

# Life Expectancy Issues in Life Care Planning

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### Abstract

The present paper is a review of life expectancy and life expectancy determination in forensic applications, including life care planning. Part One discusses basic aspects of life expectancy determination and five current approaches to the challenge. Part Two begins with general considerations regarding modification of the standard population life table for individual comorbidities and other influences on mortality, and then provides a stepwise analysis of life expectancy determination by this method, including selection of the baseline life table, identification of relevant medical comorbidities and other conditions, quantification of mortality effects of those factors with information from the medical literature, combination of mortality indices to generate adjusted mortality rates, and construction of a new, modified life table reflecting the effects of mortality influences. Part Three reviews potential future developments in life expectancy determination, and Part Four furnishes a summary. Appendices provide support for the text.

This article addresses some of the strengths, weaknesses, and controversies in current practice of life expectancy determination, with the goal of furthering understanding and application of the science (and art) involved in achieving the most scientifically-accurate and valid results in life care planning. Specific aspects discussed include a) selection of the baseline life table; b) identification of relevant influences for the individual under scrutiny, including the multiplicity of elements, both positive and negative; the effects of socio-economic factors, functional status, and medical care; and potential sources of information; c) accurate quantification of influences, encompassing the proper use of mortality indices; d) combination of baseline mortality rates and comorbidity effects, including overlap in mortality effects of relevant conditions; “double-dipping” by use of standard life tables; and adjustment for secular trends in mortality; and e) generation of a modified life table and adjusted life expectancy. The article ends with a brief discussion of possible future advances in the science and practice of life expectancy determination.

*Keywords:* Comorbidity, comparative mortality, excess death rate, expected mortality, hazard ratio, life care planning, life expectancy, life expectancy determination, life table, relative risk, mortality ratio, observed mortality, rated age, rating up, relative risk

### Part One: Introduction and Theoretical Considerations

Deutsch (2010) defined life care planning as “a consistent methodology for analyzing all of the needs dictated by the onset of a catastrophic disability through the end of life expectancy” (p. 4). Life expectancy determination is an integral component of the life care plan, including in litigation estate and pension planning (OECD, 2011). The accuracy

of the estimate is critical for the ethical nature economic and legal legitimacy of the proposal. Goodrich (2013) in an editorial opined that “... life expectancy is a critical issue and arguably the single most significant variable affecting the total value of the life care plan” (p. 1).

Most life care planners will defer the determination of accurate life expectancy to standard population life tables or other experts involved in a case. However, knowledge of the considerations and process behind life expectancy determination may assist the planner in generating the most valid strategy for a given individual. Kush et al. (2013) anticipated that:

As life care planners become more familiar with the concept and science of life expectancy, we are hopeful that collaborative efforts to advance the knowledge of the impact of one on the other may take place, and real scientific evidence may emerge. (p. 45)

There is still a relative lack of specific information on life expectancy determination for life care planning, although a series of articles in the July 2013 issue of the *Journal of Life Care Planning* presented some basic considerations (Krause & Saunders, 2013; Kush et al., 2013; Rosen et al., 2013; Shavelle & Strauss, 2013).

### Definitions of Life Expectancy

The human lifespan or survival time – the actual number of years that a person will live – is the outcome of a complex calculus including genetic, developmental, environmental, physiological, psychobehavioral, and sociocultural influences (Ben-Haim et al., 2018; Crosse, 2011). In the present context, life expectancy refers to the average anticipated survival time, or years of life remaining, of members of a defined population group of people (population) with certain identifying demographic characteristics such as gender, geographic location, nationality, and race, with which an individual of interest may be identified (Giannias et al., 2014). Life expectancy is a population parameter, a statistical construct based on the actual mortality rate of members of the group in a given time interval, determined by combining a series of age-specific mortality rates in a life table to replicate the mortality experience of the population (Vachon & Sestier, 2013).<sup>1</sup> Day et al. (2015a) described life expectancy as “... the average for an individual represented by a given cohort [group of people] if such an individual could (hypothetically) live life repeatedly” (p. 1106).

By definition, life expectancy is an average value for members of a population; it is currently not possible to accurately predict any given individual’s exact lifespan or survival time (i.e., the number of years, months, and days of life remaining), but it is both necessary and feasible to determine summary measures of survival likelihood (Shavelle & Delaney, 2010). Tolley et al. (2016) observed that “... ”

only an answer as a statistical probability can be obtained. A statistical answer entails an average or ‘expected value’ with an associated level of uncertainty” (p. 262). Singer (2005) concluded that “The best we can do in our precise life table calculation of life expectancy is to admit that this is a conditional estimate, and that its future forecast is NOT an equally precise estimate” (p. 105).

Life expectancy is not the same as median survival time, which is defined as the “middle” anticipated lifespan of a population (the 50th percentile), or the point at which half of the members will be deceased (i.e., live less) and half will be alive (i.e., live more).<sup>2</sup> Median survival is generally less susceptible to the presence of unusual or outlying results (DeVivo, 2002), but may underestimate life expectancy in conditions with high initial or early mortality such as severe myocardial infarction (MI), cerebrovascular accident (CVA), or trauma (Anderson, 2002). Median survival may be used in the legal setting to satisfy the condition of “more likely than not”, but is a different quantity from life expectancy, with its own uses, such as determination of probabilities of survival at a particular age or for a given duration (Vachon, 2019). Tolley et al. (2016) argued that the most relevant measure of life expectancy probability, “more likely than not”, was the interval between the 25th and 75th percentiles (as determined from the life table), with the latter most important as the upper boundary of the range of most probable survival (p. 267).

### **Importance of Life Expectancy Determination Demography and Epidemiology**

Life expectancy serves as a key indicator of development and health in a population and is a basic guide to economic and social planning and policy, such as social security systems (Wilmoth, 2000).

### **Medical Evaluation and Treatment**

Reasonably accurate estimation of life expectancy (i.e., prognosis) is important in determination of the validity, timing, and application of screening tests for medical conditions. It is critical for establishing appropriateness and cost-utility of treatment in disease conditions which are likely to result in limited lifespan, such as cancer (Gill, 2012; Yourman et al., 2012).

### **Insurance and Planning Functions**

Life expectancy determination has obvious implications for disability, health, life, and long-term care insurance. It is used in underwriting and pricing of insurance products and essential to reasonable forecasting of future payments in annuity instruments such as those funding structured settlements (Reid, 2013; Ryan & Harbin, 2006) as well as valuation in life settlement investment (Bhuyan, 2009). Other related applications include Medical Cost Projections

(MCPs) and Medicare Set-Asides (MSAs). Life expectancy determination has a prominent role in personal financial and particularly retirement planning (Krueger, 2011).

### **Determination of Damages in Medicolegal and Personal Injury Tort Actions**

Life expectancy is widely employed as an index of reduced survival in the establishment of damages and compensation (Brookshire et al., 2007), e.g., in wrongful death actions (Ireland, 2016).

### **Life Care Planning**

Accurate life expectancy determination is a crucial facet of both qualitative and quantitative life care planning (Day et al., 2015b). However, as noted, life care planners do not routinely perform life expectancy determination (unless they are specifically qualified to do so); many rely upon standard life tables such as those published by the U. S. National Center for Health Statistics (NCHS; Arias & Xu, 2019), without adjustment, for baseline values, and defer more precise opinion on life expectancy to other experts such as biostatisticians, epidemiologists, and forensic economists. Without specialized education and experience, physicians may have no expertise in life expectancy determination (see Anecdotal or Experiential Opinions by Clinicians below), although stand-alone certification in life care planning has been available for physicians, particularly specialists in physical medicine and rehabilitation, or physiatry, since 2013 (CPLCP Certification Board, 2020; Rosen et al., 2013).

### **Measurement in Life Expectancy Determination**

In epidemiology, public health, biostatistics, and other areas of scientific evaluation, parameters such as life expectancy have several important characteristics. *Validity* is the extent to which the life expectancy determination measures what it is intended to measure (the average expected length of life), and *accuracy* is the degree to which the life expectancy represents the true value of the eventual lifespan) of the individual; in life expectancy determination they may be considered to be roughly synonymous. Both can be compromised by bias or systematic error, or uncontrolled and/or unrecognized influences on life expectancy which are not considered or recognized in an analysis. *Reliability* is the degree to which repeated applications of the life expectancy determination procedure will provide the same or similar result, and *precision* is how close repeated trials will be to each other; these also be thought of as roughly synonymous, although reliability generally refers to the process, and precision to the end result, of such a determination (Trajkovic, 2008).

### **Ethics in Life Expectancy Determination for Life Care Planning**

Inaccurate life expectancy determination in life care planning presents significant ethical and moral risks. Underestimation of life expectancy (in which case the individual outlives the expectancy) may result in unmet need for services not included in the life care plan and inadequate provision of resources for the individual's requirements. Overestimation of life expectancy (in which case the person dies before the anticipated time) may result in unfair or wasteful provision of excessive assets for the reasonable needs of the individual. Goodrich (2013) declared that "...the life care planner has a fiduciary responsibility to adequately fund a plan that will allow the evaluatee to maximize his or her health and independence regardless of retention from the plaintiff or the defendant" (p. 1-1). There are a number of other sources of ethical guidance for the life care planner:

- Multiple professional organizations (AANLCP, 2020; AAPLCP, 2020; IARP, 2007 and 2015; ICHCC, 2020) provide practice guidelines and standards for life care planners, most commonly asserting that practitioners must maintain objectivity in client assessment and should not advocate for subjects of their planning activities (including, by inference, inaccurately estimating life expectancy).
- Gibbs et al. (2013) presented a proposed model code of conduct for psychiatrists working in life care planning encompassing six areas of responsibility related to the life care planning process.
- Reid (2013) extensively discussed the ethical risk of these errors in life care planning, especially underestimation (which may become a self-fulfilling prophecy), noting societal concerns (in terms of inefficient resource allocation), humanitarian concerns for the subject of the plan (both in terms of quality of life and survival), and professional concerns in terms of the validity of the plan and consequent credibility of the planner. This author opined that life care planners were obviously accountable for the validity and reliability of the life care plan that they generated, but also responsible, within their particular jurisdictional and legal context, for acting if they perceived a clear danger to the subject from underestimation of life expectancy.
- Berens & Weed (2019) provided an excellent chapter on ethical issues for the life care planner, outlining general considerations, guidelines for conduct, and many specific recommendations for practice.

### **Current Life Expectancy Determination in Individuals**

There are at least six methods of life expectancy appraisal presently in common use: this section addresses a) anecdotal opinion by clinicians, b) use of unmodified population life

tables, c) "rating up", d) use of observed life expectancy, and e) clinical prediction models, and Part Two concentrates on life table modification.<sup>3</sup> (It should be noted that all of these techniques focus predominantly or completely on negative influences on life expectancy. Positive influences on life expectancy are discussed in Part Two, Step Two below.)

### **Anecdotal or Experiential Opinions by Clinicians**

This approach involves a subjective impression or judgment of the individual providing the life expectancy determination, usually a physician, relying heavily or solely upon clinical professional observations, and experience. However, Day & Reynolds (2015) asserted that "A medical degree (and/or clinical experience) is neither a necessary nor a sufficient qualification for being an expert on life expectancy" (p. 2 of 6). This tactic often does not include application of formal statistical analysis or use of the life tables (the latter of which is basic to life expectancy determination; see discussion in next section), or any systematic process leading to the conclusion. Vachon (2020) commented that "Life expectancy derives from a scientific calculation based on mortality rates, not from medical insight. No amount of clinical introspection will generate a life table." Physicians are often not aware of medical evidence on longevity for a given condition, and their clinical experience may be limited to those with recent injuries, excluding long-term survivors (Krause, 2002). As a result of these shortcomings, these life expectancy opinions are not robust, subject to many sources of bias and error (Christakis, 1999; Vachon, 2019), and vulnerable to multiple lines of attack by opposing counsel (Rice et al., 2000; Strauss & Shavelle, 1998a). Sammon et al. (2015) found accuracy of anecdotal appraisal to be inferior to actuarial methods; clinicians generally underestimate life expectancy (Clarke et al., 2009b; Leung et al., 2012) and demonstrate great variability in their estimates (Wirth & Sieber, 2012); they are particularly inaccurate in cancer patients (Glare et al., 2003; Walz et al., 2007; Wilson et al., 2005).

### **Use of Unmodified Life Tables<sup>4</sup>**

Life expectancy is determined in defined collections of individuals, either by use of census and other demographic data to generate standard life or mortality tables or use of proprietary client information to produce insurance tables. The final life expectancy value represents a summary of the much more extensive information contained in the life table, such as age-specific mortality rates and annual survival probabilities (Kush et al., 2013). Day et al. (2015b) contended that "Any rational, evidence-based assessment of life expectancy must be associated with a corresponding life table" (p. 254).

Life tables express the survival experience of a group under the influence of a process, in this case mortality (Burch,



2018). General population life tables are usually *period* tables based on observed age-specific mortality rates; these are “synthetic” tables fitting that information into a customary format (Arias & Xu, 2019). *Cohort* or generational tables are less commonly used, and present actual life expectancy data for a defined group of people over their entire lifetimes. Cohort tables may be somewhat more accurate than period (Ayuso et al., 2018), but they require accumulation of information from the birth of the first to the death of the last member of the group. Individual insurance and annuity tables are fairly specialized to relatively narrow groups with limited generalizability and thus are used less often than the general population tables in life expectancy analysis. There are five basic types of life tables, ranked in general order of mortality rates from highest to lowest (Pokorski, 1988):<sup>5</sup>

1. General population tables, which provide expected mortality data for a large group of interest, e.g., the entire U.S. population
2. Cohort tables,<sup>6</sup> which depict mortality in a more narrowly defined collection of persons, e.g., the Framingham study (Kit, 2020)
3. Group insurance tables (in which most of the covered individuals will be working and thus somewhat healthier than the general population)
4. Individual insurance tables (in which most substandard and uninsurable lives will have been eliminated)
5. Annuity tables (in which participants are self-selected)

Life tables for the general U.S. population are created by the NCHS. These period tables are based on actual mortality rates derived from census data and most commonly provide life expectancy estimates for large demographic groups (e.g., by gender and race), stratified by age. Comprehensive versions are generated from the results around the decennial census conducted in the United States every 10 years and Medicare data, with annual updates; prior versions remain readily available. At the time of this writing, the most up-to-date life tables for the U.S. are based on 2017 final mortality statistics, population estimates for 2017 based on the 2010 decennial census, and 2017 Medicare data for persons aged between 66-99 years (Arias & Xu, 2019).

