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Towards better excess mortality measurements: Mortality's natural variability

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Abstract

This report investigates the natural variability of mortality, which determines the thin line between excesses and normal variation within expectations. I propose a model for mortality's weekly variability based on a Poisson model, driven by potential non-stationary influences that act nation-wide and fast, on time scales of a week.

Results reveal the presence of a significant amount of non-stationary influences that add to mortality's weekly variability, with a magnitude of 3%±1% (1% - 5% with 95% confidence) times baseline mortality, on top of the standard (Poisson) variability. This additional variability is consistently found across 30 European countries (462M people) during 2017-2019. A long-term analysis in The Netherlands (17M people) reveals the same variability between 2010-2019, with substantial increase since 2020 to approx. 5%. Mortality variance may thus very well itself be used as event indicator when variability is higher than expected.

The findings in this report are relevant for all models of mortality and its variability in general, and in particular for developments towards better excess mortality measurements. The additional variability found scales with both baseline mortality and population size in a different way compared to Poisson variability. For a typical mortality baseline of approx. 0.02% per week, the additional variability becomes dominant in populations above approximately 5M people.

A number of causes for the variability found are suggested, of which the most promising, temperature, will be investigated in a follow-up to this report.

Statement of Interest

I declare that this work was done with an interest in science, and personal safety for myself, loved ones, and humanity. Pro bono, independent, without payroll, not funded. The only competing interest was time taken from my normal job (indy app developer in entertainment and music). If you want to support my work, feel free to buymeacoffee.com/AndreRedert, or consider one of the apps at rodotti.nl and qneo.net.

1. Introduction

Recently, excess mortality has risen sharply in many countries around the world, and a substantial part of the excess still has an unknown cause. An essential step in finding the cause is to determine excess mortality in the first place. Excess is defined as observed mortality minus expected mortality, and the latter depends on models that predict mortality based on mortality and/or other types of available data. National institutes typically provide a yearly mortality prediction, the "baseline", based on statistics of observed mortality in a few past years [Cbs1], aided by more complex long-term models that involve state and expected evolution of demographics, wellfare, etc [Sto].

A major issue with national baselines is that they are highly subjective [Lev]. In the context of excess mortality, there clearly is a need for a more objective mortality model. At the same time, other requirements for a baseline are less relevant, e.g. there is no need for forecasting as excess mortality itself requires observing actual mortality.

In The Netherlands, several excess mortality analyses have recently been made that are independent of national baselines, each with its own subjectivity. A geographically-differential analysis was used in [Re1] which additional applied a z-score still based on a past year, a temporal-differential analysis effectively defined a baseline via short-term past and future near the present [Re2], and a fixed baseline in combination with a Poisson model was used throughout a short analysis period [Mee].

Besides a magnitude, a baseline has also an interval around it defining the variability that is considered normal, or non-excess. The interval is either determined via statistics on past years, or by a model such as Poisson models [Poi] that provide a direct, fixed connection between magnitude and interval. This model's main assumption, independence of mortality among people in a population, is highly plausible: Physical bodies of separate people are not interconnected, and the final events in a death process occur locally, within a single body, independent of other people. This independent part of mortality is reflected very well by the Poisson model.

Before death, however, people *are* connected in various ways. A local causal effect between people is that observing death results in mourning and fear, causing stress and subsequent deaths and thus dependencies within e.g. families, neighbours and care-homes. Also, local outbreaks of contagious diseases may cause correlated mortality within care-homes and hospitals. Such and other causal effects are beyond the Poisson model, but local effects do not aggregate strongly on a national level, and can thus be neglected for nations with a substantial population.

A confounding effect in a population is that people do share certain aspects on national scale. For example, staffing in hospitals is better during day-than-night and week-than-weekend, causing patients to experience a shared daily/weekly mortality rate. The strongest such confounders may come from nation-wide shared influences as news, weather, etc. Such effects cause additional variability on the fine temporal scale of days or weeks.

