Loneliness as a health risk, *and what the smoking comparison rests on*

Where the comparison between poor social ties and smoking came from, and what happens to the risk of loneliness once health and circumstances are counted.

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IN PLAIN WORDS

A 2010 study pooled 148 studies of social ties and death and drew its result on a chart beside smoking. That chart is a likely origin of the comparison between loneliness and smoking. The study covered ties of every kind, such as being married, seeing friends or belonging to groups, and found people with stronger ties more likely to be alive at follow-up. Very few of those studies measured the feeling of loneliness. Later work separated being alone (few contacts) from feeling alone (loneliness). In the two large cohorts that adjusted most fully for illness, money and mood at the start, being alone still predicted an earlier death, though less strongly, while feeling alone stopped predicting it. Pooled reviews, which mix studies adjusted to different degrees, still find a smaller link for loneliness. Researchers disagree about what the gap means.

A bar chart from 2010

Holt-Lunstad et al. (2010) gathered 148 prospective studies with 308,849 participants, about half of them from North America and just over a third from Europe, with an average age of 63.9 at the start and an average follow-up of 7.5 years. Every study had measured some aspect of a person's social relationships and then counted who died. Pooled with a random-effects model, the odds of being alive at follow-up were 1.50 times higher for people with stronger relationships (95% CI 1.42 to 1.59). The studies disagreed with one another far more than chance allows (I^2 of 81%), and the size of the effect depended on what had been measured. Complex measures that combined marital status, network size and participation gave the largest odds ratio, 1.91. Living alone gave 1.19, with an interval that included no effect. Eight of the studies had measured loneliness, and for those the figure was 1.45 (1.08 to 1.94).

One choice in the method shaped how the result was read later. Where a study reported several models, the authors took the one with the fewest statistical controls, so that studies would be comparable. In their own analysis of moderators, controlled estimates were smaller than raw ones for structural measures such as network size, though not for functional measures such as perceived support. The authors also wrote that causality was hard to establish, since nobody can be assigned at random to be isolated.

The paper's Figure 6 plotted the log odds ratio for social relationships beside the size of other well-known risks, among them alcohol, physical inactivity, obesity and a bar labelled smoking fewer than 15 cigarettes daily. That bar is the likely source of the popular sentence about fifteen cigarettes, though we could not trace who first wrote the sentence. On the chart, the bar for the overall social relationships estimate is shorter than the smoking bar. Two other bars drawn from the social relationships data are longer, one for complex measures of integration and one for contrasts of high against low social support. The editors' summary of the paper says the influence is comparable with smoking and alcohol, which describes the chart fairly, but the one-line synopsis on the same page goes further and says that social relationships have "as much influence on mortality risk" as established risks

such as smoking. In their conclusion the authors also put the size of the effect beside that of quitting smoking. So a sentence saying that loneliness is as harmful as smoking fifteen cigarettes a day makes two moves of its own, swapping all social ties for one feeling and the least-adjusted estimates for causal ones, while the step from a comparable size to an equivalence had already been taken in the journal's synopsis.

The updates that split isolation from loneliness

The same group's follow-up (Holt-Lunstad et al., 2015) took isolation, loneliness and living alone as separate exposures. We could read only its abstract. Across the studies that had controlled statistically for several possible confounds, the odds ratios were 1.29 for social isolation, 1.26 for loneliness and 1.32 for living alone, and the authors found no difference between objective and subjective measures. The abstract adds that initial health status influenced the findings and that the associations were stronger in samples with an average age below 65.

A later synthesis does not show that equivalence. Wang et al. (2023) pooled 90 prospective cohort studies with 2,205,199 adults, again known to us from the abstract only. For death from any cause in the general population, the pooled effect was 1.32 for social isolation (1.26 to 1.39) and 1.14 for loneliness (1.08 to 1.20). The two intervals do not overlap, though the abstract reports no formal test of the difference. Isolation was also linked to cardiovascular death (1.34), and both were linked, more weakly, to death from cancer. A 2025 review restricted to older adults, which we read in full, included 86 studies, 54 of them with data on loneliness and 37 on isolation, and reported hazard ratios of 1.14 for loneliness and 1.35 for isolation (Nakou et al., 2025). The authors call the two associations similar, although the isolation estimate is the larger. For loneliness the studies were highly inconsistent (I^2 of 84%), an Egger test pointed to probable publication bias, and studies that used validated loneliness scales gave a lower estimate (1.10) than those using a single question.