The methodology of life table construction is not complicated (it is based largely on arithmetical calculations), but details are beyond the scope of the present paper. Anderson (1999) and Singer (1998) provided excellent overviews, and the stepwise process is explained in the Technical Notes section of the annual NCHS updates (e.g., Arias & Xu, 2019, p. 57ff.). Basically, the life table begins with actual mortality rates, generally designated as *mx* for the population of interest. An arbitrary population number is chosen as a starting point at age 0 yrs (the radix), which by convention (and convenience) in population tables is 100,000 persons. Three basic steps are followed: a) the actual age-specific

mortality rate (or more precisely, the probability of death) is applied to each age stratum in the table (usually 1 year) to determine the average (hypothetical) population contributed by each year, in person-years of life; b) the total number of person-years is determined for each age interval, and c) the cumulative person-years at each age stratum are divided by the number of persons alive during the interval to determine the life expectancy for that interval (Anderson, 2002).

Life expectancy values from life tables generated in this way are readily available, and are routinely used in insurance, legal, and medical settings, including disability and Workers Compensation (WC) insurance (e.g., for setting reserves on claims and determining cost projections).<sup>7</sup> However, the life expectancy figures provided by a standard life table are average population values, and thus may not be accurate for an individual with one or more conditions that significantly influence their life expectancy. For example, the table figures may undervalue mortality and thus overestimate the life expectancy of persons who have significant health problems or engage in behaviors that increase their risk of death (e.g., cigarette smoking or excessive alcohol use); conversely, the table figures may overrate mortality and thus underestimate the life expectancy of those who have significant compensating factors (Krause & Saunders, 2013; see Use of the Modified Life Table for Life Expectancy Determination in Individuals below).

### **Observed Life Expectancy by Condition**

Some empirical studies (i.e., based on observed data or experience rather than theory or modeling) quantify life expectancy in terms of actual number of years of life found to be remaining in a population in the presence of one or more specific disorders. This method can either determine the average number of years for a given set of conditions such as age or functional category (e.g., Keeler et al., 2010), or a more “generic” approach using number or severity of conditions (e.g., Cho et al., 2013). This technique is particularly useful in assessing the overall life expectancy effects of limited numbers of influences and in examining the simultaneous effects of multiple conditions (comorbidity or multimorbidity). For example, DuGoff et al. (2014) determined that each additional chronic disorder (ranging from 0 to 10+) decreased life expectancy by a marginal decline of 1.8 yrs (ranging from 0.4 yrs for the first to 2.6 yrs for the sixth condition). In addition, Vachon (2019) noted that the “constant absolute” decrease in life expectancy by these techniques still varied with the individual’s age and other demographic factors such as geographic origin (p. 615). In practice, the number of disorders that can be considered in this way is limited, and it is not as precise as other methods useful for life expectancy determination in the medicolegal setting.

Some researchers estimate life expectancy by statisti-

cally “matching” individual patients to those with similar characteristics and known lifespans in large existing databases. Krause (2002) furnished an extensive discussion of this approach, noting requirements that a) the index person was representative of the database, or came from the same population; b) a subset of cases could be identified from the database and matched to the individual; and c) perhaps most importantly, that the process accounted for most of the salient mortality influences, including nonmedical factors such as socioeconomic status. More recently, DeVivo (2018) described derivation of life expectancy tables in this way from the National Spinal Cord Injury Statistical Center (NSCISC) database, incorporating patient age and neurologic function category (although also noting that other relevant factors were important in the final life expectancy determination in patients with spinal cord injury or SCI).

Certain variations on these observational approaches are not scientifically sound or appropriate, such as deducting a fixed number of years or percentage of life expectancy for a given condition (due to nonlinearity of the life table), or inappropriate comparison to groups of individuals with a given condition with known life expectancies (Vachon, 2020).

#### ***Rated Age (“Rating up” or “Impaired Risk Rating”)***

This method involves advancing an individual’s chronological age by a certain number of years to reflect the effects of one or more medical conditions, then assuming that the person’s mortality is that of the reference population for the advanced rather than original age (Kita, 2006). The rated age reflects what underwriters at individual insurance companies believe a claimant’s life expectancy will be; different companies use different factors to formulate rated ages, so the values may vary significantly, and typically several estimates are obtained and combined in a given case. This process requires review of the individual’s medical records to determine a “customized” age rating, but has the virtue of simplicity, allowing use of the standard life table with no modification. However, there are several problems with this approach:

- The information by which rated age is determined is usually proprietary to the insurer or other entity and thus carefully guarded, leaving it largely inaccessible to those outside of these organizations.
- Jones (2013) opined that the rated age (or “set forward”) practice has the virtue of simplicity, but “requires considerable judgment and is difficult to collect and maintain for a large population of claims” (p. 17).
- This procedure typically involves collecting several “rated ages” from different insurers, so it is difficult to know which, if any is accurate, and to this author’s knowledge there is no generally accepted objective method for combining multiple estimates. For example, the U.S. CMC

(2020) only requires use of the “median of all rated ages submitted” (p. 43) in WC claims.

- There are multiple technical concerns with this process:
  - Vachon (2020) observed that human mortality progresses exponentially with age (known as a Gompertz function),<sup>8</sup> but the mortality associated with most chronic diseases increases at a much less than exponential rate, concluding that rating up forces an improper mortality pattern onto the individual with such a disorder.
  - Adjustment for comorbidity uses demographic parameters including excess death rate (EDR) or mortality ratio (MR; see Part Two, Step Three below); as discussed at length in that section, use of a constant MR projected into higher attained ages will usually overestimate mortality and thus underestimate life expectancy. The rating up technique provides results similar to increasing the annual death rate from census data by a fixed amount each year, i.e., applying a fixed mortality ratio (MR) to the entire life table (see, for example, Anderson, 2002, Endnote 5.1, p. 125, and Singer & Schmidt, 2000, p. 146).
  - Strauss et al. (2001) pointed out that this method may result in a reasonable mean estimate of life expectancy but given an erroneous variance of the estimate. This does not allow more detailed approximation of risk for a given individual, specifically generation of the distribution of the survival curve and values in an adapted life table (which, for example, allow determination of the probability of death and survival at any given age). The authors cited the example of a “normal” man and a child with cerebral palsy, both of whom would have a rated age of 58 yrs and consequent calculated mean survival time of 20 yrs, but who would have completely different risk probability distributions, i.e., the risk of death at any time, and thus mortality patterns (p. 81).
- Due to potential differences in the health characteristics of the underlying population, group insurance data may not be appropriate as a basis for rated ages in workers compensation (WC) or disability claims.
- In an MSA blog entry advocating for use of independent underwriters for rated age determination, MEDVAL (2011) opined that:
 

Rated ages as provided by a life company are not always true indications of diminished life expectancy. As any broker will attest, they are as much about an individual life companies’ appetite for financial risk as true mortality risk. Some companies will return a standard on a cancer patient or quad [tetraplegic patient]

just because they do not want to assume the risk of providing lifetime payments. And their aggressiveness will shift during any given year based on the number of certain types of substandard risks that they have already written. Rated ages are mostly about pricing annuities in a competitive market environment. . . . Finally, based on CMS' requirements that a median rated age be calculated when more than one is submitted, the rated ages used in MSAs can be artificially low. But there is no requirement to submit more than one rated age so anyone that meets their absurdly strict requirements for formatting and independence will do. The result from an independent underwriter is generally one or two years greater than a median rated age that has to factor in a standard or an unreasonably low rated age. This has the potential of saving thousands of dollars per MSA whether funded as a lump sum or annuity."

- In a comparison of five methods of estimation of mortality rates at advanced ages, Strauss et al. (2005) demonstrated that "rating up" overestimated older-age mortality (and thus underestimated life expectancy) by approximately 20% (the most of the five techniques compared). In addition, with clear relevance to life care planning, Strauss et al. (2000b, 2001), and Shavelle (2020) found that this method overestimated expected present value (EPV) of lifetime care costs when compared to other approaches.

### ***Clinical Prediction Models***

These simulations use statistical techniques and algorithms to apply population data on mortality for various disorders to the demographic information for an individual to estimate life expectancy. Early examples included the Charlson Comorbidity Index (CCI), developed to predict risk of death within one year of hospitalization for patients with one or more of 19 specific conditions (Charlson et al., 1987; Charlson et al., 1994) and the Elixhauser Score, which employed administrative data to anticipate in-hospital mortality and other patient outcomes (Elixhauser 1998; University of Manitoba, 2019). Both of these measures utilize regression analysis, and have been validated (Austin et al., 2015). Gagne et al. (2011) reported that a combination of the two indices predicted mortality up to one year. Beck et al. (1982a and b) illustrated a simple method of characterizing mortality risk based on life table and additional mortality data for use in clinical decision making (the "DEALE"). Clarke et al. (2009a) described development of a computerized clinical prediction model based on age, gender, and 19 comorbid disorders (the Measure of Actuarial Life Expectancy or MALE). Mariotto et al. (2013) devised a complex comorbidity-adjusted prediction process using SEER-Medicare Data and Cox proportional hazards modeling which estimated life expect-

tancy somewhat more accurately than the standard life tables available at the time. Lee et al. (2014) predicted life expectancy in older adults based on a clinical point system and the Gompertz survival function (see Footnote #7), although these authors were only able to validate the results to life expectancies up to 10 yrs.

It appears that while these instruments may have some utility for individual life expectancy estimation, they are not yet sufficiently accurate or robust to serve as a basis for medicolegal decision making. Sammon et al. (2015) evaluated nine life expectancy calculators available to the public via the Internet at that time, concluding that "it is unclear whether available statistical models provide any advantages over freely available government tables" (p. 754). Yourman et al. (2012) reviewed 16 non-disease specific prognostic indices for older adults, determining that this approach may improve accuracy of clinical decision, but that multiple problems with bias and generalizability prevented wide application. Rector et al. (2016) studied eight prediction models described in 11 papers published between 2011 and 2016, deciding that acceptable performance for estimating 1–10-year life expectancy in older adults was "feasible" (p. 27), but that validation and actual usage in practice were yet to be established.

### **Part Two: Life Table Adjustment Methodology Use of the Modified Life Table for Life Expectancy Determination in Individuals**

#### ***Usual and Prevailing Practice***

Adjustment of baseline life expectancy (either general population or based on proprietary insurance data, such as intercompany studies) for mortality influences and/or illness or injury resulting from tort is accepted and customary practice in the disability, health, and life insurance and life settlement industries (Academy of Life Underwriting, 2019), and forensic and legal settings. Elements of this approach date back at least to the 17th century (Ciecka, 2008; Jones, 2010), but quantitative application of excess mortality risk (and the basic concept of addition of mortality influences on the general population values) is credited to the early 20th century work of Rogers and Hunter (1919; Clarke et al., 2009b). For example, insurers routinely correct life table results for comorbidity, e.g., using table ratings and flat rate additions to premiums (Kita, 2006). Although there is some disagreement about precise methods, life table modification has been described in both major reference texts (Anderson, 2002; Brackenridge, Croxson, & Mackenzie, 2006; Lew & Gajewski, 1990; SOA, 1998) and many more focused articles (e.g., Anderson & Marion, 2005; DeVivo, 2018; Krause & Saunders, 2013; Kush et al., 2013; Lai et al., 1996; Shavelle & Strauss, 2013; numerous commentaries by Singer, e.g., 1992, 1998, and 2005a and b; Slesnick & Thornton, 2008; Strauss



et al., 2005; Vachon, 2019; and Vachon & Sestier, 2013). Strauss & Shavelle (1998b) specified that a life expectancy calculation requires “a combination of (a) multivariable survival analysis on a sufficiently large sample, to estimate the patient’s mortality rate during the study period, and (b) a method of extrapolating the rate over the whole life span” (p. 97), i.e., life table adjustment. Anderson (2002) summarized the modern process as “routinely creating a computerized life table for each individual case, and then combining clinical experience with the results of scientific follow-up studies (i.e., epidemiology) to produce sound estimates of life expectancy for legal purposes” (p. 8). It is also accepted practice in the medicolegal setting; for example, DeVivo (2002) related that “preference for the NSCISM-produced life expectancy tables over general population tables has been confirmed in appellate court” (p. 50).

#### **“Double-Counting” in Life Table Adjustment**

General population life tables incorporate nearly every influence on mortality, no matter how large or small. Krause & Saunders (2013) observed that standard life tables included healthy adults as well as those with typical representative ranges of health conditions, behaviors related to health, and access to health care services observed within the general population, and thus “they do not represent estimates for only healthy adults or those who do not report significant health behavioral issues (e.g., smoking)” (p. 52). Singer (2006) reflected that mortality in any random sample of the general population could be visualized “... as an aggregate rate, a weighted mean of a series of groups, a large fraction of persons in good health, and smaller fractions definable by progressively increasing excess mortality risks” (p. 43).

A common criticism of use of the general population life table as a basis for adjustment in life expectancy determination is that since it represents all members of the population to which the individual belongs, all medical and other conditions potentially affecting an individual’s life expectancy are already incorporated into the table data, and thus correction for comorbid disorders can “double count” the effects, overestimating mortality and underestimating life expectancy.<sup>9</sup> For example, Donnelly (2015) stated:

Cherry-picking aggravating health factors and making a 100-percent deduction for them from average life expectancy overlooks the fact that average life expectancy figures already include thousands of people with the same aggravating health factors, and so an actuarial deduction for that condition, e.g., obesity, high blood pressure, etc., has already been made. Without truly precise and rarified actuarial expertise, a defense expert might, say, subtract five years off work life or general life, but have no clue or no real legal basis to show whether that condition had already taken 2.5 years off everyone’s average life expect-

tancy. ... Worse yet, the average life expectancy cohorts contain thousands of people with worse life-shortening conditions, such as cancer or the whole host of genetic or environmental factors that reduce life expectancy much more markedly, and which this plaintiff patient does not have. So, will the defense expert subtract those from his subtraction or give life-extending credit for Hamlet’s “thousand natural shocks that flesh [and life expectancy tables] is heir to,” but which this plaintiff fortunately dodged?” (Page 2 of 2)

Some analysts (e.g., Kessler, 2004 and 2020) proposed that a more appropriate starting point for life table modification is an insurance-based data set which eliminates many of these potential influences on mortality, i.e., that of a “healthy individual” (2020, p. 50; candidates for life insurance will have been vetted for many mortality influences, with more thorough evaluation corresponding to group, individual, and annuity policies). Shavelle (2012) suggested subtraction of a correction factor (e.g., 40%) from the general population mortality rates, based on the difference between those and the mortality rates of life insurance policy holders. Although both of these approaches have theoretical justification, use of the general population life table as a substrate for adjustment has several advantages.