This report is one step towards a better method for determining excess mortality, and investigates the natural variability of mortality rather than its magnitude, focusing on the difference between excesses and normal variation within expectations. This work is motivated by a preliminary observation that weekly mortality consistently exhibited substantially higher variability over time than can be explained by a Poisson model only. This suggests that some common cause affects entire populations in a fast and coordinated way, in contrast with regular seasonal effects that act over months. Many phenomena are candidates for this common cause, e.g. news, and weather/environmental aspects as rainfall/humidity, sun-hours, air quality etc. In a further report, I will investigate temperature as proximal cause (it may typically act as a catalyst for other causes), as it seems the most promising: it is the main element in seasonal and heat-wave mortality, is known to vary strongly on daily basis, and has been shown to affect mortality instantaneously (within 0-3 days) in the case of extreme heat [Xia].

Next follow my model for mortality variability, experiments that measure its parameters , and a conclusion. The experiments involve populations of The Netherlands (17M people), one of its municipalities Rotterdam (0.6M), and 30 European countries (462M), over years 2010-2023.

2. Mortality variability model

In this section I propose a simple model for mortality's weekly variability based on generic stochastic modeling and sources of additional variability that act on different time-scales.

2.1 Poisson model

An often used model for a population's mortality is the Poisson model [Poi]. It has a single parameter λ that equals both absolute mortality's mean and variance within a specific period. The Poisson distribution is very well approximated by a normal distribution, which will ease my analysis:

$$M(t) = \lambda + \sqrt{\lambda}\mathcal{N}(t) \tag{1}$$

t	Integer number indicating a unit time period, in this report a week
$M(t)$	Absolute mortality in week t
λ	Poisson parameter
$\mathcal{N}(t)$	Zero-mean, unity-variance uncorrelated stationary normal-distributed process

For mortality's mean and (standard) deviation, one finds trivially:

$$\begin{aligned} \mu_M &= \lambda \\ \sigma_M &= \sqrt{\lambda} \end{aligned} \tag{2}$$

Vice versa, this enables easy estimation of paramater λ from mortality observations $M(t)$. Alternatively, overall considerations may be used, e.g. if people live about 100 years, on average about $0.02\,\%$ of the population will die per week, leading to:

$$\lambda = \beta P \tag{3}$$

β	Proportion of population that dies per week, typically ~ 0.02%
P	Population size

The Poisson model's mathematical property $\mu_M = \sigma_M^2$ originates from the mechanical assumption that the probability of death for each person is independent of that of other persons. Even if every person has its own specific probability (β_i for person i), the mortality within the entire population, or any subpopulation, will adhere to a Poisson model with some λ reflecting the overall properties of that population (with the population's β the mean of all personal β_i's).

The independence assumption sounds highly plausible, but should be treated with care. It is true that the physical bodies of separate people are not interconnected, and that the final events in a death process occur locally, within a single body, independent of other people. This independent part of mortality is reflected very well by the Poisson model.

2.2 Non-stationary model

Before death, people *are* connected in various ways. A local causal effect between people is that observed deaths instill mourning and fear, causing stress and subsequent deaths and thus dependencies within e.g. families, neighbours and care-homes. Causal effects are incompatible with the Poisson model, but local effects do not aggregate strongly on a national level, and can thus safely be neglected.

A confounding effect in a population is that any time up to the moment of death, people share local environments, e.g. staffing in hospitals is better during day-than-night and week-than-weekend, causing patients to experience a shared daily/weekly mortality rate. The strongest such confounder comes from the population-wide shared environment as news, weather, etc, of which the seasonal effect is the most common. These confounders act via the population-shared λ; the Poisson model is still very well applicable but becomes so-called non-stationary, with time-dependent $\lambda(t)$, see Figure 1.