Disease onset has been studied less. Valtorta et al. (2016) reviewed longitudinal studies from high-income countries and reported that people with poor social relationships had a 29% higher risk of a first coronary event (relative risk 1.29, interval 1.04 to 1.59), and a risk of stroke higher by 32%. Unlike the 2010 review, they took the most adjusted model from each study. Only three of their 23 papers had measured loneliness, while most measured isolation. In a subgroup analysis they found no evidence that the association with coronary heart disease differed between loneliness and isolation, a test that three loneliness papers could not have made very sensitive. A funnel plot with apparent gaps and a fixed-effect model that gave lower pooled estimates (1.18 for heart disease, 1.19 for stroke) led the authors to suspect small-study effects, which could reflect reporting bias.

Being alone and feeling alone, in the same people

Pooled estimates mix studies that adjusted for different things. Two large cohorts measured isolation and loneliness side by side, and each applied the same sequence of adjustments to both.

Steptoe et al. (2013) followed 6,500 people aged 52 and older in the English Longitudinal Study of Ageing for an average of 7.25 years. Isolation was an index scored 0 to 5 that counted contact with children, other relatives and friends, marital status and membership of organisations; loneliness was a three-item version of the UCLA scale. The top fifth on each counted as isolated or lonely. In absolute terms, 21.9% of the isolated group died during follow-up against 12.3% of the rest, and 19.2% of the lonely group against 13.0%. After adjusting for age and sex, the hazard ratio for isolation was 1.50; after adding wealth, education, marital status and ethnicity, and then baseline illness, mobility and depression, it was 1.26 (1.07 to 1.48). Loneliness began at 1.26, came down to 1.14 (0.98 to 1.34) once wealth, education, marital status and ethnicity were added, and reached 0.92 (0.78 to 1.09) with the health measures, an interval that includes no association. Loneliness was more closely tied to poor health at baseline than isolation was, particularly arthritis, impaired mobility and depression. Excluding deaths in the first two years left the isolation result in place, and adding loneliness to the isolation model did not shrink it.

Elovainio et al. (2017) ran the same comparison in the UK Biobank, with 466,901 people aged 40 to 69 followed for about six and a half years, during which 11,593 died. Here isolation was a three-item score and loneliness two questions. With adjustment limited to age, sex, ethnicity and existing chronic disease, the hazard ratios were 1.73 for isolation and 1.38 for loneliness. Adding socioeconomic factors, smoking, alcohol, activity, blood pressure, body mass, grip strength, depressive symptoms, cognitive scores and self-rated health brought isolation to 1.26 (1.20 to 1.33) and loneliness to 0.99 (0.93 to 1.06). For loneliness, depressive symptoms and self-rated

health accounted for the largest shares. With both exposures in one model, only isolation predicted death. The cohort recruited about 5% of those eligible and 95% of the analysed sample was white; the authors note the low response rate and say selection bias matters most for prevalence figures, which were not the focus of their study. Both studies are observational, and by the authors' own accounting isolation keeps roughly a third (UK Biobank) to a half (English cohort) of its association with death after adjustment, while loneliness keeps none.

What adjustment can and cannot settle

The finding that loneliness drops to a hazard ratio near one has a strong opposing reading, and it should be put at full strength. Adjusting for a variable removes its influence whether it is a confounder (something that causes both loneliness and death) or a mediator (something loneliness causes, which then causes death). Elovainio et al. themselves framed depressive symptoms and poor self-rated health as mediators, reporting the percentage of excess risk each one explained. If loneliness leads to depression and depression shortens life, a fully adjusted estimate of 0.99 says only that loneliness predicts nothing beyond the factors that were adjusted for, which is a weaker statement than that it does no harm. Holt-Lunstad et al. (2010) made the parallel point that adjusting for health behaviours could understate the effect of social relationships, since part of that effect might run through those behaviours.