**Established Practice.** Use of the U.S. population life tables as a basis for modification is generally accepted (Singer 2005a, 2006), and this method has been adopted by the American Academy of Insurance Medicine (AAIM, 2015).

**Elimination of Occult Influences.** As noted above, a person’s eventual lifespan is the sum total of all mortality influences over their lives, including positive and negative, and direct and indirect effects. These elements may act, and interact, in complex ways about which we presently have only an incomplete and imperfect understanding (and may never comprehend fully). Artificially eliminating some of these factors, e.g., by evaluation for insurance purposes, may partially remove these effects, but can never eliminate all of them, and risks introducing uncontrolled bias into the process.

**Relatively Low Prevalence of Confounding Risks.** Slesnick & Thornton (2008p. 191) argued that “... even though life expectancy data include individuals with various medical problems, such people are likely to constitute only a very tiny percentage of the group sampled”. For example, in 2016 in the general U.S. population the prevalence of risk factors for mortality included 7% and 3% for coronary heart disease and MI, 11% for diabetes, 12% for total cholesterol > 240 mg/dl, and 15% for chronic kidney disease (ADA, 2020; Virani et al., 2020, Table 27-1, p. e590).<sup>10</sup> Even though comorbidity is common, the overall population prevalence is also relatively low; Dugoff et al. (2014) reported that the percentage of the Medicare population they studied with multiple chronic disorders ranged from 11.9% (zero) to 2.3%

(10+ conditions). Thus, although the standard life tables include individuals in the population with most if not all conceivable health conditions and behaviors, the influence of any particular element or combination of factors on overall life expectancy is likely to be relatively small.

A few conditions were significantly much more common, including obesity at 31%, and high blood pressure at 46% (Virani et al., 2020, Table 27-1, p. e590), although even these high-prevalence conditions were still in the minority in the general U.S. population. Complex mathematical models have been proposed to correct for such high background mortality effects (Touraine et al., 2019; Wang et al., 2013), especially if one or more of those conditions cross-contributed to other causes of death relevant to the index condition (e.g., cigarette smoking and obesity influencing mortality both from cancer and cardiovascular disease). However, in these cases the life expectancy analyst may simply want to apply a correction factor to the mortality index to account for potential significant effects,<sup>11</sup> with a clear explanation of the rationale and process for the trier of fact.

**Nonrepresentation by Standard (Unmodified) Tables.** Vachon & Sestier (2013) contended that use of the general population life expectancy was appropriate when differentiating factors about the individual were not known, but that assumption that the “general population contains everyone and is therefore an appropriate estimate for anyone” (p. 541) was not valid when individual information was available.

As an illustration, the mean annual income in the United States is a general average. If one wants to estimate the expected annual income of a Fortune 500 chief executive officer [CEO], the general U.S. mean is inappropriate, even if all CEOs are included in the larger average. The fact that they are CEOs makes them a distinct subgroup with its own average” (p. 541).

Kush (2010p. 7) succinctly described this as the “common but erroneous argument that the average includes the extreme and therefore the average should stand for the extreme”.

**Built-In Correction.** Currently used modification methods often already account for mortality included in general populations, as reflected in the standard life table. The commonly used mortality indices, EDR and MR (see Part Two, Step Three below) both involve comparison of mortality rates in a population of subjects with a risk factor or disease to a population of subjects without that condition, termed “observed” (those with the factor) and “expected” (those without the factor). “Expected” populations may be chosen from the general population, group life or selected insureds, Medicare, Medicaid, or some other representative reference group, corresponding as closely as possible to the “observed” population except for the factor of interest (usually matched at least by age, gender, and race, and often by geographic location and/or time period). If the “observed” and “expect-

ed” populations are carefully or even reasonably matched, and unless there is some unrecognized or recognized source of selection bias),<sup>12</sup> the distributions of other conditions influencing mortality should be similar in the two populations, controlling for the prevalence of the conditions.<sup>13</sup> As noted by the AAIM (2015): “The purpose of the initial comparison population group is to act as an expected ‘mortality filter’, removing the expected mortality experience of the group except for the impairment in question” (p. 25). Thus, the EDR and MR indicate the effect of the risk factor, disorder, or condition in question over and above its effect in a general population – if the general population (used to generate the life table) and the “expected” population (used to generate the mortality metrics EDR and MR) are comparable, the EDR or MR should represent a true effect of the condition, independent of other mortality influences.

#### ***Extended Uses of the Life Table***

In addition to life expectancy, the (modified) life table allows determination of other information of interest to the actuary, life care planner, or life expectancy analyst, including age-specific mortality and survival probabilities and median survival time. For example, if an individual’s life expectancy is less than their median survival time (the age at which 50,000 people remain alive, given a radix of 100,000 persons), they have a greater than 50% chance (i.e., more likely than not) of outliving a life care plan based on the life expectancy. Day et al. (2015b) provided a short but thoughtful analysis of how the life care planner should use mortality, survival, and life expectancy information from the life table, including questions of how far to extend the life care plan beyond the life expectancy in cases in which the probability of survival (and thus the life expectancy) is both high (many years) and low (a few years). These authors suggested that “These questions have not been adequately addressed in the literature” (p. 264) and recognized the need for exploration by multidisciplinary professionals.

#### ***Adjustment for Secular Trend in Mortality<sup>14</sup>***

General population mortality rates tend to decrease with time (Virani et al., 2020, Table 27-1, p. e590), and both demographic experts (Canudas-Romo et al., 2016) and attorneys (Kessler, 2020) have advocated routine adjustment to account for this phenomenon during the life expectancy determination process. The reasons for this overall trend in death rates are complex but include decline in the mortality from infectious (particularly influenza and pneumonia) and cardiovascular disease, as well as in infant mortality (especially among those of low birth weight), and improved nutrition and public health measures such as decrease in smoking, affecting both cancer and heart disease (Cutler & Meara, 2001). Improvements due to advances in health care are

subject to disagreement (see below) but may be attributable to general improvements such as increased organ transplantation, genetic substitution, and enhanced anticancer treatments (Ryan & Harbin, 2006).

The general practice in the United Kingdom and Australia is to use projected (cohort) life tables incorporating assumptions regarding future life expectancy improvements (e.g., the Ogden tables; Government Actuary's Department, 2020). In contrast, the United States typically relies upon recent or current (period) tables (DeVivo et al., 2018), although some life expectancy experts in the United States do correct for observed trends in mortality over time in specific conditions (e.g., Shavelle, 2016). Ayuso et al. (2018) demonstrated that period life tables based on current death rates may underestimate life expectancy as much as 30% when compared to cohort tables based on a combination of actual past and forecasted future death rates; similar studies found underestimates of 15% in the UK (ONS, 2013) and 7% by the U.S. Social Security Administration (Bell & Miller, 2005). Booth & Tickle (2008) submitted that experts in both the United States and United Kingdom tend to conservatively underestimate mortality decline and described an "assumption drag" in which expectations tend to lag rather than lead actual experience (p. 9).

Nevertheless, attempts to anticipate future changes in mortality and life expectancy are complex and cannot be done with total accuracy. Some of the sources of uncertainty include:

- Human life expectancy increased by an average of about 2.5 years per decade since the mid-19th century (Oeppen & Vaupel, 2002), and between 1900 and 2017, average life expectancy in the U.S. general population (for both genders and all races) increased from 47.3 yrs to 78.6 yrs, or an overall average of 0.27 yrs/yr; this overall trend may continue (Waldron, 2005). However, the rate of increase since 1945 has been slower, changing from 65.9 yrs to 78.6 yrs, or an overall average of 0.18 yrs/yr (Bastian et al., 2019), and a clinical and demographic study by Canudas-Romo et al. (2016) anticipated an increase in life expectancy of only about 1.0 year per decade going forward between 2010 and 2040.
- Research has shown conflicting effects of trends in population health. For example, Stewart et al. (2009) cautioned that in 18-year-old people in the United States negative effects of the increasing prevalence of obesity may outweigh the positive effects of decreased smoking by 2020.
- Jones (2010) argued that "... because the expected future life expectancy increases are concentrated in the older age group, the impact is not likely to be significant" (p. 206).
- The ONS (2013) study cited above cautioned that the im-

provement in mortality experience assumed by the model (50 years into the future) became less reliable with time, and Day et al. (2015) further warned that "it is uncertain whether historical trends in reduced mortality rates will continue for another 50 or 100 years" (p. 1108).

- Vachon (2013) posited three reasons "to forego correcting or adjusting for future advances and expected decreases in mortality", stating: First, it is not certain that such advances will materialize. This may seem pessimistic, but the steady advances in survival could be seriously hampered by either catastrophic events or prolonged behavioral or environmental changes that temporarily or permanently change the mortality landscape.<sup>15</sup> The rise in the prevalence of obesity, for example, could very well prove to be detrimental to future life spans. The second reason has to do with uncertainty. Even if a decline in future mortality rates was certain, the precise pace or extent of decline may remain uncertain. What values would then be used, given that opinions offered as expertise cannot be based on pure speculation? The third reason is much more pragmatic, and some would argue much more compelling. The rules of evidence dictate to some extent the parameters. An expert cannot in all cases simply introduce conjecture about the future state of the world. Much like an economist may be constrained to use the current prevailing interest rate, the life expectancy expert may be limited to employing current, measurable, observable, and somewhat tangible mortality rates. The rules need not be this way, of course, and some jurisdictions, such as in the UK, mandate the use of projected rates—i.e., rates that vary over time in the future. But it is beyond the scope of the expertise to try to change the rules of the court and compliance is indicated. (p. 548ff.)
  - In research demonstrating a linear decrease in life expectancy with increasing comorbidity, Dugoff et al. (2014) speculated that "... a consequence of living with more conditions is higher mortality and thus shorter life expectancy. If the burden of chronic disease in the elderly continues to increase, it is possible that life expectancy improvements in older ages could slow" (pp. 691-692).
  - DeVivo et al. (2018) provided data demonstrating that correcting for anticipated life expectancy improvements overestimated life expectancy in individuals with SCI (even when only the single condition was considered); these authors cautioned that the approach was "not exact", and that investigators should describe any adjustments to expected future life expectancy and present both unadjusted and adjusted findings.
- The life expectancy analyst and life care planner should exercise caution in attempting to anticipate and incorporate both positive developments in health care and salubrious

effects of life care plans in life expectancy determination. Meagher (2019) reviewed many of the variables contributing to the difficulty in anticipating future mortality and thus life expectancy rates, opining in the end that this effort required “modesty and a crystal ball”. The best approach may be for the analyst or planner to perform life expectancy determination both with and without correction and present both options, with clear explanation, to the trier of fact.

### **Overview of the Life Expectancy Determination Process**

There are five major sequential tasks, and corresponding outcomes, involved in life expectancy determination for an individual using the modified life table approach:

1. Step One: Selection of the appropriate standard population life table to determine the baseline age-, gender-, and race-specific life expectancy and provide the substrate for the adjustment.
2. Step Two: Identification of relevant medical disorders and other characteristics that may influence the individual’s mortality. This involves review of the available information to identify medical diagnoses and other influences and selection of those with significant mortality effects, resulting in a list of all potentially relevant conditions.
3. Step Three: Quantification of the comparative mortality (increased or decreased) for those conditions. This entails selection of the best and most appropriate (as defined below) available research studies relevant to the individual and determination of usable indices (EDRs and/or MRs). The result should be a catalog of mortality indices associated with the conditions identified in Step Two with supporting literature.
4. Step Four: Combination of the baseline population mortality rates and the mortality information particular to the individual (from Step Three) to obtain a new schedule of modified mortality rates. The outcome of this step should be a schedule or table of combined age-specific comparative mortality figures ready to be incorporated into a modified life table.<sup>16</sup>
5. Step Five: Application of that mortality information to the selected standard life table to generate a new life table and consequent life expectancy.

### **Who Should Determine Life Expectancy?**

Shavelle & Strauss (2013) opined that accurate, evidence-based formulation of summary measures of survival (i.e., life expectancy determination) required: a) knowledge of the subject’s medical history and current status, as well as functional capability; b) an understanding of the basics of life expectancy [and] the factors related to survival; and c) ability to use the available scientific literature (p. 27). Vachon (2019) advised that “There is no clear bright-line rule that

identifies the threshold one needs to cross to qualify as an expert” in life expectancy determination (p. 612). He extensively discussed the requirements for competence in life expectancy determination, which he felt included academic work or formal training in actuarial science, demography, biostatistics, statistics, or epidemiology; knowledge of accepted life expectancy concepts and facility in using and applying them; and reliance upon known facts about the subject of the analysis and scientific knowledge about the conditions affecting them. Day & Reynolds (2015), paraphrasing Rothman and Greenland, concluded that “Today ... life expectancy experts have achieved a separate identity. Being either a physician or a statistician, or even both simultaneously, is neither a necessary nor a sufficient qualification for being an expert on life expectancy. What is necessary is an understanding of the principles of the scientific methods for calculating life expectancy and the experience to apply them.”

Thus, both clinical knowledge (and experience) and familiarity with the life expectancy determination process are necessary, especially when the subject of the analysis has multiple conditions likely interacting in complex ways to influence their longevity. Some attorneys (Kessler, personal communication, August 14, 2020) believe that at least three separate experts are required: a) a clinician, to assess the medical aspects of the claim; b) an insurance medicine specialist, to determine the appropriate mortality influences; and c) an actuarial consultant, to apply the mortality influences on the individual of interest. Actuaries and statistical experts will not have a clinical background, although they can learn important aspects of clinical medicine through exposure and experience. As discussed above under Anecdotal or Experiential Opinions by Clinicians, no matter how well clinically trained or experienced, physicians are not in and of themselves qualified to perform life expectancy determination without specialized knowledge, including education in insurance medicine or extensive study of the life expectancy determination literature; the necessary combination of skills is possessed by relatively few individuals.

### **Physical Examination of the Subject**

A question that arises at this point is whether it is necessary for the life expectancy analyst to physically examine the individual in question. Sestier (2010, Section 6, p. 3 of 5) addressed this concern in life expectancy determination as follows:

... it was not necessary for [the life expectancy analyst] to have examined [the claimant], in order to render an accurate analysis of his life expectancy. In fact, in the insurance industry, such calculations are made after reviewing the applicant’s medical records. In cases that are subject to litigation, where the issue is, for example, the decedent’s life expectancy if an accident had not



occurred, nearly all the calculations are made after a subject's death. Even when a subject is alive, an examination of the subject occurs only in a minority of cases; the immense majority of such reports are established after a careful review of the complete medical brief.