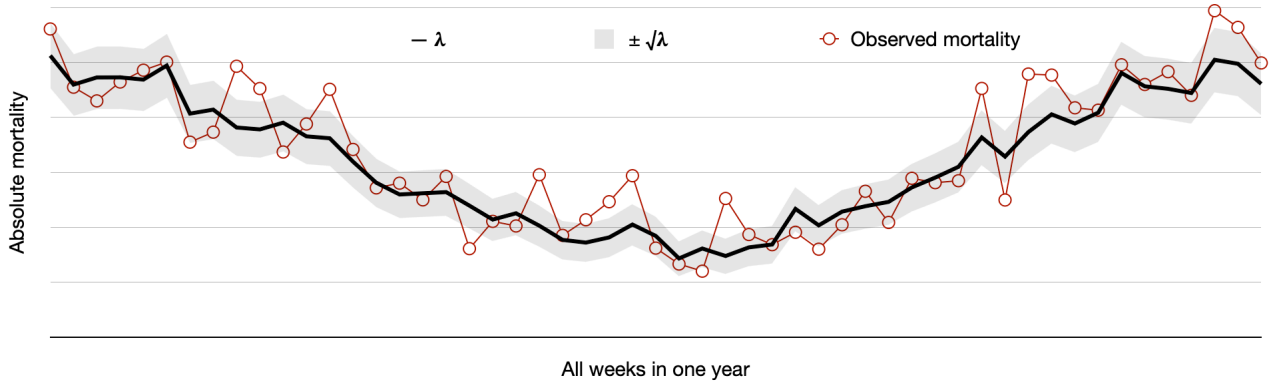

Figure 1: An illustration of how observed mortality (red circles) originates from Poisson noise (grey) around a baseline (black) that has slow, long-term (monthly or more) variability e.g. due to seasons, and possible additional fast, short-term (monthly down to time resolution: weekly) variability via some yet unidentified additional, external cause.

2.3 Decomposition of variability by time scale

I decompose $\lambda(t)$ into two complementary parts, a global baseline $b(t)$ that varies slowly over months or more, and everything else as $\bar{b}(t)$. The $\bar{b}(t)$ will automatically, by complement, be zero-mean and vary fast, in time frames of a week (the resolution used in this report) to at most a month:

$$\begin{aligned} \lambda(t) &= b(t) + \bar{b}(t) \\ b(t) &= \{F * \lambda\}(t) \\ \bar{b}(t) &= \lambda(t) - b(t) \end{aligned} \tag{4}$$

$b(t)$	Baseline, slowly changing on long-term (monthly), due to e.g. seasons
$\bar{b}(t)$	All short-term additional variability in $\lambda(t)$, from monthly down to time resolution (weekly)
$*$	Temporal convolution
F	Smooth time-symmetric averaging filter, Gaussian-shaped with deviation of ±3 weeks

This decomposition is without any model assumptions or restrictive modeling; it is just an identity, for any filter F. I chose a specific filter that ensures monthly variations in $\lambda(t)$ will be collected in baseline $b(t)$, while fast weekly variations will end up in the complement $\bar{b}(t)$. The $\bar{b}(t)$ represents variability from any possible additional nature.

2.4 Model for fast variability

In order to be able to measure anything meaningful about $\lambda(t)$, a restrictive model *is* needed, otherwise the amount of freedom in $\lambda(t)$ will equal or surpass that of observations $M(t)$ leading to underdetermination. I model $\bar{b}(t)$ as follows:

$$\bar{b}(t) = r(t)\mathcal{N}(t) \tag{5}$$

$r(t)$	Magnitude of additional mortality variability $\bar{b}(t)$, itself *slow-changing* as baseline $b(t)$
$\mathcal{N}(t)$	Same as in (1), note all appearing $\mathcal{N}$'s are *separate*, *independent* random variables

The fact that both $b(t)$ and $r(t)$ are modeled as slow-changing, on time frames of at least a month, ensures that weekly observations $M(t)$ will suffice for estimation of model parameters $b(t)$ and $r(t)$.

2.5 Combined variability

For mortality, the net result of (1) and (4)-(5) is (with the two random terms indicated by equation number in subscript):

$$\begin{aligned} M(t) &= b(t) + r(t)\mathcal{N}_{(5)}(t) + \sqrt{b(t) + r(t)\mathcal{N}_{(5)}(t)}\,\mathcal{N}_{(1)}(t) \\ &\approx b(t) + r(t)\mathcal{N}_{(5)}(t) + \sqrt{b(t)}\,\mathcal{N}_{(1)}(t) \\ &= b(t) + \sqrt{b(t) + r^2(t)}\,\mathcal{N}(t) \end{aligned} \tag{6}$$

The last term in the top row volves a complex product of the two random variables. Assuming $r \ll b$, that is, the additional mortality variability is substantially smaller than baseline mortality level, the product term can be neglected and one arrives at the bottom equation with only one random term with a magnitude combining $b(t)$ and $r(t)$.

