. *J Pers* **65**, 529–565.
48. Ryan RM & Bernstein JH (2004) Vitality. In *Character Strengths and Virtues: A Handbook and Classification: A Handbook and Classification*, pp. 273–290 [C Peterson and MEP Seligman, editors]. New York: Oxford University Press.
49. Kim ES, Park N & Peterson C (2011) Dispositional optimism protects older adults from stroke: the Health and Retirement Study. *Stroke* **42**, 2855–2859.