However, life care planning is a more comprehensive undertaking, and in-person evaluation of individuals subject to this process, which may include physical examination, is both highly desirable (Weed, 2019) and recommended in some form by the AANLCP (2020), AAPLCP (2020), IARP (2007 and 2015), and ICHCC (2020). For example, the Standards of Practice of the American Academy of Physician Life Care Planners (2020) stated: "Whenever possible, and/or practicable, physician life care planners perform personal interviews and examinations of subjects to: collect information which support the objective findings included in the medical records and other relevant documents; and to discover objective finding which may not be identified in the medical records and/or other relevant documents."

#### **Step One: Selection of the Baseline Life Table**

There are several considerations regarding selection of the appropriate baseline population life table for adjustment:

- As noted above, the U.S. NCHS publishes life tables based on gender and race, stratified by age in one-year intervals.<sup>17</sup> In general, the table most specifically matching the demographic characteristics of the individual in question should be used.
- The U.S. Census Bureau uses four categories of race (White, Black, American Indian and Alaska Native, and Asian and Pacific Islander) and two ethnic categories (Hispanic and non-Hispanic). These divisions are not scientific in nature but are mandated by the Office of Management and Budget to promote consistency in federal record keeping and data presentation (U.S. Census Bureau, 2020).
- The U.S. NCHS tables are readily available in spreadsheet form (Arias & Xu, 2019) to allow adjustment computations, and life expectancies for fractional ages can easily be obtained (Tucek, 2009). However, the user must manually enter formulas to allow recalculation with varying EDRs or MRs.
- When dealing with historical (noncurrent) incidents, it may be more appropriate to use the most contemporaneous table corresponding to that event rather than the most recently available version. If the individual for whom the life expectancy is being determined is deceased, the historical table most concurrent with the date of interest (e.g., date of tort or death) is used. If that person is still living, the most recent available general population life table is used. If the individual has lived in a country other than the U.S. for a sufficiently long interval that the

person's mortality would more accurately be reflected by the demographics of the adopted country, then a base life table from that country should be used (Dolan, 2019).

- Some demographic experts (Canudas-Romo et al., 2016) and attorneys (Kessler, 2020) have advocated adjustment to the life table to account for the overall decrease in general population mortality rates with time. This issue is discussed under Influence of Medical Care, Including the Life Care Plan below.

#### **Step Two: Identification of Relevant Conditions Influencing Life Expectancy**

This step includes comprehensive review and evaluation of available medical and other records and identification of all diagnoses and other conditions and factors potentially having a significant effect, positive or negative, on the individual's life expectancy. This should be completed by the life care planner in the course of initial and ongoing assessment for healthcare needs (Johnson & Weed, 2013), and may give the planner some additional insight into relevant life expectancy considerations for the individual.

#### **Multiplicity of Influences on Life Expectancy**

As previously noted, an individual's ultimate lifespan is a function of many biological and psychosocial influences, and the eventual outcome will be an integral of all of these factors, in some cases with unforeseeable events included (e.g., accidental injury or premature death). This multiplicity of determinants complicates life expectancy determination in a number of ways.

**Comorbidity is Common.** The simultaneous presence of two or more medical or other conditions in the same individual is the rule, rather than the exception, and is increasingly common in aging persons (Marengoni et al., 2011; Ward & Schiller, 2013). For example, Salive (2013) found multimorbidity in 62% of persons aged 65-74 yrs and 82% of those at least 85 yrs old. Singer (2005a) acknowledged this effect, noting that "... in my experience, there are multiple risk factors in most of these cases, with an average of 4 or 5 per case, and a range of 1-11 factors" (p. 49). Dugoff et al. (2014) reported that life expectancy decreased by an average of 1.8 yrs for each of 21 comorbidities in the Medicare population (age 67 yrs) that they studied.

**What Conditions to Include?** Comorbidity introduces the problem of how many elements to include in a life expectancy determination, since at present it is not practical to incorporate all potentially relevant factors. Even with a finite number of identifiable conditions affecting mortality in an individual, the analyst must decide the relative influence and significance of each element, as well as the degree of impact, e.g., severity, of each condition (Krause & Saunders, 2013). (This issue is addressed in greater detail under Step Four,



Number and Significance of Conditions to Consider below.)

**Interactive Effects.** Multimorbidity introduces interactive effects among elements which may be difficult to define. In some cases, the life expectancy effects of multiple disorders will be essentially independent, e.g., asthma, cancer, and seizure disorder. However, for many combinations of conditions, e.g., diabetes, hypertension, and obesity, there will be significant overlap in the life expectancy effects. For example, Singer & Milano (2007) analyzed data from the Multiple Medical Impairment Study (MMIS; SOA, 1998) and found that approximately one-third of combined disorders (two “impairments” plus elevated blood pressure) each showed less than additive (sub-additive), additive, and synergistic (supra-additive) effects. (This problem is discussed in more detail under Step Four, Duplication of Mortality Effects and Adjustment for Overlap of Conditions below.)

#### **Identify Relevant Medical, Behavioral Health, and Nonmedical Factors**

Krause & Saunders (2010) presented a comprehensive theoretical risk model of mortality which included biographic and injury-related, psychological, environmental, behavioral (both protective and risk), and health and secondary condition-related components, as well as a further breakdown of economic influences. Krause et al. (2013) refined the original model, adding a “stress-coping” component. In general, only elements for which there is credible evidence of substantial effect on life expectancy should be included.<sup>18</sup> Significant contributors may include the following general areas of influence:

**Biographical or Demographic.** This category encompasses factors such as gender, geographic location, and ethnicity and race (Koenig et al., 2008; Weinstein & Pillai, 2016). Age is obviously a variable in life expectancy, although the effects vary with other demographic factors. As Krause & Saunders (2010) cautioned, these elements “represent a starting point rather than a concluding point” (p. 26). Socio-economic influences are considered in Influence of Socioeconomic Factors (SEFs) below.

**Medical or “Physiological” (Including Injury).** In rare cases family history may exert an appreciable effect on longevity (Newman & Murabito, 2013). However, although family history of significant medical disorders may be a risk factor for illness in the index individual, and/or considered in underwriting for insurance (Woodman, 2006), it is usually both too remotely removed and uncertain in nature (i.e., poorly quantifiable) to be considered in life expectancy determination.

The primary drivers of increased mortality will be chronic medical conditions, most of which exert a negative influence on life expectancy (Foreman et al., 2018) and scientific literature contains thousands of research articles providing quan-

titative information on these effects. In general, as societies develop, chronic and degenerative disorders (e.g., circulatory and metabolic conditions such as hypertension and diabetes, and osteoarthritis) replace infectious disease as the major cause of disability and mortality (the theory of epidemiologic transition; Omran, 1971). This category will include objective effects of trauma, such as level of damage and American Spinal Injury Association (ASIA) grade in spinal cord injury (SCI; Strauss et al., 2006). The individual may also develop secondary conditions which add to the overall burden of disease in the course or as a consequence of primary illness or injury (see Development of Secondary and Subsequent Conditions below).

**Behavioral Health or “Psychological”.** This category comprises a wide variety of behavioral and psychological elements. Negative influences on mortality include a) mental health conditions such as mood disorders (with or without suicidality), especially depression (Cuijpers et al., 2014) and other forms of psychopathology, including psychological distress (Russ et al., 2012); b) substance abuse and dependence, including alcohol, illicit drugs, licit drugs with significant potential side effects, particularly opioids, and tobacco (e.g., Gleib & Preston, 2020);<sup>19</sup> and c) generally maladaptive personality traits such as neuroticism, or persistent negativism (Jokela et al., 2014; Krause et al., 2009). Conversely, multiple studies have demonstrated an overall positive effect of “lifestyle” practices such as “proper” diet, regular exercise, avoidance of excess alcohol use and cigarette smoking, and a high level of socialization (Colpani et al., 2018; Loeff & Walach, 2012; Yanping et al., 2018). Personal characteristics such as resilience (i.e., the ability to adapt to adversity; Lamond et al., 2009) and overall positive affect (Martin-Maria et al., 2016) and attitude (Elliott et al., 2013; Surtees et al., 2006), may exert a beneficial effect; other potential protective factors for life expectancy include the presence of strong interpersonal relationships, social participation, and emotional support (Ding et al., 2015; Krokstad et al., 2017); spiritual beliefs (Lucchetti et al., 2011); degree of life satisfaction and “happiness”; and marital status (Robards et al., 2012).

**Nonmedical.** This category includes activities, habits, and practices (lifestyle behaviors) which may pose an increased risk of mortality. Examples of negative influence include engaging in dangerous sports such as hang gliding, motorcycle riding (Beck et al., 2007), motor racing, and mountaineering (Woodman, 2006), and other motor-vehicle related risks such as impaired driving (Impinen et al., 2010) and failure to use auto seat restraints (Mbarga et al., 2018). A history of criminal offenses is associated with increased mortality, even after controlling for substance abuse (Skardhamar & Stirbekk, 2013). Occupational elements which are rarely considered but which may be relevant are engaging in hazardous occupations,<sup>20</sup> and, particularly, the deleterious effect of unem-

ployment (Clemens et al., 2015).

### ***Development of Secondary and Subsequent Conditions***

As noted above, individuals may develop early or late secondary and associated conditions in the course or as a consequence of a pre-existing illness or new injury which increase mortality beyond that of the baseline condition (Jensen et al., 2012; Rimmer et al., 2011). For example, persons with diabetes may experience diabetic nephropathy, neuropathy, or retinopathy, and may need to undergo extremity amputation, and those with poorly controlled hypertension may experience atherosclerotic disease, CVA, or dementia, heart failure, and nephropathy. More general disorders include chronic pain, especially if widespread (Da Silva et al., 2018; Macfarlane et al., 2017), overweight and obesity, pressure ulcers, sleep disturbance, and compromise of the ability to engage in physical activity and exercise and/or nutritional status (especially in the individual with multiple fractures or significant wounds, such as soft tissue injuries or burns). Other characteristics such as feeding and walking ability and bladder and bowel continence status, and thus need for external care, may affect life expectancy after severe trauma such as SCI or TBI (Shavelle & Delaney, 2010). Rimmer (2011) outlined a detailed model for identifying and differentiating pre-existing and associated conditions, prevalent comorbidities, and treatment complications in physical, psychological, and social realms. Jensen et al. (2012) extensively discussed secondary disorders in persons aging with SCI (as a representative population for physical disability); common examples which may influence life expectancy included chronic pain, mood disturbances such as depression, both non-urinary and urinary tract infections (including consequent septicemia), obesity, pressure sores, and venous thromboembolism (VTE; Table 1, p. 375). Access to and quality of health care (see below) and degree of compliance with medical treatment (Currie et al., 2012; Guihan & Bombardier, 2012) may be important factors.

In general, the analyst performing life expectancy determination (and life care planning) should rely upon the most recent available information on actual medical and behavioral health disorders, medications, and personal habits and characteristics for the person, including documented and relevant secondary conditions. Deutsch (2020) pointed out that life care plans generally should not include effects (costs) of potential medical complications; however, inclusion or exclusion of potential influences on life expectancy will depend upon the specific circumstances, including the natural history of disease, even if they are reasonably foreseeable (e.g., limb amputation in diabetes or peripheral vascular disease) or even highly likely (e.g., need for heart and/or lung transplant). If necessary, the life care planner's reasoning should be clearly explained for the trier of fact, including the basis

for inclusion, likelihood of occurrence, and potential effects on the life expectancy determination from the life care planning perspective.

### ***Potential Sources of Information***

Ideally, the analyst should review and consider all sources of information about an individual, including medical, civil, legal, and other reports, depending upon availability. Medical records may comprise hospital charts, including nursing documentation; clinician office progress notes, including those from allied health practitioners such as occupational, physical, and speech language therapists, and nutritionists and social workers; pharmacy records; and specialist consultations, Independent Medical Evaluations (IMEs), and similar assessments (Forensis Group, 2013). Case summaries by attorneys, e.g., in WC cases, are often useful sources of information, either in and of themselves, or as a "flag" for medical information (or other considerations) that the analyst may have otherwise overlooked. Investigative consumer (inspection) reports and civil records may provide information on high-risk behaviors such as motor vehicle violations. Legal records may document criminal behavior and provide an indication of credibility of an individual, although this is a controversial issue (Laudan & Allen, 2013). If the examiner is skilled and experienced at IME, they may elect to perform a formal evaluation specifically for the life expectancy determination (Vachon & Sestier, 2013). The analyst should attempt to cross-relate and corroborate these information sources whenever possible.

The evaluator will likely need to exert some judgment about the quality and applicability of the available information. Subjective or self-reported material may not be reliable (Barth, 2009), and should be corroborated whenever possible. Diagnostic error is a significant problem in both general medicine (National Academies of Sciences, 2015a) and compensation systems (Hendler, 2013). Medical conditions are sometimes inaccurately diagnosed, and a medical disorder accepted under a claim may not be valid, or exert an actual, appreciable effect on life expectancy; this is true of both medical and behavioral health (BH) diagnoses, including conditions such as complex regional pain syndrome (Barth & Talmage, 2015) and functional illnesses such as somatic symptom disorders (Ross, 2015).

As noted above, physical examination findings may or may not contribute to life expectancy determination. Shavelle & Delaney (2010) listed many physical examination findings that may be important in the pediatric population; but results in adults are probably less useful, unless they are manifestations of a significant but previously unsuspected disease process.

Life expectancy determination after a specific illness or injury event should usually be deferred until all resultant pri-

mary and secondary conditions are mature. Some disorders have a disproportionately high early mortality (e.g., MI and severe trauma), and others require a long period of stabilization, e.g., up to two years for maximum recovery from SCI and TBI (Thomas & Barnes, 2010). A basic assumption of life table modification methodology is that once the risk of premature death (as measured by either EDR or MR) has reached a reasonably constant level, time since onset of the contributing condition(s) can be ignored (Anderson, 2002, Endnote 3.5, p. 122).