2.6 Equivalent model normalized by population

The same model with parameters normalized by population is:

$$\begin{aligned} \beta(t) &= b(t)P^{-1}(t) \\ \rho(t) &= r(t)P^{-1}(t) \\ m(t) &= M(t)P^{-1}(t) \\ M(t) &= \beta(t)P(t) + \sqrt{\beta(t)P(t) + \rho^2(t)P^2(t)}\,\mathcal{N}(t) \\ m(t) &= \beta(t) + \sqrt{\beta(t)/P(t) + \rho^2(t)}\,\mathcal{N}(t) \end{aligned} \tag{7}$$

where m, β, ρ are the per-population versions of M, b, r. In (7), both $\beta(t)$ and $P(t)$ have become time-dependent compared to (3). For analysis periods covering less than a few years, $P(t)$ may well be taken as constant. The two terms in the square root that regulate mortality variance differ by a factor P.

It may be that a source of additional variability ρ exist that, like β, has the property of being reasonably constant across different populations. Then, for sufficiently small populations the β term associated with Poisson noise may dominate, but for larger populations, the ρ term associated with additional variability will inevitably take over at some point.

2.7 Estimation of parameters from mortality observations

Estimation of model parameters b, r (or β, ρ) over time can be done via mortality's local mean $\mu_M(t)$ and deviation $\sigma_M(t)$ near time t:

$$\begin{aligned} \mu_M(t) &= b(t) &&= \beta(t)P(t) \\ \sigma_M(t) &= \sqrt{b(t) + r^2(t)} &&= \sqrt{\beta(t)P(t) + \rho^2(t)P^2(t)} \end{aligned} \tag{8}$$

I use the following estimators for $\mu_M(t)$ and $\sigma_M(t)$ based on regular 1st and 2nd moments of mortality observations $M(t)$, using the same filter F defined in (4):

$$\begin{aligned}\mu_M(t) &= \{F * M\}(t)\\ \sigma_M(t) &= C \cdot \sqrt{\{F * (M - \mu_M)^2\}(t)}\end{aligned} \tag{9}$$

C Normalization constant (≈ 1.1 similar to N vs $N-1$ in conventional variance estimators, depends on F)

2.8 Indicative parameter

I define parameter k, indicative of the amount of additional mortality variability relative to Poisson variability:

$$k(t) = \frac{\sigma_M(t)}{\sqrt{\mu_M(t)}} = \sqrt{1 + \frac{r^2(t)}{b(t)}} = \sqrt{1 + \frac{\rho^2(t)}{\beta(t)} P(t)} \tag{10}$$

When $k = 1$, weekly mortality exhibits only Poisson variability, without additional variability ($r = \rho = 0$). For $k > 1$, additional weekly mortality variability exists via $r, \rho > 0$. At $k > \sqrt{2}$, the additional variability exceeds Poisson variability.

For $k < 1$, weekly mortality variability is below the amount possible by a Poisson model only, and no r, ρ exist to explain such dynamics. Real events causing $k < 1$ are temporal mortality correlations due to e.g. deterministic processes in the population, or data providers that interpolated missing data, etc. Also, estimator (10) will exhibit measurement noise leading to $k < 1$.

In this report, I will use plain $k(t)$ only for visual inspection, while statistics μ_k, σ_k over time will be used numerically; k-estimator noise is minimized in μ_k and revealed in σ_k.

3 Results

Next follow experiments with simulations to test the proposed estimators, and experiments with real observed mortality for The Netherlands and its city Rotterdam, and from 30 European countries. Data was available from 2010-2022 [Cbs,Eur], except for Rotterdam 2019-2022 [Cbs]. The per-population paramaters β, ρ will be used to account for population changes over time.

3.1 Simulations

Table 1 shows several mortality simulations with true and estimated β, ρ for several populations P. Each simulation includes 3 years with seasonal changes via $\Delta\beta$, and a yearly population growth of 0.5%:

$$\begin{aligned}P(t) &= P \cdot 1.005^{\frac{t}{52}}\\ \beta(t) &= \beta + \Delta\beta \cdot \sqrt{2} \cos 2\pi \frac{t}{52} + \rho \cdot \mathcal{N}(t)\end{aligned} \tag{11}$$

I added the $\sqrt{2}$ to have σ_β, the estimated deviation of β, match $\Delta\beta$.

