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Why stochastics? And why in Banach spaces? – Epidemic example

Ivan Yaroslavtsev was awarded the Stieltjes Prize for the academic year 2018–2019. His PhD thesis entitled *Martingales and stochastic calculus in Banach spaces* was done under supervision of Mark Veraar and Jan van Neerven in Delft and it was devoted to exploring new connections between stochastic analysis and geometry of Banach spaces. This is a very classical topic for the Delft Analysis group, which is interested in approaches solving stochastic PDEs, and many fundamental results in this direction have been obtained earlier by Mark and Jan (e.g., the theory of Wiener stochastic integrals in Banach spaces). During his PhD, Ivan Yaroslavtsev used to further extend this direction by working with general martingales. In particular, he discovered certain martingale decompositions, stochastic integral inequalities, Burkholder–Davis–Gundy inequalities, and weak differential subordination inequalities, all of which turned out to hold only for martingales with values in Banach spaces having the so-called UMD property. All this work was done both solely and in collaboration. Currently, Ivan Yaroslavtsev is doing his postdoc at Max Planck Institute for Mathematics in the Sciences (Leipzig, Germany) and his research is focused on stochastic dynamical systems.

Stochastic processes appear as an inherent important ingredient for many models. The reader can find a vast literature on applications of stochastics in various fields of physics, mechanics, economics, biology, et cetera., see, e.g., [2,4]. Our goal here is not to describe all of these applications; instead we are going to focus on a currently more substantial topic as an illustration of stochastic approach: epidemics.

Assume that there is an epidemic of a certain disease spreading over a fixed population. (Poor reader, if you find the infection topic too dramatic, please presume that this 'disease' is nothing but the Pokémon Go game downloads or a number of gummy bears sold.) In this case we are definitely interested in predicting the number of infected people or, say, the infected area. Fortunately there have been done a lot in this direction during the past century, and for instance we can exploit the so-called *SIR model* (Susceptible–Infected–Recovered model), described, e.g., in [15, Section 2.4 and 3.3]. In this model we have three functions over time: S, the number of healthy people in the population, which still did not have the disease; I, the number of infected people; and R, the number of recovered people, which now have an immunity and so will not be infected again. (The model has many branches exploited in various applications, see, e.g., [15], but for now such a simplified form is more than enough.) We assume that nobody is born and nobody dies, so $S+I+R=N$ is a constant and for a discrete time line (representing for example the number of days after the disease appearance) according to the SIR model we will have the following recurrence

$$S_{n+1}=S_n-\frac{\beta S_n I_n}{N},\quad I_{n+1}=I_n+\frac{\beta S_n I_n}{N}-\gamma I_n,\quad R_{n+1}=R_n+\gamma I_n,$$

where β interprets the infectivity chance and γ stands for the recovery chance. Now, we will simplify this model a bit by supposing


Photo: Anna Popova
Ivan Yaroslavtsev (on the left side)

that $S \approx N \approx \infty$ (i.e. the number of infected people is still pretty low comparing to the total population, this seems to hold, e.g., for HIV, SARS-CoV-1, MERS, et cetera), so we will have only

$$I_{n+1} = I_n + (\beta - \gamma) I_n. \tag{1}$$

Equation (1), though being useful for a small extremely densely populated society, does not tell us what happens if we have a big territory over which the infection spreads so the area effects start playing a role. Now we are going to make our model — hopefully — more realistic which would reflect such a setting.