### ***Relative Dearth of Positive Influences on Mortality in Life Expectancy Determination***

Many potentially positive and/or protective effects on mortality (as outlined under Behavioral Health or “Psychological” above) are rarely considered in current life expectancy determination practice, for several reasons:

- Vaillant & Mukamal (2001) specified four difficulties with research into the relevance of protective (and risk) factors in aging and mortality, including a) selective advocacy of one factor at the expense of others, with consequent inability to separate association and cause; b) confusion about causal direction, e.g., social support as an independent or dependent variable; c) the change in the importance of relevant predictor variables over time; and d) inability to control for inaccurate self-report.
- A basic principle in life expectancy determination is use of objective information and evidence-based research. However, it is difficult to operationally define and apply terms like “proper” (diet) and “regular” (exercise), and these factors are difficult to quantify in a meaningful or credible way, outside of controlled conditions in a research setting.
- The mechanisms of action of many of these elements are not well established, and there may be complex interactive effects, often overlapping with medical or physiologic factors, that confound results and limit both conclusions and generalizability.<sup>21</sup>
- Due to methodological challenges, research may be of lower quality, especially in terms of controlling for confounding factors. Effects may be narrowly defined, e.g., results of self-reported screening instruments or symptom inventories, which may be subject to validity (bias) and generalizability problems. Effect sizes may also be low and compromised by practical limitations (e.g., Kim et al., 2016).
- Even if credible data on mortality effects for a given positive attribute is available at a group or population level, trustworthy and usable information may not be obtainable for a given individual undergoing life expectancy determination. Many of these influences are impossible to reliably quantify in a given person, often relying upon

self-reported or subjective test results or proxy measures, as opposed to negative risk factors, which can often be defined by objective physiologic measurements and diagnostic criteria. For example, a diagnosis of diabetes or hypertension is relatively straightforward by established standards, but it is much more difficult to convincingly establish a person’s positive attitude, resilience, or marital or work satisfaction, and usually not feasible to establish these in the same way clinically in an individual as in a research study.

- Positive behavioral health elements such as coping skills and resilience may be labile and take years to stabilize after a traumatic event (Krause & Saunders 2010).
- DeVivo et al. (2018) observed that the assumption that positive effects on life expectancy may not be the same in the general population as in persons with an illness (in this case SCI).

Research suggests that some currently positive (and currently underused) influences on mortality may be substantial and important, especially in combination, but at this point we are limited in our ability to rationally include these effects into life expectancy analysis, for all of the reasons cited above. Further study and development will likely clarify the roles of some factors, especially BH influences. The best approach at present may be to include positive influences when they are substantial and verifiable, e.g., when an individual engages in a robust exercise program which can be corroborated.

### ***Relationship of Functional Capability and Disability to Mortality and Life Expectancy***

There is strong evidence that increased mortality in certain conditions is determined by the consequent functional capability limitation, i.e., disability,<sup>22</sup> rather than intrinsic characteristics of the condition (Landi et al., 2010; Marengoni et al., 2009; St. John et al., 2014):

- Disability scores increase with the number of comorbid conditions (Haagsma et al., 2011; Verbrugge et al., 1989), and mortality risk increases with number and severity of functional limitations (Hutton & Pharoah, 2002; Hemming et al., 2005).
- Self-reported functional impairment is strongly linked to subsequent mortality (Reuben et al., 1992; Bernard et al. 1997); this effect is especially evident in the elderly (Cutler et al., 2013; Lubitz et al., 2003), and compression of both morbidity and disability (i.e., delay in both to a shorter period in the final years of life) has been related to longevity (Terry et al., 2008).
- Lee et al. (2006) found that a prognostic indicator incorporating behavioral, clinical, demographic, and functional measures predicted low, intermediate, and high mortality at four years.

- Differential mortality by ASIA impairment level or Frankel grade in severe SCI or TBI and neurological disorders such as cerebral palsy clearly demonstrates the effects of increasing functional capability limitation (Cao et al., 2013; Middleton et al., 2012; Shavelle & Delaney, 2010).<sup>23</sup> Functional decline, independent of comorbidities, has been shown to decrease longevity in cerebral palsy (Strauss et al., 2004) and SCI (Liem et al., 2004).
- The Barthel Index of function has been found to be an independent risk factor in hospitalized geriatric patients (Ryg et al., 2018),<sup>24</sup> after acute exacerbation of chronic obstructive pulmonary disease or COPD (Lahosa et al., 2019), and after infrainguinal bypass surgery for critical limb ischemia (Kodama et al., 2017), and to predict five-year survival after cardiac surgery (Marcassa et al., 2016).

Specific impairment and disability factors which affect mortality include immobility, cognitive and intellectual deficits, swallowing problems and consequent need for tube feeding, and bladder or bowel incontinence (Thomas & Barnes, 2010). Shavelle & Strauss (2013) listed a number of potential physiologic mechanisms for this mortality effect in sedentary or immobile individuals, including decreased cardiopulmonary function and reserves; increased risk of VTE, e.g., deep venous thrombosis and pulmonary embolism (PE); bone demineralization with consequent effects on musculoskeletal (e.g., osteoporosis) and urinary systems; skin breakdown, increasing the risk of infection; insulin resistance, and adverse effects on the immune system. Friedman et al. (2015) suggested that multimorbidity was associated with both systemic markers of inflammation and functional limitation, and that inflammation at least partially mediated the link between comorbidity and function (see also Krause & Saunders, 2013; Krause et al., 2013).

#### ***Influence of Socioeconomic Factors (SEFs)***

There is an extraordinarily complex relationship among certain SEFs and longevity. The most prominent influence on mortality is per capita income (National Academy of Sciences, 2015b), which is in turn largely a function of education level (Ketenci & Murthy, 2018; Lantz et al., 2010), but many other related elements also vary with SEFs, including health behaviors and lifestyle (Stringhini et al., 2010), occupation, and insurance status (Rask et al., 2009). This interaction includes considerations of ethnicity and race, as well as access to both prenatal and lifetime medical care (although Bilal et al. [2019] found a strong association between SEFs and life expectancy even in an environment of universal, high quality healthcare coverage), nutrition, lifestyle factors (e.g., substance abuse), and rural vs. urban living environment (Cutler et al., 2006). Seeman et al. (2008) reported that education and income effects were independently and negatively asso-

ciated with specific cumulative biological risk factors such as cardiovascular, metabolic, and inflammatory influences. Societal economic and financial fluctuations, including crises, may affect mortality (Bohk & Rau, 2015).

Krause and colleagues conducted several studies indicating that SEFs may also affect mortality in persons with specific medical conditions, particularly SCI. Krause (2002) detected a significant difference in mortality between spinal cord injury patients in the lowest and highest income quartiles in the study. Krause et al. (2004) determined that inclusion of family income and WC insurance coverage in SCI patients increased life expectancy from 68% to 81% of normal.<sup>25</sup> Krause & Saunders (2010) again found a strong relationship between preinjury income and later mortality (also providing a thoughtful analysis of the limitations of the current state of knowledge), and Krause et al. (2011) reported a stepped relation between low, middle, and high household income and mortality after SCI.

#### ***Influence of Medical Care, Including the Life Care Plan***

Two major medical management considerations in life expectancy determination are the impact of overall care quality (especially presumed improvements obtained under a life care plan) and the anticipated effects of future advances in medical science and technology.

**Increased care quality.** Overall, there is a significant relationship between societal health care expenditure and life expectancy at birth (Jaba et al., 2014), and insured patients may have lower mortality than those who are uninsured (Woolhandler & Himmelstein, 2017). However, as outlined in the previous section, environmental influences such as educational level, employment, and income (Rogot et al., 1992) and socioeconomic status later in life (Lin et al., 2003) are important supervening determinants of mortality in the U. S. general population. In addition, many confounding factors complicate the relationship between health care spending and life expectancy, and health care expenditure does not correlate directly with quality of care or with life expectancy, especially in the United States (Burke & Ryan, 2014; Roser, 2017).

Some commentators have asserted that life expectancy is increased by enhanced quality of care obtained under legal agreements for people with significant illness and injury. For example, Plioplys (2012) stated that “If optimal medical care can be assured for these [cerebral palsy] patients, their life expectancy, compared to published results, would be increased by an additional 10 years of age” (p. 31). Krause & Saunders (2013) maintained that “... it is important to consider the favorable economic situation generally produced by the life care plan and litigation in developing the life expectancy estimate” (p. 56). However, there are still differences of opinion over whether life expectancy reduced



by primary and comorbid conditions is counterbalanced by higher quality of care, for example under a life care plan, and convincing evidence is still lacking:

- The Dartmouth Atlas Project (<https://www.dartmouthatlas.org/>) has conducted research on Medicare healthcare spending and utilization since 1999; among their findings were the observations that more medical intervention, beyond what is reasonable and necessary, does not produce better outcomes (or significantly prolong lifespan) for either populations or individuals (e.g., Fisher & Wennberg, 2003; Fisher et al., 2003).
- Shavelle et al. (2007) discussed this problem in some detail, opining that “quality of care” was a vague term encompassing considerations of caregiver accessibility and expertise (which was not necessarily a function of formal qualification), as well as quantity of care and supportive equipment provided. These authors pointed out that differences between “normal, standard care available in most Western societies” and “care expected given that the patient has a carefully prepared and well-funded life care plan” may be much less than expected and maintained that the most important determinant of life expectancy was severity of disability rather than care quality per se (p. 255).
- Bonfiglio (2010) concluded that “Unfortunately, there is no medical literature for individuals with catastrophic injuries that projects life expectancy *based on the level of care* [italics in original] that is typically outlined in a life care plan” (pp. 22-23).
- Kush et al. (2013) reported that “Currently we are aware of no evidence that receiving higher quality of care or a greater quantity of care and exceeding that which is already considered *reasonable and necessary* [italics in original], or that which is provided routinely in industrialized settings, carries any significant life expectancy benefit.” These authors went on to observe that “As of now, the question of whether there might be a type of care that actually will increase life expectancy is not fully understood, and evidence that one regimen might be better than another is quite sparse” (p. 45).
- Day et al. (2015b) noted that evidence that well-designed and -funded life care plans “must impact mortality risk, and thus life expectancy” is “scant or non-existent” (p. 264).
- Based on four U. S. studies with high literature citation rates, Kaplan et al. (2019) concluded that only 5-15% of variation in premature death was attributable to health care, with behavioral and social factors, including alcohol and drug abuse, tobacco use, and high-risk sexual behavior accounting for “substantially more of the variability in premature mortality than health care does” (p. 270).
- Vachon (2019) stated that “... the invocation of good care

as an absolute guard against hazards cannot be asserted without supporting, empirically substantial evidence”, which he qualified as “sufficient studies can be produced to show that a good care/bad care divide exists and yields statistically significant disparities in survival” (p. 615).

**Future Advances in Medical Care.** The potential effects of advances in medical care were addressed under Adjustment for Secular Trend in Mortality above. Although the overall historical trend is toward decreased mortality, at least partially attributable to improvement in health care, countervailing influences make reliance on this pattern in the future less assured. Deutsch (2020) pointed out that life care plans generally should not anticipate effects of potential future technology; in particular, it is difficult to make the quantitative predictions needed for incorporation into life expectancy determination (and life care planning) based on some presumed or projected rate of decline in mortality, and such adjustments should be made with caution, if at all.

#### **Summary of Considerations in Step Two**

In identifying the relevant mortality influences in an individual’s life expectancy determination, the analyst needs to consider several factors:

- The multiplicity of potential contributing factors, including demographic, biological and medical, behavioral health, and nonmedical elements, with both positive and negative effects on mortality, and their potential interactions
- Development of secondary and subsequent conditions
- The causal, qualitative, and quantitative consequences of functional capability and disability
- The complex influence of SEFs
- The impacts of medical care, both in terms of potentially increased care quality under a life care plan or settlement agreement, and general improvements in healthcare

#### **Step Three: Quantification of Increased Mortality for Relevant Conditions**

Valid life expectancy determination is critically dependent upon the availability and methodologic quality of research for any given condition and the appropriate application of the research outcome(s) to the estimation process (Krause & Sanders, 2013). This step involves selection of medical information to estimate increased or decreased mortality associated with the selected conditions from Step Two.

To be useful for life expectancy determination for an individual, a given mortality factor it should have two characteristics: First, its effects must be quantifiable, within a reasonable degree of medical certainty. This usually involves medical research under controlled conditions, logically determining acceptably accurate conclusions. Second, it must be significant to the individual of interest, both as generalizable



to the person from the research and demonstrated by objective documentation and/or credible subjective corroboration. The latter significance condition may be more difficult to achieve; for example, some influences may not be reliably measurable in a given individual, e.g., the effects of marriage (Robards et al., 2012) or “sedentary” lifestyle, and there may be obvious discrepancies between self-reported characteristics, e.g., alleged consumption of a “healthy” diet, and recorded information, e.g., body mass index (BMI).

### ***Comparative Mortality: Excess Death Rate (EDR) and Mortality Ratio (MR)***

Comparative mortality, or the relationship between observed (O) and expected (E) deaths attributable to a given condition,<sup>26</sup> is an essential factor in quantifying the life expectancy consequences of a given disorder and expressed as EDR and MR. Ingle (2000) and Pokorsky (1988) provided excellent overviews of the mathematics of comparative mortality determination.

#### **Observed and Expected Deaths or Mortality Rates.**

Observed deaths or rates are those actually occurring from a condition, often as determined by original clinical research or obtained from large databases such as the NCHS or NSCISC. Expected deaths or rates are those taking place in some defined comparison group, often the general population appropriately matched to the observed deaths group by age, gender, and geographic location.

**EDR and MR.** The EDR is the arithmetic difference between observed and expected deaths or death rates (i.e., O minus E), generally expressed per 1000 persons, and indicates the absolute increase in mortality, compared to the baseline for a population, attributable to a condition. EDR corresponds to attributable risk (Riffenburgh, 2012) as well as the “burden of disease” in conventional epidemiology, as well as flat extras in insurance underwriting, and is considered by some authors to be a more representative measure of the actual effect of a disorder than relative parameters such as MR (Streiner & Norman, 2012). The MR is the arithmetic ratio of observed to expected deaths or death rates (i.e., O divided by E), and indicates the relative mortality, compared to the population baseline, attributable to a condition. MR is one of the Bradford Hill (1965) criteria of causation (in the form of “strength of the association”) and corresponds to table ratings in insurance underwriting.

Both of these parameters are useful as mortality indices. EDRs are less commonly found in published research reports but are not difficult to calculate if appropriate information is available, and somewhat easier to combine when considering multiple conditions and mortality influences. MRs are much more commonly presented in epidemiologic research, in the form of hazard ratio (HR), or relative (RMR) or standardized (SMR) mortality ratios; HRs are often determined by

Cox proportional hazards analysis, which considers multiple mortality influences and attempts to control for confounding variables (Walters, 2009).