Simulation				Estimation $\mu \pm \sigma$		
Population P	Baseline & seasons $\beta \pm \Delta\beta \cdot 10^6$	Additional variability $\rho \cdot 10^6$	Indicator k	Baseline & seasons $\beta \cdot 10^6$	Additional variability $\rho \cdot 10^6$	Indicator k
10k	200 ± 30	0	1	183 ± 45	117 ± 24	0.9 ± 0.2
10k	200 ± 30	5	1.0	198 ± 51	114 ± 30	0.9 ± 0.2
0.1M	200 ± 30	5	1.0	198 ± 29	37 ± 9	0.9 ± 0.2
0.1M	200 ± 30	25	1.1	202 ± 32	42 ± 10	1.0 ± 0.3
0.1M	200 ± 30	50	1.5	199 ± 34	58 ± 12	1.4 ± 0.3
1M	200	0	1	200 ± 4	12 ± 3	0.9 ± 0.2
1M	200 ± 30	5	1	201 ± 28	13 ± 4	1.0 ± 0.3
1M	200 ± 30	10	1.2	199 ± 27	15 ± 3	1.2 ± 0.3
1M	200 ± 30	20	1.7	196 ± 29	20 ± 5	1.5 ± 0.4
10M	200	0	1	200 ± 1	4.1 ± 0.8	1.0 ± 0.2
10M	200 ± 30	0	1	198 ± 30	3.8 ± 0.7	0.9 ± 0.2
10M	200 ± 30	3	1.2	199 ± 28	4.8 ± 1.0	1.1 ± 0.3
10M	200 ± 30	5	1.5	198 ± 29	6.1 ± 1.5	1.5 ± 0.4
10M	200 ± 30	10	2.4	199 ± 28	10 ± 2	2.5 ± 0.6
100M	200 ± 30	5	3.7	198 ± 28	4.8 ± 0.9	3.7 ± 0.8

Table 1: Estimated model parameters for several 3-year mortality simulations.

For simulated data, indicator k's range estimates always encompass true k. The estimated β captures both mean mortality and seasonal variations well, for populations P of 0.1M or more. Additional weekly variability ρ is estimated well for sufficiently large ρ or populations P, or simpler, for $k \geq \sqrt{2}$, when ρ exceeds regular Poisson noise. Below that, ρ is overestimated, mistaking some Poisson noise for additional weekly variability (Table 1 shows it is not caused by seasonal variation $\Delta\beta$, and, not shown, it is also not caused by yearly population growth).

3.2 The Netherlands and Rotterdam

The Netherlands is a country with a population of $P \approx$ 17M people, and Rotterdam is one of its cities with $P \approx$ 0.6M. Figure 2 shows mortality from 2010-2022 for The Netherlands, and 2019-2022 for Rotterdam due to data availability, excluding the first few weeks of 2019 due to the processing tail of filter F in (4) and (9). Estimated baselines $\beta(t)$ neatly follow the overall shape of mortality $m(t)$. In The Netherlands, mortality has several peaks for influenza (Jan 2018) and covid (Mar 2020 and several later). Rotterdam shows no easily visible events.

At all times, a weekly mortality variability is visible. In Rotterdam, the weekly variability appears stronger due to its smaller population, Poisson noise is then relatively stronger, see e.g. (7) bottom equation for $m(t)$, where $\beta(t)$ is *divided* by $P(t)$.

In The Netherlands, indicator $k(t)$ varies between 1 and 2 during years 2010-2019, with only one bump to 4 during the Jan 2018 influenza wave. Since 2020, k has changed behaviour, starting with a bump to 9 in the first covid wave. In Rotterdam, indicator $k(t)$ varies slightly around 1, and starts to vary slightly more halfway 2021.

