5. Appendix

A 'real world' problem

In the model of *epidemic spread* presented here, we consider strictly positive C^∞-functions on a time interval $\mathcal{I} := [\,0\,,\,T\,[\,,\ T \in \mathbb{R}_+ \cup \{+\infty\}$. Remember these functions form a convex cone and a multiplicative group $\mathcal{C} \subset C^\infty(\mathcal{I})$ and $\mathcal{C}_1 = \{\, f \in \mathcal{C} \,|\, f(0) = 1 \,\}$ is a convex subgroup of $\mathcal{C}$. Also, it is easily seen that

$$E \circ J \equiv Id_{\mathcal{C}} \quad \textit{and} \quad J \circ E \restriction \mathcal{C}_1 \equiv Id_{\mathcal{C}_1} \qquad [\text{ cf. (4.1) }]\,, \tag{A.1}$$

where $E : \mathcal{C} \to \mathcal{C}$ and $J : \mathcal{C} \to \mathcal{C}_1$, respectively, are the mappings

$$Ef_t \,=\, \exp \frac{d}{ds} \log f(s)\,\big|_t \quad \text{and} \quad Jf_t \,=\, \exp \int_0^t \log f(s)\, ds\,. \tag{A.2}$$

Remember also that the exponential integral and the exponential mean of f over $[\,a\,,\,b\,] \subset \mathcal{I}$ equal

$$Jf\,|_a^b = \exp \int_a^b \log f(s)\, ds \quad \text{and} \quad \bar{f}_{[a,b]} \,=\, \exp \frac{1}{b-a} \int_a^b \log f(s)\, ds\,, \quad \text{respectively.} \tag{A.3}$$

For simplicity, the *herd size* is normalized to one in the following. The non-susceptible ('immune') fraction of the herd is represented by a function $h : \mathcal{I} \to [\,0\,,\,1\,]$ (0: no immune members, 1: total herd immunity). More precisely, $h(t)$ is defined as the *unsusceptible* fraction, comprising the immune members, but also those who are currently sick, and those who have died, i.e. $h(t) = 1 - s(t)$, $s(t)$ denoting the susceptible fraction. (Note that in our model, $h(t) = 1 - s(t)$ is *not equal* to the sum $i(t)+r(t)$ of the infected and recovered fractions, as is the case for example in the SIR model, cf. [SM], since we do not require that recovered members remain immune.)

Finally, the *incidence*, defined as the fraction of *symptomatic* ('acute') new infections per time, is represented by the function $u : \mathcal{I} \to \mathbb{R}_+$.

The *reproduction factor* indicates how many members are infected on average by one contagious member. By itself, it says thus nothing about the underlying time scale. Yet time enters naturally with the *transmission time* T_{tr} which is the average time during that one sick member infects R susceptible ones. Then obviously $u(\,t + T_{tr}\,) \,=\, R \cdot u(\,t\,)$ and we can state the following

Proposition 1 (Reproduction factors)
With Eu_t as above, the reproduction factor, usually defined as $R := \frac{u(\,t+T_{tr}\,)}{u(\,t\,)}$, equals:

$$R \;=\; (\,\bar{Eu}_{[t,t+T_{tr}]}\,)^{T_{tr}}, \tag{A.4}$$

i.e. $R^{\frac{1}{T_{tr}}}$ is the *exponential mean* of Eu over the transmission interval $[\,t,\,t+T_{tr}\,]$.

Proof : Immediate consequence of Theorem 3 (T3-3).

Remarks

1. Since $Eu : t \mapsto Eu_t$ is continuous, there always exists a time $\tau \in [\,t\,,\,t+T_{tr}\,]$, such that $Eu_\tau \,=\, \bar{Eu}_{[t,t+T_{tr}]}$, and hence, due to (A.4),

$$R^{\frac{1}{T_{tr}}} \,=\, Eu_\tau \tag{A.5}$$

2. Trivially, the transmission time T_{tr} introduces a *natural time scale* in which T_{tr} is the unit of time. Hence in this scale, for a certain $\tau \in [\,t\,,\,t+1\,]$,

$$R \,=\, Eu_\tau\,. \tag{A.6}$$

From now on we work in this natural time scale, i.e. with $T_{tr} = 1$. Moreover, we identify $R = R_t$ with $Eu_t = \exp \frac{d}{dt} \log u(t)$, which amounts to refining the definition of R by replacing the exponential mean of Eu over a transmission cycle by its *current* value at time t. This exactly is summarized in the following

Proposition 2 . In the natural time scale (in which $T_{tr} = 1$), the reproduction factor is the exponential derivative of the incidence.