### ***Potential Sources of Information for Comparative Mortality Data***

The published medical literature now contains thousands of research articles which provide useful statistics on mortality from a wide variety of conditions affecting life expectancy; these studies furnish various levels of detail about the research, from detailed breakdowns of raw data (allowing calculation of mortality rates, and EDR or MR) to summary measures, most often HR and SMR. Gibbs et al. (2013) provided an excellent overview of use of medical and behavioral health resources in life care planning. The introductory paragraphs and/or discussion sections of research papers on specific medical research often contain useful literature overviews, and review articles may contain summaries of multiple morbidity parameters such as RMRs for various conditions of potential interest (e.g., Katz & Katz, 2017). Intercompany mortality studies and proprietary experience data from individual insurance companies provide much information (Woodman, 2006), but are usually not available to those outside of the organization.

### ***Study Quality and Appropriateness***

There is a hierarchy of quality in the design of medical research studies (Grimes & Schulz, 2002),<sup>27</sup> and in general research articles used in life expectancy determination should be of the highest quality obtainable to provide a valid basis for calculations and conclusions about life expectancy (Martinsson et al., 2016). However, less rigorous study methodology (e.g., case-control designs) may also provide useful information (Glasziou et al., 2004), and the analyst may need to balance features to achieve the best results (Vachon & Sestier, 2013). There is corresponding continuum of quality in clinical research, with wide variability in methods and control for potentially confounding variables; “Differences in demographic characteristics, sampling frames, construct definitions, and variables included in the analyses all contribute to such variability” (Reid 2013, p. 63). The life expectancy analyst must have a good working knowledge of these characteristics in order to judge the applicability and acceptability of a given research study for life expectancy determination, and guides to article selection are available (Kita, 1990; Singer & Kita, 1991).

A fundamental requirement in selection of research for life expectancy determination is matching of the characteristics of the claimant and subject population of the study as closely as possible, with the goal of eliminating or eliminating potentially confounding variables such as age, country of origin or geographic location, gender, race, SES, and comorbidity.

This is particularly true in life care planning; as Krause et al. (2013) observed: “life expectancy is a population parameter, whereas the life care plan is an individualized plan. This makes it essential that research used as the foundation of the life expectancy be matched to key characteristics of the individual to ensure the estimate reflects the appropriate population” (p. 51). Mismatching may introduce bias, or systematic error, which may favor one outcome over another (Pannucci & Wilkins, 2010). There is a trade-off between bias and sampling error (inaccuracy resulting from selection of subjects or data); Richards & Donaldson (2010) pointed out that “... it is usually the case that less biased estimates are obtained for more narrowly specified characteristics. However, sampling errors usually increase as populations are disaggregated into smaller and smaller subpopulations. Thus, there is usually a tradeoff: less bias but larger possible error” (p. 4).

Several authors have commented on the potential for faulty generalization in medical studies used for life expectancy determination. Anderson (2002) remarked that “The medical literature is almost entirely produced by the most developed countries, and at any given time death rates and life expectancies tend to be quite similar in those countries” (Endnote 5.11, p. 129). Rosen et al. (2013) stressed that life expectancy data is often collected at tertiary care and/or research centers “providing ‘cutting-edge’ care” and may not reflect the care available to a specific patient outside of such an environment (p. 4). Similarly, Kush (2013) concluded that “... in industrialized countries, poor care or complete lack of care seldom applies to persons who are the subjects of studies of survival published in peer-reviewed medical journals” (p. 42). Thus, the life expectancy analyst needs to consider the particular circumstances of the individual subject of the assessment in relation to any medical literature utilized. A basic consideration is the use of the general population life table for comparison with unselected groups of patients in medical studies (Ingle, 2000; Naslafkih & Sestier, 2001); conversely, it may be desirable for the life expectancy analyst to use a comparison group or table other than that for the general population if they suspect that significant selection bias (e.g., an insured or working population) is present (AAIM, 2015).

DeVivo et al. (2018) opined that when using general population mortality rates as expected numbers of deaths, the tables used should be concurrent with the period of exposure in the study population rather than the most recent available. On the other hand, it may be appropriate to use the most up-to-date information, to control for factors like improvement in mortality over time (for various reasons) and the most up-to-date medical care (AAIM, 2015). Thomas & Barnes (2010, p. 205), citing the research of Strauss et al. (2007) on longevity of cerebral palsy patients, noted a “pessimism bias” inherent in the use of retrospective data which cannot incorporate improved survival due to future progress in med-

icine and rehabilitation.

### ***Single vs. Multiple Sources of Mortality Information***

There will be variation in mortality indices (EDR, and HR or MR) and life expectancy estimates among published studies on the same condition, attributable to differences in case definition and construct definitions (e.g., of life expectancy itself), subject demographic characteristics, sampling time frames, and other variables included in the analysis (Reid, 2013). When multiple values for mortality parameters for any comorbidity are available in the medical literature, the analyst may want to consider combining the findings to reach some consensus among them (Vachon & Sestier, 2013; Vachon, 2019).<sup>28</sup> Shavelle et al. (2019) recommended weighting MRs by the number of subjects in each study to produce a composite value, but aggregating mortality results from different research studies may simply involve some judgment on the part of the analyst. When the evaluator encounters widely disparate figures, they should carefully study the methods used to determine those values to inform judgment about an appropriate final value. Vachon & Sestier (2013) proposed that “Reaching a conclusion as to the mortality associated with her condition will require that the expert exert some judgment in the face of multiple studies, not all of which will yield the same results. Ultimately, the opinion should discuss the findings from the literature review and acknowledge and justify any selection or judgment calls” (p. 545). These authors also noted that properly performed meta-analyses (which combine results of multiple research studies to derive an overall “consensus” conclusion) can be useful in life expectancy determination.

### ***Potential Errors in Step Three***

There are many possible pitfalls in selection of medical research publications to serve as a basis for life expectancy determination:

- Inclusion of information not relevant to the subject of the analysis, or subjective or non-quantifiable data
- Use of lower-quality medical studies, when results from research using higher quality methodology may be available
- Use of mortality indices from cohorts that are poorly matched or even dissimilar in demographic characteristics, e.g., pediatric vs. adult or uninsured vs. insured populations, or from different time intervals; the latter especially refers to outdated cohorts, resulting in comparison of older data (with less advanced medical treatment) with the situation more appropriate to the individual undergoing the life expectancy determination (Freeman, 2013)
- Use of a mortality index obtained from a single study when there is variability in the values available in the

wider medical literature

#### **Step Four: Combination of Baseline Mortality Rates and Comorbid Mortality Effects**

This step involves integration of the influences identified in Step Two and quantified in Step Three to generate a schedule of modified EDRs or MRs to be used to build a new life table. (The basic input required for the modified table is the adjusted mortality rate,  $mx$ ; see Appendix Two.) In general, this process entails addition of the mortality indices for the individual conditions, although there are many qualifying concerns.

#### **Number and Significance of Conditions for Inclusion**

Theoretically, the additive approach allows for an indefinite number of elements to be incorporated into the analysis, as long as mortality data is available for them, although practically speaking the investigator needs to select a manageable number of relevant conditions for inclusion. (Due to the prevalence of comorbidity, it is unlikely that an analyst will only use a single mortality influence in a life expectancy determination, but it may occur, especially in younger individuals.) Potentially positive and negative effects were discussed in Development of Secondary and Subsequent Conditions and Influence of Medical Care, Including the Life Care Plan above. Shavelle et al. (2007) observed that:

How can all these factors be incorporated into an estimate of a given individual's life expectancy? It is evidently not feasible to take them all into account in a scientific analysis. The rational approach is to work with the available data as far as possible" (pp. 256-257).

In establishing the influences for inclusion in the life expectancy determination (Steps Two and Three above), the investigator will usually be working with hazard (HR) and/or mortality (MR) ratios from medical research, usually by Kaplan-Meier or Cox proportional hazards analysis (Collett, 2015). The medical literature generally treats individual hazard or odds ratios of 2.0 or less as not significant with regard to causation (Hegmann et al., 2014; Melhorn et al., 2014). Similarly, Streiner & Norman (2012) cited a "rule of thumb" in epidemiologic analysis that HRs and ORs  $< 0.5$  (for positive or preventive effects) or  $> 2.0$  (for negative effects or risks) are significant. Strictly applied, these criterion levels might initially eliminate many influences. However, when combined, a sum of smaller influences may reach significance.<sup>29</sup> The analyst may thus consider constructing a hierarchy of EDRs or HRs/MRs and using only those which they consider to be most influential.

#### **Heterogeneity of Conditions and Levels of Mortality Influence**

There may be subdivisions or sub-levels of mortality

influence which may need to be considered for any condition, which may increase the accuracy of life expectancy determination (Shavelle & Delaney, 2010). For example, the mortality of a given type of cancer may vary with tumor histology, stage, grade, treatment, and response (National Cancer Institute, 2020). Mortality effects of coronary artery disease (CAD) may vary with the number of vessels affected, calcium score, prior MI, and ejection fraction or EF (NCHS, 2020). The long-term mortality of diabetes is influenced by insulin dependence, long-term glycemic control, and the presence of complications, e.g., the "triopathies", including nephropathy, neuropathy, and retinopathy (Brownrigg et al., 2016; Tancredi et al., 2016). Behavioral conditions such as smoking may demonstrate a dose-response effect on mortality (Lugo et al., 2017), and mortality of substance abuse is affected by many variables (Walker et al. 2017). An individual's risk profile may vary both with the characteristics of a given disorder and progression of the person's illness (Fasano, 2009).

Thus, the life expectancy analyst may need to make judgment decisions on how deeply to go into permutations based on the particular situation and availability of adequate data. In cases in which large amounts of information become difficult to manage and make it difficult to extract reasonable conclusions, the evaluator may need to simply acknowledge the variability involved and limit the number of conditions and levels considered. In most cases it is desirable to explain the thought process so that the reader or trier of fact can appreciate the considerations involved.

#### **Secular Trends<sup>30</sup>**

The mortality effects of either a primary condition or its sub-levels may not be static with time and may change with progression of the disorder, e.g., non-insulin dependent diabetes may evolve to insulin-dependent diabetes with complications including the "triopathies" (and consequent functional impairment); poorly controlled lipid disorders or hypertension may result in atherosclerotic cardiovascular disease or CVA; and obesity may result in a number of consequent disorders, including cancer, dyslipidemia, hypertension, and chronic kidney disease (Bray, 2004). Shavelle et al. (2019) speculated on the effects of functional deterioration in persons suffering CVA, although they noted that "We are not, however, aware of any empirical evidence on this topic" (p. 4). The analyst may need to consider these changes if the progression is rapid or the life expectancy determination encompasses a long enough period of time.

#### **Duplication of Mortality Effects and Adjustment for Overlap of Conditions<sup>13</sup>**

A significant problem with the combination of EDRs and/or MRs in adjustment of baseline life tables for comorbid-

ity is overlap of mortality effects and consequent potential for duplication of their influence. Although many medical disorders are independent (e.g., asthma and cancer), many others are not. For example, cigarette smoking, diabetes, dyslipidemia, hypertension, obesity, and lack of physical activity all contribute to CAD (Boudi, 2020), and each condition exerts its own mortality influence in addition to contributing to that of the vascular disorder. As previously noted, Singer & Milano (2007) analyzed EDR data from the MMIS (for single or paired conditions plus hypertension) and found that approximately one-third of combined disorders each showed less than additive (sub-additive), additive, and synergistic (supra-additive) effects.<sup>32</sup> It is important to consider these variations; in general, simply adding mortality indices when the overall effects are sub-additive will overestimate mortality (and thus underestimate life expectancy), and adding synergistic influences will underestimate mortality and overestimate life expectancy (DeVivo, 2002).

There is no clear consensus on adjustment for overlap in the medical literature:

- Chiang (1991) reasoned that “Because there is no simple statistical method available for cause-specific mortality analysis when risks are dependent, independence of risks is generally assumed. But some researchers have questioned the validity of the assumption ... which has become the focal point in the discussion of analysis methods. Perhaps there is no unique answer to the question of risk independence. The answer probably depends on the risks involved and, possibly, on the population under study” (pp. 285-286).
- Singer (2005) related: “In such cases, I often discount the total EDR by 20% or more, to allow for overlap in the multiple risk factors and for future improvement in medical care with general reduction in mortality” (p. 49). Unfortunately, the author did not provide any rationale for the correction factor).
- Thomas & Barnes (2010) stated that “The review of the totality of the world literature would indicate that each factor may need a reduction in the range of 10–20%. ... The exact percentage reduction has to be a matter of clinical opinion”, and “made according to the circumstances of the specific individual” (p. 207). However, again, the authors did not cite any specify literature supporting those figures.
- Vachon & Sestier (2013) addressed the question as follows:

Another limitation is in the assumption of independence. When a patient suffers from multiple conditions, to what extent are any of them duplicative? That is, if someone suffers from obesity, hypertension, coronary artery disease, and diabetes, it would be unwise to simply cumulate all the risks measured individually. To some

extent, the excess mortality from diabetes mellitus or hypertension is related to heart disease. To count each risk individually and then sum all of them would certainly factor in significant double or triple counting. An individual should not be taxed several times for what are in fact several risks overlapping and somewhat blending into one. The expert could, of course, try to identify a study or article addressing survival for patients with this particular complex of pathologies, but this may prove fruitless. In such cases, some epidemiologic judgment may be called for as to which risks are independent and which overlap partially or completely. In some rare cases, the global risk may actually be greater than the sum of the parts, when some synergy between the risks exists (for instance, it may be so that the excess mortality associated with smoking and asbestos exposure is greater than the sum of each individual risk). In complex cases like these, the exercise of calculation is not simply formulaic. If independent judgment may, or should, be exercised in such complicated cases, it is of course no license to jettison any relation to scientific foundations. Complexity does not grant a license to speculate groundlessly; the opinion must remain within the realm of the epidemiologically sound (p. 550).

- DeVivo et al. (2018) acknowledged the assumptions that the effects of additional risk factors were independent and that the effects of comorbidities were the same in persons with SCI as in the general population, and cautioned that “In many cases, these assumptions will not be entirely valid, so careful consideration is warranted when there are important additional risk factors to be included in an individual projection of life expectancy” (p. 5).