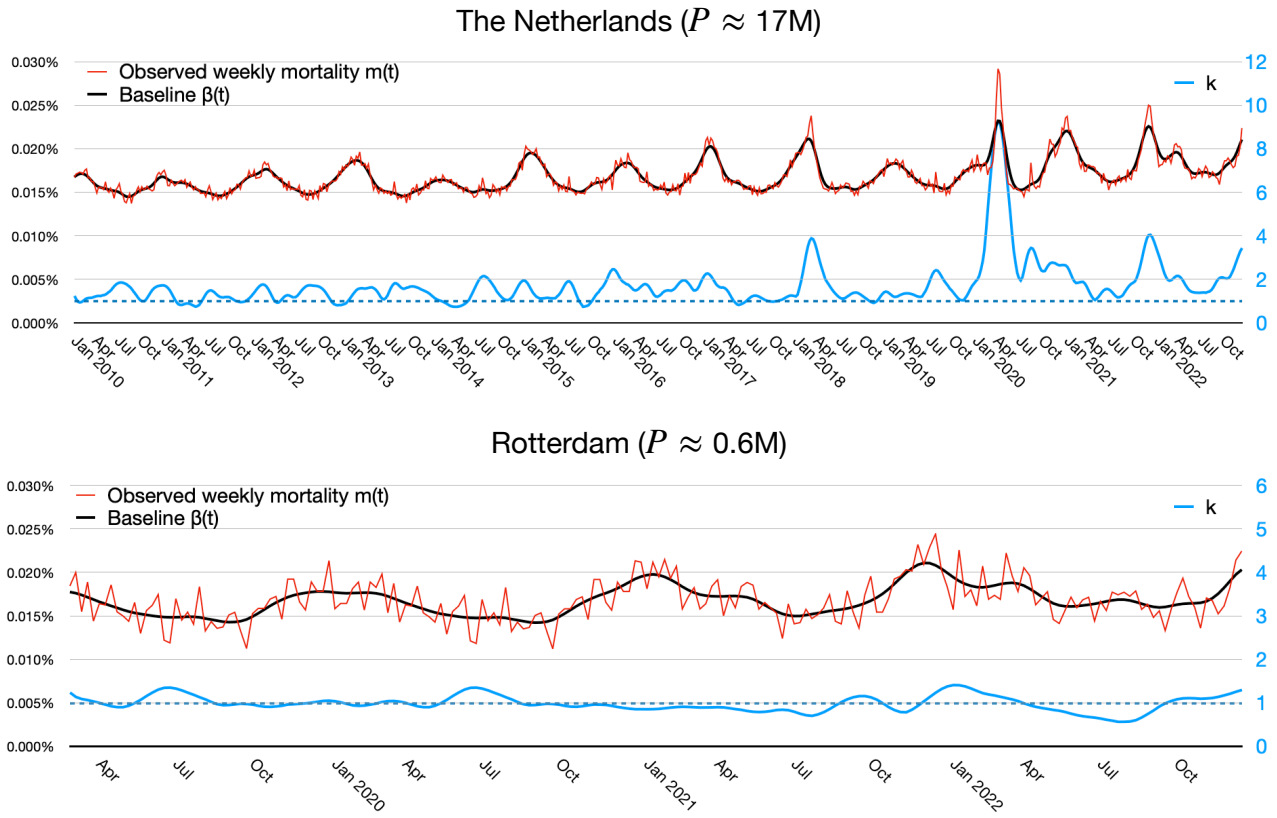

Figure 2: Weekly mortality in The Netherlands and its city Rotterdam, by observed $m(t)$, estimated baseline $\beta(t)$ and indicator $k(t)$. Note the different time scales due to data availability.

Table 2 shows statistics. In The Netherlands from 2010-2019, $k = 1.5 \pm 0.6$ and thus additional weekly variability was always present, with an average magnitude *exceeding* that of Poisson variability. From 2020 onwards, mortality baseline β grew by about 12%, and variability almost doubled to $k = 2.7 \pm 1.2$, substantially and systematically higher than before. The ratio ρ/β grew from approx. 3% to 5%. For Rotterdam, Table 2 shows no significant mortality events in 2020, with baseline mortality β rising only from 2021 onwards by about 8%.

Region	Population P	Time Period t	Baseline & seasons $\beta \cdot 10^6$	Indicator k	Additional variability $\rho \cdot 10^6$	ρ/β
Netherlands	17M	2010-2019	163 ± 13	1.5 ± 0.6	4.8 ± 1.8	2.9%
		2020-2022	185 ± 20	2.7 ± 1.2	8.9 ± 6.2	4.8%
Rotterdam	0.6M	2019	159 ± 12	1.1 ± 0.1	-	-
		2020	162 ± 15	1.0 ± 0.1	-	-
		2021	175 ± 19	0.9 ± 0.2	-	-
		2022	175 ± 13	1.0 ± 0.3	-	-

Table 2: Statistics for The Netherlands and its municipality Rotterdam. The population size of Rotterdam is insufficient to reliably estimate additional mortality variability ρ (according to $k < \sqrt{2}$).