Every mathematical model makes simplifications. An essential assumption of ours is a *homogeneous environment*. This means, to put it simply, that the pathogene finds the same conditions for spreading throughout the herd. In particular, singular spreading events or new infections from outside are neglected. As far as these conditions are met, the essence of the disease spread is captured by the following *model equations*, which relate the immune and fatal fractions to the incidence

$$h(t) \;=\; h(0) \;+\; w_i \int_0^t \exp\,(\frac{\tau - t}{T_i})\, u(\tau)\, d\tau \;+\; w_f \int_0^t u(\tau)\, d\tau \;, \qquad \text{(A.7)}$$

$$\text{with}\, w_i \;=\; \frac{B}{100 - A} \quad \text{and}\, w_f \;=\; \frac{C}{100 - A}\,.$$

The first integral represents the recovered uninfectable ('steril') fraction of the herd and the second integral stands for the members that died up to time t. The decay time constant T_i limits the mean duration of immunity after infection, and the weighting factors w_i and w_f introduce the percentage A of the *asymptomatic* ('silent') cases, B of *steril-immunized* cases, and C of *fatal* cases into the equation (so if all infected survivors become non-infectiously immune, then obviously $B + C = 100$).

Under the assumed homogeneous conditions, the exponential derivative of the incidence is directly proportional to the susceptibility. By virtue of proposition 2, this means

$$\begin{aligned} R_t \;&=\; Eu_t \;=\; s(t)\, Eu_0 \;=\; R_0\,[\,1 \,-\, h(t)\,] \\ &=\; R_0\,[\,1 - h(0) - w_i \int_0^t \exp\,(\frac{\tau - t}{T_i})\, u(\tau)\, d\tau \;-\; w_f \int_0^t u(\tau)\, d\tau\,]\,, \end{aligned} \qquad \text{(A.8)}$$

and so the incidence becomes due to (A.1), (EI), and (A.8)

$$\begin{aligned} u(t) \;&=\; u(0)\; J\, Eu_t \;=\; u(0)\, \exp \int_0^t \log Eu_\tau\, d\tau \\ &\;\overset{\text{cf. (A.1)}}{=}\; u(0)\; \exp \int_0^t \log\{\, R_0\,[\,1 - h(0) - w_i \int_0^\tau \exp\,(\frac{\theta - \tau}{T_i})\, u(\theta)\, d\theta \;-\; w_f \int_0^\tau u(\theta)\, d\theta\,]\,\}\; d\tau\,, \end{aligned} \qquad \text{(A.9)}$$

where $u(0)$, $h(0)$ and R_0 represent the initial incidence, the initial herd infection, and the reproduction factor at time $t = 0$, respectively.

Equation (A.9) describes the development of an epidemic under homogeneous conditions. Other elements, such as a vaccination rate, are easily introduced into equations (A.7-9).

The exorbitant dynamics that an epidemic can develop at higher reproduction factors is also reflected in the derivative of $u(t)$, which follows directly from (A.9)

$$\frac{du}{dt}\Big|_t \;=\; u(t)\, \log\{\, R_0\,[\,1 - h(0) \,-\, w_i \int_0^t \exp\,(\frac{\tau - t}{T_i})\, u(\tau)\, d\tau \,-\, w_f \int_0^t u(\tau)\, d\tau\,]\,\}\,. \qquad \text{(A.10)}$$

We have integrated equation (A.9) for basic reproduction factors varied from $R_0 = 0.5$ to $R_0 = 3.0$ using a numerical routine implemented in the AMADEUS program, cf. [Hei]. The initial conditions are $h(0) = 0$ and $u(0) = 10^{-5}$ (in common parlance: 'incidence one', i.e. one sick person in a population of 100.000), and we assume $T_i = \infty$ and $B + C = 100$ percent, that is every surviving infected member ends up permanently immune. We also allow $A = 20$ percent asymptomatic cases.