The potential interactive effects of mortality influences thus require careful consideration of the specific elements of the individual situation and any available research evidence, as well as exercise of judgment (and clear explanation) by the analyst. There may be several possible approaches to this conundrum:

- The Life Expectancy Project (2020) suggested that simple addition of EDRs may be an acceptable default in situations in which both sub- and supra-additive effects are present and appear to balance.
- Some analysts do not separately consider disorders subsumed by or contributing to other conditions, e.g., coronary artery disease vs. MI, and dyslipidemia as a contributor to atherosclerosis (Milano, 2010).
- Some evaluators advise application of an overarching correction factor as above; in this case the investigator would need to clearly explain the rationale for the chosen value.
- If there are a small number of comorbidities in a given individual, it may be possible to estimate their relative



effects if specific data on the interactive mortality are available. For example, the MMIS provided much useful data in this regard; the Octabaix Study Project (Ferrer et al., 2017) furnished HRs for various combinations of two- and three- disease comorbidity; and Willadsen et al. (2018) developed odds ratios (ORs) for groups of three, four, and five comorbidities.

- It may in some cases be possible to address this problem using the separate prevalence of specific comorbidities in a population (e.g., diabetes and hypertension) to inform and guide “epidemiologic judgment”. The analyst can compare the mortality of each disorder alone with that of the mortality of combinations of the disorders, and make adjustments as necessary (see, for example, DeVivo, 2002, and Kush, 2015; the Life Expectancy Project [2020] outlined a procedure considering three factors). Again, this approach is limited to a small number of comorbid conditions.

### ***The Problem of Maximum Effect***

An additional concern posed by the presence of multiple conditions is superseding of influence, especially when one or more conditions is slowly developing and chronic. For example, DeVivo (2002) pointed out that the mortality effects of SCI, especially at higher spine levels and degrees of neurologic impairment, may overshadow those of active smoking, and that an individual’s lifespan may be reduced by SCI to the point that heart and lung disease due to tobacco use do not have time to develop. Additional examples would include the long-term effects of alcohol abuse and diabetes, many forms of cancer, and severe kidney, liver, and vascular disorders.

### ***Uncertainty or Ambiguity in Effects of Conditions***

When there is uncertainty about the presence, effect, or salience of a given disorder or combination of conditions, the analyst may elect to present alternative scenarios, clearly explain the options and their ramifications in the text of the report (including presentation of additional modified life tables) and leave the final determination of the importance of the mortality effects of the disorder to the trier of fact (Vachon, 2019).

## **Step Five: Generation of New Life Table and Life Expectancy Estimate**

### ***Apply New Age-Specific Mortality Rates to Standard Life Table***

As discussed under Overview of the Life Expectancy Determination Process above, the outcome of Step Four is a schedule or table of combined age-specific comparative mortality figures, based on either EDRs or MRs, ready to

be combined with the baseline age-specific mortality rates. These are used to generate the modified life table, in standard fashion. The analyst incorporates the age-specific EDRs into the baseline general population life table, usually copying them into the appropriate column on the worksheet and recalculates the table (Roberts & Donaldson 2010; Singer 2005).<sup>33</sup> Anderson (2002) illustrated a “double-table” method, with baseline and modified tables side by side; the new life expectancy resulting from the modification is then read directly at the appropriate age stratum.

Vachon & Sestier (2013) provided a discussion of the mathematics of life expectancy (pp. 542-544) and a demonstration of the calculations involved in a typical life expectancy determination (“Calculation Illustrated”, pp. 546-548). The authors presented a hypothetical case of an individual with four medical comorbidities and completed a stepwise application of the methodology described in detail above, using both EDRs and MRs for the medical disorders. They provided both a summary table of the combined mortality rates and a composite modified life table showing the effects of the increased mortality on the individual’s life expectancy.

### ***Potential Errors in Step Five***

Vachon (2019) stressed the point that neither mortality rates nor the life table operate in a linear fashion, and thus, except for small time intervals (e.g., within one year), linear interpolation is not appropriate, and will result in significant error. Accurate life expectancy determination requires calculation of the entire life table according to accepted methods such as the one described.

Rosen et al. (2013) observed that “there is a paucity of literature on predicting the longevity of those individuals with chronic conditions who have already outlived their statistical average life expectancy” (p. 8). However, the “statistical average life expectancy” is by definition an average (measure of central tendency), with many potential values both above and below it; many people will experience an actual lifespan that exceeds their life expectancy, which provides part of the rationale for use of survival probabilities as opposed to discrete life expectancy values (see Part Four below).

## **Part Three: Future Advances in Life Expectancy Estimation and Use in Life Care Planning**

### **Enhancements of Current Methodology *Improvements Due to Accumulated Information***

The natural accretion of empirical data on various conditions and influences on life expectancy provides important opportunities to refine life expectancy determination:

**Variability in Conditions.** Earlier in the article, illustrations including cancer, CAD, diabetes, and smoking were provided in Part Two, Heterogeneity of Conditions above,



but there are many other potential cases. Day et al. (2015a) noted that differences in functional capacity in cerebral palsy produced marked divergence in life expectancy, and suggesting that stratification by capability (e.g., using various functional Classification Systems) may improve predictive accuracy. Rehm et al. (2017) reported differential mortality outcomes for alcohol abuse by both pattern and volume of consumption and resultant disease entity. Many of the more subjective factors discussed in Part Two, especially behavioral health and positive influences, may eventually be found to have a significant effect on life expectancy. For example, Prior et al. (2017) described complex interactive effects of perceived stress and multimorbidity.

**Behavior of Mortality Indices.** As discussed above, the use of constant EDR or MR in life table modification may be appropriate for some conditions, but both PLE and LDR have been found to conform most closely to empirical data for many situations (Day et al., 2015a; Strauss et al., 2001; Strauss et al., 2005). The accumulation of further information about the natural history and time courses of various disorders may allow more focused application of these techniques.

**Projected Mortality Rates.** Increased amounts of reliable data allow advancement of work on predicted mortality rates, which may eventually be more widely incorporated into life expectancy determination (Day et al., 2015a). Bell & Miller (2005) analyzed both past and potential future influences on US population mortality and anticipated continued but somewhat slower increases in life expectancy to 2100. Similar studies have been provided for the United Kingdom (Office for National Statistics, 2019) and on a global scale (World Health Organization (WHO), 2020).

### ***Interactions of Conditions***

There has been a relative dearth of research on the interactive effects of various conditions and influences, both positive and negative, on life expectancy since the SOA's MMIS (1998) and the studies of Milano & Singer (Milano & Singer, 2007; Singer & Milano, 2007). The methodological challenges of this work are formidable; for example, Calderon-Larranaga et al. (2019) provided an excellent conceptual overview of the interplay between multimorbidity and cognitive and physical function. Much of the research into comorbidity involves counts of conditions (Cho et al., 2013; Dugoff et al., 2014; Nunes et al., 2016) and/or demographic factors (Jani et al., 2019). Willadsen et al. (2019) explored combinations of three, four, and five conditions, finding a progressive increase in mortality up to six-fold increased risk, although the patterns were generally additive and not synergistic. Since comorbidity is the rule rather than the exception, understanding of the complex interplay of conditions will be crucial to increasing accuracy of life expectancy.

### ***Improvement in Measurement and Accuracy of Life Expectancy Determination***

Both Shavelle & Strauss (2009) and Xu (2020) examined challenges and difficulties in life expectancy determination in the life settlements industry. The former authors detailed a method of independently assessing the accuracy of life expectancy determinations both for individual raters and underwriters and rating firms in this setting. It appeared that the technique would be applicable in other contexts, including life expectancy determination in forensic and medico-legal situations, if the necessary data were available. The latter researcher illustrated a complex approach to analyzing underwriting performance using the concept of mortality multipliers which may be useful to life expectancy determination for those with suitable mathematical background.

### ***Correction for Background Mortality***

Given the concern about "double-counting" by use of standard population life tables as a comparison or expected baseline (see "Double-Counting" in Life Table Adjustment above), methods of accurately correcting for background mortality for given conditions are of interest. As noted, some researchers have presented complex mathematical models for accomplishing this goal (Touraine et al., 2019; Wang et al., 2013). However, these techniques are not accessible for analysts without sophisticated mathematical backgrounds. These processes may allow for increased accuracy in life expectancy determination as they evolve and become simpler and into more widespread use.

### ***Use of Survival Probabilities vs. Single Life Expectancy Value in Life Care Planning***

The outcome of the life table modification process extensively detailed in Part Two of this article is a set of age specific life expectancy values which can then be used to anticipate an individual's lifespan for forensic and legal purposes. Many authors have advocated actuarial use of the survival probabilities of the modified table, instead of single overall life expectancy figures, as a basis for economic analysis (Anderson, 2008; DeVivo, 2002; Krause & Saunders, 2010; Strauss et al., 2001). This approach is still critically dependent upon an accurate life table, with valid age-specific mortality probabilities throughout the life expectancy, but may provide a more rational economic basis for tort settlements and life care planning.

### ***New Approaches Using Predictive Modeling***

Advances in predictive modeling may also offer improvements in the clinical prediction model approach described in Part One. For example, Maier et al. (2019) applied artificial intelligence techniques (random survival forest) to a large historical data set and enhanced mortality prediction by 6%.

Xu & Hoesch (2018) described three applications of “big data” predictive analysis and processing currently in use by insurers, both to identify higher-risk claims and less commonly in life expectancy determination, and proposed a modification algorithmically combining a physiological factor (a currently and commercially available chromosomal telomere test) and medical and psychological information from an individual’s “digital footprint” to generate life expectancy. Kang & Adibi (2018) outlined current challenges to predicting personalized life expectancy and described a combination of multiple technologies, including electronic and mobile health data collection and monitoring, cloud computing, “big data”, and the Internet of Things, which can be applied to overcome them and generate accurate estimates.

#### Part Four: Summary

There are many different approaches to life expectancy determination currently in use, with different levels of practice in different applications. These range on a continuum of accuracy from reliance upon anecdotal clinical experience and opinion and use of the standard life table (the usual default life care planning practice) to proprietary actuarial or insurance information and rated ages (e.g., MCPs and MSAs) to life table modification (the standard in forensic cases for economic damages) and very complex academic statistical calculation and research (e.g., in epidemiology and medical applications).

Present practice in forensic life table modification in medicolegal cases, involves five main steps, including selection of an appropriate baseline life table; identification of relevant conditions influencing the subject’s life expectancy; reliable and evidence-based quantification of those influences; synthesis of those mortality effects into mortality rates that can be combined with the baseline life table; and generation of a new life table and determination of life expectancy. The present paper described many considerations along each of these steps.

Significant current controversies in life expectancy determination include a) whether and how baseline mortality influences from pre-existing conditions embedded in the standard population life table may affect the accuracy of

individual life expectancy determination (i.e., “double-counting”); b) the effects of positive and negative influences on life expectancy and incorporation into overall life expectancy determination; c) the overlap or duplication of influences (the overarching issue of competing risks); d) and incorporation of secular (time) effects (e.g., of improvement in health care, either overall or by effects of legal award), functional capacity, and SEFs. Improvements in all of these areas are forthcoming with the accumulation of new and more detailed empiric data on life expectancy in various medical conditions and continued evolution of thought and practice in life expectancy determination.

At this point in time, like many other aspects of modern medicine, life expectancy determination for individuals is an inexact science, based on probabilities and not certainties. It is currently impossible to account for all variables contributing to a given person’s eventual lifespan. Even if all relevant influences could be identified, their effects are uncertain, and interactions among them present an unmanageable level of complexity. However, as Shavelle et al. (2007) observed:

[It is incorrect to assert] that because a scientific analysis cannot take account of every factor relevant to life expectancy of a given individual, nothing scientific can be said about an individual’s prognosis for survival. If this were true then standard government life tables would be irrelevant to an individual, and economists and others have been wrong to refer to them. It would also mean that life insurance actuaries and medical directors, who routinely decide whether to offer insurance to individuals and at what price, have no basis for making such decisions. . . . How can all these factors be incorporated into an estimate of a given individual’s life expectancy? It is evidently not feasible to take them all into account in a scientific analysis. The rational approach is to work with the available data as far as possible” (pp. 256-257).

The present state of the art is that we cannot predict the future, including an individual’s precise lifespan, but we can determine a person’s likely life expectancy with increasingly accurate and precise methods, with proper methodology and application of the knowledge and experience at hand.

**Appendix: Acronyms**

AAIM: American Academy of Insurance Medicine  
 ASIA: American Spinal Injury Association  
 CAD: Coronary artery disease  
 CCI: Charlson Comorbidity Index  
 CEO: Chief Executive Officer  
 CI: Confidence interval  
 CMS: (U. S.) Centers for Medicare and Medicaid Services  
 COVID-19: Coronavirus Disease 2019  
 CVA: Cerebrovascular accident (stroke)  
 DEALE: Declining Exponential Approximation of Life Expectancy  
 EDR: Excess Death Rate  
 EF: Ejection fraction  
 EPV: Expected Present Value  
 HALE: Health-adjusted life expectancy  
 HDL: High density lipoprotein  
 HR: Hazard Ratio  
 IBS: Irritable bowel syndrome  
 IME: Independent Medical Evaluation  
 LDR: Log-linear declining relative risk  
 MALE: Measure of Actuarial Life Expectancy  
 MCP: Medical Cost Projection

MD: Medical Doctor  
 MMIS: Multiple Medical Impairment Study  
 MPH: Master of Public Health  
 MR: Mortality Ratio  
 MSA: Medicare Set-Aside  
 NBA: National Basketball Association  
 NCHS: (U. S.) National Center for Health Statistics  
 NSCISM: (U. S.) National Spinal Cord Injury Statistical Center  
 OR: Odds Ratio  
 PE: Pulmonary embolism  
 PLE: Proportional life expectancy  
 RMR: Relative Mortality Ratio  
 SCI: Spinal cord injury  
 SEER: (U. S.) Surveillance, Epidemiology, and End Results Program  
 SEF: Socioeconomic factor  
 SMR: Standardized Mortality Ratio  
 SOA: Society of Actuaries  
 TBI: Traumatic brain injury  
 U. S.: United States  
 VTE: Venous thromboembolism  
 WC: Workers Compensation

Age (years)	Age-specific mortality rate	Probability of dying between ages $x$ and $x+1$	Number surviving to age $x$	Number dying between ages $x$ and $x+1$	Person-years lived between ages $x$ and $x+1$	Total number of person-years lived above age $x$	Expectation of life at age $x$
$x$	$m_x$	$q_x$	$l_x$	$d_x$	$L_x$	$T_x$	$e_x$
0–1	0.005806	0.005777	100,000	578	99,493	7,860,752	78.6
1–2	0.000382	0.000382	99,422	38	99,403	7,761,259	78.1
2–3	0.000248	0.000248	99,384	25	99,372	7,661,855	77.1
3–4	0.000193	0.000193	99,360	19	99,350	7,562,483	76.1
4–5	0.000149	0.000149	99,341	15	99,333	7,463,133	75.1
5–6	0.000141	0.000141	99,326	14	99,319	7,363,800	74.1
6–7	0.000126	0.000126	99,312	13	99,305	7,264,481	73.1
7–8	0.000114	0.000114	99,299	11	99,294	7,165,176	72.2
8–9	0.000104	0.000104	99,288	10	99,283	7,065,882	71.2
9–10	0.000095	0.000095	99,278	9	99,273	6,966,600	70.2
10–11	0.000093	0.000093	99,268	9	99,264	6,867,327	69.2
20–21	0.000795	0.000795	98,937	79	98,897	5,875,727	59.4
30–31	0.001351	0.001351	97,872	132	97,806	4,891,263	50.0
40–41	0.001938	0.001936	96,321	186	96,228	3,919,862	40.7
50–51	0.004038	0.004030	93,797	378	93,608	2,967,831	31.6
60–61	0.009134	0.009093	88,226	802	87,825	2,054,253	23.3
70–71	0.018600	0.018428	77,697	1,432	76,981	1,219,730	15.7
80–81	0.049351	0.048163	57,839	2,786	56,446	531,161	9.2
90–91	0.152543	0.141733	24,560	3,481	22,819	111,405	4.5
100+	0.456525	1.000000	1,894	1,894	4,148	4,148	2.2

### Appendix Two: Life Table Terminology

An abridged version of the U.S. life table for the total population (Arias & Xu, 2019, Table 1, p. 10) is shown below, with explanation of each term in the table (AAIM, 2015; Pokorsky, 1988; Shavelle, 2012; Singer, 2004; Vachon, 2020).