3.3 Europe

Table 3 shows k's mean μ_k and deviation σ_k over time for 30 European countries in the period April 2017-December 2019, based on observed mortality from [Eur]:

$$\begin{aligned} \beta &\approx 198 \pm 31 \ \cdot 10^{-6} \\ \rho &\approx \ 7.2 \pm 2.0 \ \cdot 10^{-6} \\ \rho/\beta &\approx 3.3\% \pm 1.0\,\% \end{aligned} \tag{12}$$

Figure 3 illustrates the strong linear (Pearson) correlation of k^2 with population size P, and ρ with β. Despite the non-linearity of (10), the limited ranges in (12) lead to linear correlations. Note also that these correlations are illustrative; the temporal mean μ_k^2 of k^2, was used, and no weighting by population size was applied.

The take-away is that across European countries, there consistently is an additional weekly mortality variability with a magnitude of 2% to 4% times the mortality baseline.

Country	P	$\beta \cdot 10^6$	k	$\rho \cdot 10^6$	ρ/β
Austria	8.9M	175 ± 14	1.5 ± 0.5	6.5 ± 2.1	3.7%
Belgium	11.5M	181 ± 17	2.0 ± 0.9	8.0 ± 3.7	4.4%
Bulgaria	7.0M	293 ± 25	1.8 ± 0.7	11.5 ± 4.6	3.9%
Croatia	4.1M	243 ± 19	1.5 ± 0.4	11.8 ± 3.1	4.8%
Czechia	10.6M	199 ± 14	1.8 ± 0.7	7.7 ± 3.2	3.9%
Denmark	5.8M	178 ± 13	1.1 ± 0.3	-	-
Estonia	1.3M	222 ± 17	1.1 ± 0.2	-	-
Finland	5.5M	186 ± 12	1.2 ± 0.3	-	-
France	67.3M	172 ± 14	3.2 ± 1.2	5.2 ± 2.1	3.0%
Germany	83.0M	215 ± 18	4.8 ± 3.0	7.7 ± 5.2	3.6%
Greece	10.7M	217 ± 15	2.2 ± 1.0	9.9 ± 4.3	4.6%
Hungary	9.8M	252 ± 24	2.1 ± 0.6	10.6 ± 3.2	4.2%
Iceland	0.4M	121 ± 7	1.0 ± 0.3	-	-
Italy	59.8M	204 ± 19	3.9 ± 1.8	7.2 ± 3.4	3.5%
Latvia	1.9M	280 ± 23	1.2 ± 0.3	-	-
Liechtenstein	0.04M	130 ± 18	1.0 ± 0.2	-	-
Lithuania	2.8M	266 ± 22	1.4 ± 0.3	-	-
Luxembourg	0.6M	132 ± 12	1.1 ± 0.3	-	-
Malta	0.5M	139 ± 21	1.1 ± 0.3	-	-
Netherlands	17.3M	167 ± 15	1.6 ± 0.7	5.1 ± 2.5	3.1%
Norway	5.3M	145 ± 10	1.1 ± 0.2	-	-
Poland	38.0M	204 ± 15	2.8 ± 0.9	6.6 ± 2.0	3.2%
Portugal	10.3M	205 ± 27	2.0 ± 0.8	9.1 ± 3.8	4.4%
Romania	19.4M	255 ± 21	2.6 ± 0.7	9.2 ± 2.6	3.6%
Serbia	7.0M	275 ± 23	1.7 ± 0.4	11.0 ± 2.6	4.0%
Slovakia	5.5M	187 ± 14	1.5 ± 0.4	8.7 ± 2.8	4.7%
Slovenia	2.1M	186 ± 18	1.1 ± 0.2	-	-
Spain	46.9M	170 ± 19	2.9 ± 1.1	5.5 ± 2.4	3.3%
Sweden	10.2M	164 ± 13	1.5 ± 0.4	5.9 ± 1.8	3.6%
Switzerland	8.5M	149 ± 11	1.3 ± 0.3	-	-
Total	462M	198 ± 31	3.0 ± 1.1	7.2 ± 2.0	3.3% ± 1.0%

Table 3: Results for 30 European countries, period Apr 2017-Dec 2019. Totals are weighted by population size. The ρ is measured only for 18 countries with $k \geq \sqrt{2}$.