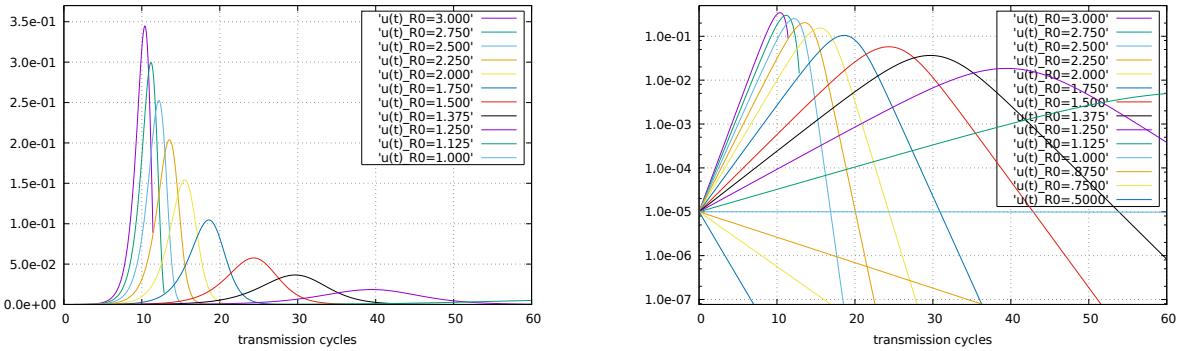

Fig. 1. *The incidence*

The graphics give an idea of what is happening if an epidemic with higher reproduction factor is let loose, i.e. if it is not contained by suitable measures. For $R_0 \gtrsim 2.7$, a considerable part of the population would get simultaneously sick at the peak.

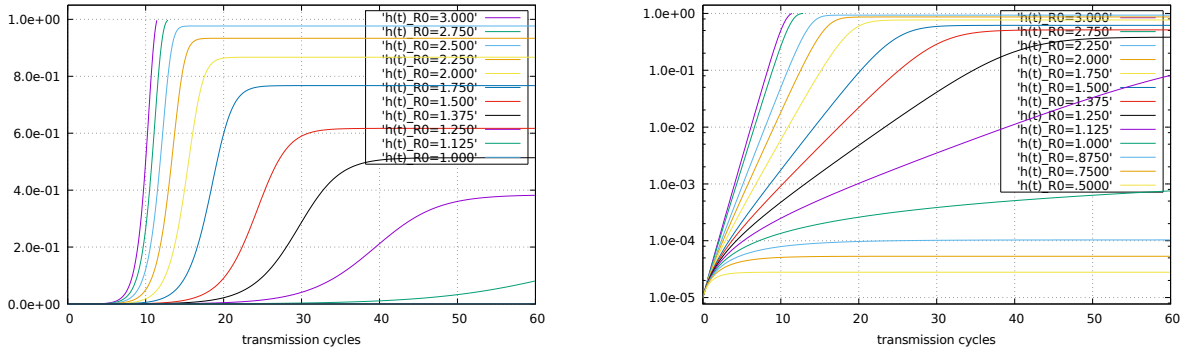

Fig. 2. *The herd infection*

With initially $R_0 \gtrsim 2.7$ and without further mesures, everybody would eventually go through the infection, be it alive or dead. The entire population would end up immune (the cuts in the two upper curves indicate when 100 % herd immunity is attained). Assuming a mortality rate of 0.2 %, for example, in a population of 82 million, as in Germany, more than 160,000 people would die. Under such conditions one can only hope for any effective vaccination, otherwise the reproduction factor would have to be kept low with painful containment measures.

6. Conclusions and outlook

The exponential derivative of a differentiable path $c : \mathbb{J} \to G$ in a Lie group has been studied and it was demonstrated how it acts *within* the group through right or left multiplication and in this way provides an *intrinsic* description of the infinitesimal change, in contrast to the classical differential that acts in *tangent* space.

Basic properties have been outlined and calculation rules derived. The elementary case of the multiplicative group $G = \mathbb{R}_+$ together with the smooth strictly positive functions $f : \mathbb{J} \to \mathbb{R}_+$ was treated in more detail, and applied to a numerical model of epidemic spread.

We are particularly interested in novel methods and applications in dynamic systems with many interacting components. The general framework in finite or infinite dimensional Lie groups might be of interest here. But this has to be left to further studies.

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