The columns of the life table, from left to right, are:

- **x**: **age** in years at last birthdate
- **$m_x$** : the age-specific **mortality rate**, the number of deaths at age  $x$  divided by the exposure (measured in number of person-years of risk; see Footnote #1). This is the sole external input into the table (e.g., from census data, where deaths are obtained from death registries or vital statistics data, and exposure from population estimates). This number can also be calculated by  $m_x = d_x/L_x$ . For example,  $m_{50} = 378 / 93,608 = 0.004038$ .
- **$l_x$** : the **number of persons alive** at age  $x$ , or “survivorship function”. By convention, the starting population (radix) at age  $x = 0$  yrs is assigned a value of 100,000. This is calculated as  $l_{x+1} = l_x * e^{-m_x}$ , or equivalently,  $l_{x+1} = -\ln(l_x)$ ; i.e.,  $l_{x+1}$  is  $l_x$  multiplied by the base  $e$  raised to the minus  $m_x$  power, or the negative of the natural logarithm of  $l_x$ . For example, of the original 100,000 persons in the hypothetical cohort,  $l_5 = 99,326$  (or 99.3%) live to age  $x = 5$ . The number alive at age  $x = 6$  is  $l_6 = l_5 * e^{-m_5} = 99,326 * 0.999859 = 99,312$ .
- **$d_x$** : the **number of deaths** in the interval  $x$  to  $x+1$  for persons alive at age  $x$ , or “decrement function”. This is calculated as  $d_x = l_x - l_{x+1}$ . For example, of the  $l_5 = 99,326$  persons alive at age  $x = 5$ ,  $d_5 = 99,326 - 99,312 = 14$  die prior to age 6.
- **$q_x$** : the **probability of dying** at age  $x$ , or number of deaths at age  $x$  divided by the number of persons alive at age  $x$  or age-specific risk of death. This is calculated as  $q_x = d_x/l_x$ . For example,  $q_{50} = 378 / 93,797 = 0.004030$ .

NOTE that there are several unique features of  $m_x$  and  $q_x$ :

- They are not identical (see Singer, 2004, p. 231); for a short interval, e.g., one year, they will be close in value, “but since  $L_x$  is always greater than  $l_x$ ,  $m_x$  will always be larger than  $q_x$ ”. For example, at age  $x = 50$ ,  $m_{50} = 0.004038$  is close to but greater than  $q_{50} = 0.004030$ , and at age  $x = 90$ , there is a wider difference, with  $m_{90} = 0.152543$  and  $q_{90} = 0.141733$ .
- In general, rates may be more useful than probabilities, because a) rates may range from zero to infinity, whereas probabilities are bound by zero and 1; b) rates can be added (the mathematical basis of life table adjustment), whereas probabilities cannot; and c) mortality rates are linked to the survival function, i.e.,  $m_x = -\ln(l_x)$ .
- The values of  $m_x$  and  $q_x$  can be derived from each

other; see, for example, Arias et al., 2017 (Calculation of the Probability of Dying, p. 61).

- **$L_x$** : **total number of person-years** lived by the cohort from age  $x$  to  $x+1$ . This is calculated as  $L_x = l_{x+1} + d_x/2$ , or the sum of the years lived by the  $l_{x+1}$  persons who survive the interval, and the  $d_x$  persons who die during the interval. The former contributes exactly 1 year each, while the latter contribute, on average, approximately half a year (people will die at various times throughout the year, averaging one-half year of life during the interval). For example,  $L_5 = 99,312 + (14 * 0.5) = 99,312 + 7 = 99,319$ . Other methods are used at age 0 and at the oldest age (see Arias & Xu, 2019).
- **$T_x$** : **total number of person-years** lived by the cohort from age  $x$  until all members of the cohort have died. This is calculated as  $T_x = T_{x+1} + L_x$ , or the sum of numbers in the  $L_x$  column from age  $x$  to the last row in the table (calculated in reverse, i.e., from the last row to the first). For example,  $T_5$  equals the sum of all  $L_x$  values from  $x = 100+$  to  $x = 5$ , or 7,363,800 person-years.
- $e_x$ : the (remaining) **life expectancy** of persons alive at age  $x$ , computed as  $e_x = T_x/L_x$ . For example, at age  $x = 5$  the life expectancy is  $e_5 = 7,363,800 / 99,319 = 74.1$  yrs.

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#### Footnotes

<sup>1</sup> The life table is a device commonly used in survival analysis; see Use of Unmodified Standard Life or Mortality Tables below for basic information, and an example is shown in Appendix Two. Mathematically, life expectancy is the average or (arithmetic) mean number of years that members of the group can be expected to survive from or at a given age, calculated by dividing the aggregate number of person-years that the group could be anticipated to live by the number of persons in the group at that age (Slesnick & Thornton, 2008). Person-years are used as a measure of involvement or exposure, allowing the analyst to account for both the number of people in the study and the amount of time each individual contributes to the variables of interest. (For example, 1000 person-years might be equivalent to 50 subjects x 20 years each, or 25 subjects x 40 years each.)

<sup>2</sup> Statistically, life expectancy is a mean (average), or sum of all observations divided by the number of observations, whereas the median survival time is the middle value of all observations, with half occurring below and half above that point. For example, if three people are ages 20, 25, and 45 yrs, the mean age is  $(20 + 25 + 45)/3 = 30$  yrs, but the median age is 25 yrs.

<sup>3</sup> This discussion does not include formal actuarial, biostatistical, epidemiologic, and life insurance analyses using complex statistical techniques.

<sup>4</sup> This is sometimes described as the life expectancy method, and should not be confused with the life table method used by economists to calculate expected present values of anticipated future costs for all possible future survival times

(Anderson, 2008; Krueger, 2001).

<sup>5</sup> The rank order presented here generally reflects increasing selectivity by actuarial risk; mortality rates tend to decrease by degree of preselection, so, for example, participants in annuities will usually have a lower risk of death than those in the general population (AAIM, 2015).

<sup>6</sup> The term “cohort”, which generally denotes a group, is used to indicate both an overarching method of life table determination (the “generational” technique discussed above) and this more specific type of life table (Guillot, 2011).

<sup>7</sup> It should be noted that certain legal jurisdictions, e.g., circuit courts and states, provide Pattern Jury Instructions, or model or sample instructions to be used as guidance by judges, which may specify use of particular life expectancy tables. See, for example, Washington State Supreme Court, 2019.

<sup>8</sup> The Gompertz survival function basically posits that mortality rate increases exponentially (as opposed to linearly, or some other defined pattern) with time (Kirkwood, 2015).

<sup>9</sup> Note that this is a different issue from duplication of mortality effects by overlap of conditions, as discussed under Part Two, Step Four below. In this case the concern is “double-counting” of mortality influences (that are presumed to be already accounted for in the standard life table) by adding mortality factors to the baseline, and in the later situation the problem is overlap of mortality effects by two or more conditions that affect mortality in the same or similar ways. (See also Footnote #29.)

<sup>10</sup> All figures were for both females and males in the U. S. population. In this paper, the term “diabetes” will be used to denote diabetes mellitus.

<sup>11</sup> This might be accomplished by adjusting the excess death rate or EDR by the population prevalence of the disorder (see Step Three below). For example, if the EDR was 0.0200 and the prevalence was 30%, the corrected EDR would be  $0.0200 \times (1 - 0.30) = 0.0200 \times 0.70 = 0.0140$ . This approach would require the analyst to set threshold values for significant prevalence, e.g., 33% or 50%, in the general population.

<sup>12</sup> Selection bias refers to systematic error introduced by the nonrandom choice of individuals or data for study. Examples of selection bias include undercoverage, nonresponse, and differential voluntary response (Tripepi et al., 2010).

<sup>13</sup> Investigators often specifically account for other potentially biasing conditions such as age, gender, weight, smoking status, and the presence of possibly influential disorders such as cancer, diabetes, hypertension, and kidney disease.

<sup>14</sup> In this context the term “secular” refers to a temporal phenomenon, occurring over a long period of time.

<sup>15</sup> Life expectancy in the United States has slightly decreased since 2014, attributed by some to the effects of increased



substance abuse, particularly opioids (Hall et al., 2020); the overall population mortality effects of the current COVID-19 pandemic are not yet clear.

<sup>16</sup> This intermediate table is easily generated using an Excel or other spreadsheet by listing age in the left-hand column and the identified relevant conditions in subsequent columns, and the specific mortality figures (usually EDRs) in the appropriate intersection cells by age (Singer, 2005).

<sup>17</sup> From 1945 to 1996 the tables were abridged and closed at 85 yrs of age. Beginning in 1997 the estimates were extended to age 100 yrs, with a composite category for ages 100 yrs and greater (Arias & Xu, 2019).

<sup>18</sup> For example, Kaplan & Feinstein (1974) used the terms “cogent” and “non-cogent” to describe comorbidities which might be expected to affect or not affect an individual’s long-term survival). Cogent disorders involved vital body systems or were associated with anatomic, behavioral, or functional effects that would either threaten life directly or increase susceptibility to fatal illnesses. Non-cogent conditions were those which had occurred in the past, with no brain or heart involvement or residual effects, or that could be well-controlled with conservative means (e.g., medications) and had no direct effect on vital organs (p. 391).

<sup>19</sup> The life expectancy effects of some of these concerns can become complicated. For example, overall population health may benefit from substitution of e-cigarettes (vaping) for tobacco use, but the increased prevalence of e-cigarette use has raised concerns about morbidity and mortality from the practice (Levy et al., 2017).

<sup>20</sup> Examples include commercial divers, lumberjacks, and offshore drilling workers; demolition and other construction workers, especially those working at heights; those in industries with significant radiation hazards (Woodman, 2006).

<sup>21</sup> An example is the positive, neutral, and negative effects of “stress” on the hypothalamic-pituitary-adrenal axis, which involve affective (emotional), behavioral, physiological, and cognitive influences, and are different for each person (Smith & Vale, 2006).

<sup>22</sup> Impairment refers to altered body structure or decreased function. Disability is a broader term which includes impairment, activity limitation, and decreased ability to participate in normal daily activities (WHO, 2018).

<sup>23</sup> The ASIA Impairment Scale (AIS) replaced the modified Frankel grading system as the standard for documentation and management of spinal cord injuries (Roberts et al., 2017).

<sup>24</sup> The Barthel Index is an ordinal scale used to measure performance in activities of daily living or ADLs. See, for example, [https://www.physio-pedia.com/Barthel\\_Index](https://www.physio-pedia.com/Barthel_Index).

<sup>25</sup> After expanding the database used in this 2004 study Strauss et al. (2008) concluded that the economic effects

were not significant, although they recognized that other confounding effects may have been present.

<sup>26</sup> These parameters can also be expressed as rates, where the number of deaths is divided by the number of people exposed or at risk.

<sup>27</sup> From highest to lowest quality, this hierarchy includes experimental studies (either randomized or nonrandomized controlled trial); observational studies of analytical design, including cohort (determine outcome from exposure), case-control (determine exposure from outcome), and cross-sectional designs (determine exposure and outcome simultaneously); and descriptive studies (involving no comparison or control group), including ecological and proportionate mortality ratio studies, and consecutive, multiple, or single case reports.

<sup>28</sup> This is a good general rule for any activity requiring reference to the medical literature, include life care planning.

<sup>29</sup> For example, combining two influences, each with an apparently insignificant HR of 1.5, would involve the calculation  $1.0 + (1.5 - 1) + (1.5 - 1) = 1.0 + 0.5 + 0.5 = 2.0$ , with the latter meeting the significance criterion.

<sup>30</sup> Note that this is a separate issue from the overall downward trend in population mortality with time, as discussed under Adjustment for Secular Trend in Mortality above. In that case, the concern was overarching effects in the entire population, but in the present instance the problem relates to the individual.

<sup>31</sup> Note that this is a different issue from “double-counting” in life table adjustments as discussed under Part One above. In that case the concern was duplication of mortality influences (that are presumed to be already accounted for in the standard life table) by adding mortality factors to the baseline, but in the present instance the concern is overlap of mortality effects by two or more conditions that affect mortality in the same or similar ways. (See also Footnote #8.)

<sup>32</sup> In a second, related study, these authors documented a marked synergistic effect in patients with both depression and diabetes (Milano & Singer, 2007).

<sup>33</sup> Life table reconstruction essentially requires use of computer spreadsheet software such as Microsoft Excel. The basic program can be used as is with addition of simple formulas by the user, and add-in modules allowing more sophisticated analysis are readily available.

#### Author Note

I have no conflicts of interest to disclose.

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