Genetic evidence offers one way past this, with assumptions of its own. Liang et al. (2024) used UK Biobank data on 476,100 people followed for a median of 12.2 years. Observationally, loneliness was associated with higher risk for 30 of 56 diseases. They then used Mendelian randomisation, which treats gene variants associated with loneliness as a natural experiment, on the 26 of those diseases with genetic data. For 20, including ischaemic heart disease, stroke, hypertension and type 2 diabetes, they found little evidence that genetic liability to loneliness was causal. For six it was potentially causal, and depression had the largest odds ratio (5.55). The authors concluded that loneliness may mostly be a marker that travels with other risks. Their outcomes were diseases rather than deaths, and their genetic data came mainly from people of European ancestry.

Read together, the two lines leave a path open, though this is our inference and neither paper reports it. The cohorts show that loneliness adds nothing to the prediction of death once circumstances, baseline health and depressive symptoms (and, in the UK Biobank, behaviours and physical measures) are known; in the English cohort the loneliness estimate had already lost significance once wealth, education, marital status and ethnicity were added, before health entered the model. The genetic study suggests that loneliness may cause depression. A route from loneliness to death through depression is consistent with both, and neither study was designed to measure it.

Randomised trials are the other route to causation. Masi et al. (2011) pooled 50 interventions meant to reduce loneliness. The 20 randomised comparisons gave a small mean effect (a standardised difference of about 0.2), smaller than in single-group and non-randomised designs, and 14 of the 20 showed no change on their own. Because the outcome they pooled was loneliness itself, the analysis says nothing about survival.

The word epidemic

How common loneliness is, and whether it is rising, are separate questions from what it does to health. Surkalim et al. (2022) searched for nationally representative prevalence data and found them for 113 countries or territories across 57 studies, but outside Europe the adult data were thin, and only four studies had measured loneliness repeatedly with comparable measures. Two found rises among Danish and Norwegian schoolchildren, and two found no change, in adolescents in the United Arab Emirates and in Swedes aged 77 and over. The authors judged the evidence on trends insufficient, while noting that some health professionals, among them a former United States Surgeon General, had called loneliness an epidemic.

The largest trend analysis we found concerns young adults. Buecker et al. (2021) preregistered a cross-temporal meta-analysis of 345 studies covering 124,855 people aged roughly 18 to 29 who had completed the UCLA scale between 1976 and 2019, with about 90% of the samples drawn from students. Average loneliness rose by 0.56 standard deviations over the 43 years. The curve was nearly flat until about 2000, and studies collected after 2012 showed no further significant rise. The authors still judged the change small and called the epidemic label exaggerated.

A 2025 release on the report of the World Health Organization's Commission on Social Connection says that loneliness affects one person in six and is linked to more than 871,000 deaths a year (WHO, 2025). The release does not say which estimate of risk lies behind that count, and we could not open the full report, so we cannot say whether it rests on

minimally or fully adjusted figures, which for loneliness differ in the cohorts above by the whole effect.

What is not yet known

- Whether loneliness has any effect on mortality of its own once depression is accounted for, or whether depression is the route by which it acts. No design we found separates the two.
- Which relative risk the 2025 estimate of 871,000 deaths a year uses, and how it would change with the fully adjusted cohort estimates.
- Whether the gap between isolation and loneliness holds in South Asia. The two cohorts above are English and British, 88% of the participants in the 2010 review came from North America and Europe, and adult prevalence data outside Europe are sparse.
- Whether repeated measures change the picture. Only one study in the review of heart disease and stroke measured social relationships more than once (Valtorta et al., 2016), and the genetic study measured loneliness at a single time point, so a passing episode and a decade of loneliness are counted the same. Liang et al. (2024) themselves point to a meta-analysis of 76 studies suggesting that loneliness in older people stays fairly stable over time, and argue that a single measure may be a fair stand-in for long-term exposure.
- Whether reducing isolation or loneliness changes survival. The randomised trials pooled by Masi et al. (2011) measured loneliness itself, and the genetic study above looked at the onset of disease rather than at death.

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