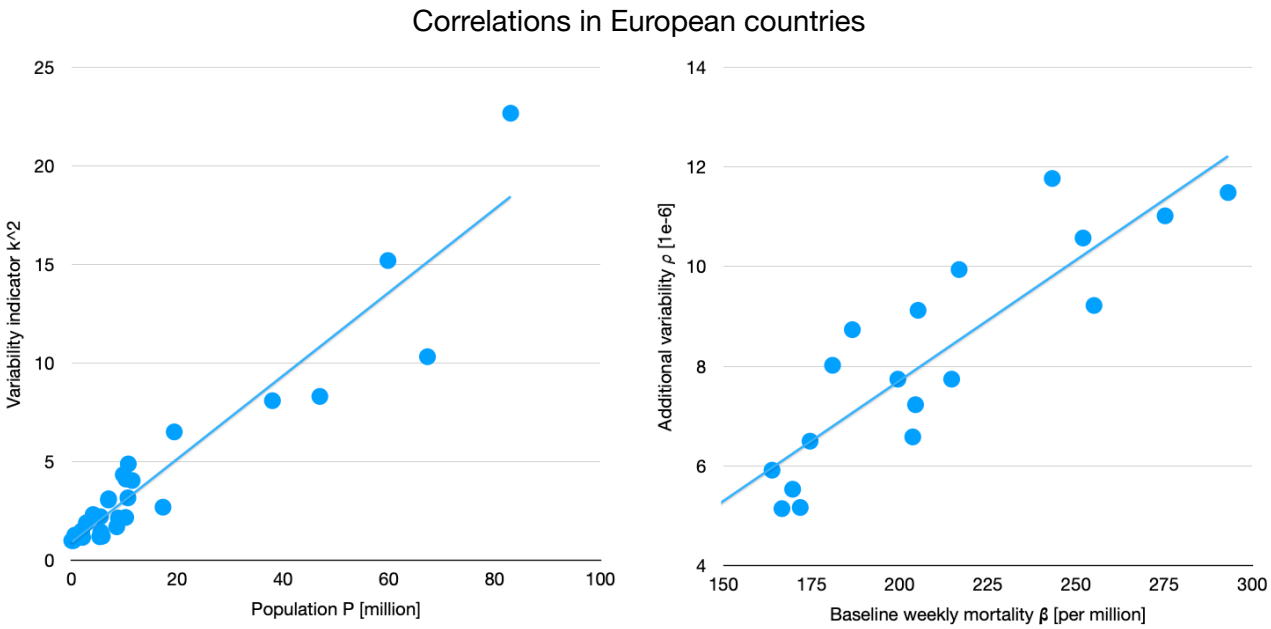

Figure 3: Correlations in European countries, illustrative, not weighted by population size. Indicator k^2 versus population size P (Pearson coefficient R = 95%, 30 countries), and additional mortality variability ρ versus baseline mortality β (with R = 87%, 18 countries with $k \geq \sqrt{2}$).

4 Conclusions

This report investigates the natural variability of mortality, which determines the thin line between excesses and normal variation within expectations. I propose a model for mortality's weekly variability based on a Poisson model, driven by potential non-stationary influences that act nation-wide and fast, on time scales of a week.

The results revealed the presence of a significant amount of non-stationary influences that add to mortality's weekly variability, with magnitude of 3%±1% (1% - 5% with 95% confidence) times baseline mortality, on top of the standard (Poisson) variability. This additional variability is consistently found across 30 European countries (462M people) during 2017-2019. A long-term analysis in The Netherlands (17M people) reveals the same variability between 2010-2019, with substantial increase since 2020 to approx. 5%. Mortality variance may thus very well itself be used as event indicator when variability is higher than expected.

The findings in this report are relevant for all models of mortality and its variability in general, and in particular for developments towards better excess mortality measurements. The additional variability found scales with both baseline mortality and population size in a different way compared to Poisson variability. For a typical mortality baseline of approx. 0.02% per week, the additional variability becomes dominant in populations above approximately 5M people.

A number of causes for the variability found were suggested, of which the most promising, temperature, will be investigated in a follow-up to this report. Finally, this study was limited in many ways, due to me, due to limited time as an independent researcher, and surely due to Nature's inaccessible magical side [Bre].

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