1. We assume that we have a bounded domain $\mathcal{O} \subset \mathbb{R}^2$ (designating the area where the disease spreads) and a process $I(t,x)$ corresponding to the *infection density* at point $x \in \mathcal{O}$ in time $t \geq 0$.
2. We assume that the infection spreads not only in time, but in space, and we will use a special operator $\Delta = \frac{\partial^2}{\partial x_1^2} + \frac{\partial^2}{\partial x_2^2}$, also known as the *Laplacian*, in order to emulate this evolution. This operator generates the *heat semigroup* (see, e.g., (8)), which is used in particular for modelling how the temperature of an object distributes in time, so such an operator perfectly fits in our setting (see, e.g., [10]). The impact of the Laplacian in the disease spreading will be controlled by a constant $\lambda \geq 0$.
3. Our recovery chance γ seems to depend on I in reality: when I is small γ is a constant smaller than β, after getting a certain level of I we have a maximum of γ (infected people get a better help as the infection is noticed), and finally, when I is getting really big, γ again falls down to the almost initial level as the capacities of the hospitals are limited. (Note that in reality γ seems to depend on time as well. Indeed, as I is big, more pharmacy companies and research institutes work on the possible vaccines or treatments. But as such breakthroughs in treatment come discretely in time, one can assume that the research impact in γ is governed by a Poisson process with the intensity depending on I. Though such a model seems to be more precise, we will not consider it here due to technical complications.)
4. The same corresponds to β: it is quite big for small I, but when I is getting bigger, β is becoming smaller, as there are less healthy people around and quarantine measures could be undertaken. To sum up, we can assume that $\zeta = \beta - \gamma$ is a Lipschitz bounded function of I ($|\zeta(x) - \zeta(y)| \leq C|x-y|$ for some $C > 0$) so that
$$\zeta(0) > 0, \quad -\infty < \lim_{x \to \infty} \zeta(x) < 0$$
5. There are always randomly appearing fluctuations, disturbing our model. We will assume here that these fluctuations are generated by a *Wiener process* W_t depending on time. (Highly likely such oscillations depend on space as well, but for simplicity we will not consider space-time white noise.) If the reader is not acquainted with such a notion, please imagine a trajectory of a molecule in the air.
6. Sometimes researches discover new diagnosing methods, or it could be a dramatic change in the political situation, and the number of infected people drastically jumps. Or the other way around, a revolutionary treatment is found, and the infected number drops down. Nobody knows, when such changes happen. It is completely unpredictable, but everybody expects, that such events will occur with a certain chance. Fortunately mathematics can model such phenomena with a *Poisson process* P_t: a process, which has jumps of size 1 happening at sudden random times which distribution is exponential, i.e.
$$\mathbb{P}(P_t - P_s = n) = \frac{(t-s)^n}{n!} e^{-(t-s)}, \quad n \geq 0, t \geq s.$$
(The *intensity* of P_t, i.e. the measure which indicates how many jumps happen in a particular time segment, which is defined by $\nu[s,t] := \mathbb{E}P_t - P_s$, could also depend on I. Nonetheless, here we assume that ν is Lebesgue.)
7. People do travel! Therefore we assume that our domain is divided into n 'clusters' $A_1, \ldots, A_n$: within such a cluster every visiting infected 'tourist' increases the infection density by a constant. (It must not be specifically a tourist, but any traveler within the population.) As again we have no idea when such a traveler comes, the corresponding impact can be modeled by independent Poisson processes $P^1, \ldots, P^n$ and the coefficients $\gamma_1(I(t)), \ldots, \gamma_n(I(t))$, where we consider $I(t)$ as a function $x \mapsto I(t,x)$ on the whole $\mathcal{O}$ so that $\gamma_1(I(t)), \ldots, \gamma_n(I(t))$, e.g., can have the following form:
$$\gamma_k(I(t)) = \psi_k\Big(\int_{\mathcal{O}} I(t,x)\,dx\Big), \quad k = 1, \ldots, n, \tag{2}$$
with $\psi_k(x)$ being of the order $\min\{1, x^{-1}\}$, so when the total infected number is small, each traveler infects about one-two persons, but when I is big, each traveler contacts less and hence infects less. (The intensity of these Poisson processes could depend on I as well due to travel restrictions acting when I is big.)

Recurrence (1) thus transforms to the following *stochastic partial differential equation*:

$$\begin{cases} \mathrm{d}I(t,x) = [\lambda \Delta I(t,x) + \zeta(I(t,x))I(t,x)]\mathrm{d}t + aI(t,x)\,\mathrm{d}W_t + bI(t,x)\,\mathrm{d}P_t \\ \qquad + \sum_{k=1}^{n} \mathbf{1}_{A_k}(x)\gamma_k(I(t))\,\mathrm{d}P_t^k, \quad t \geq 0, x \in \mathcal{O}, \\ I(0,x) = I_0(x), \quad x \in \mathcal{O}, \end{cases} \tag{3}$$

where $\mathrm{d}I$, $\mathrm{d}t$, $\mathrm{d}W$, $\mathrm{d}P$, and $\mathrm{d}P^k$ are *differentials*, which could be imagined as 'small changes of I, t, W, P, and P^k' respectively, a indicates how big is the impact of random fluctuations, b represents the dependence on P_t, $\mathbf{1}_{A_k}(x)$ is an indicator function of the cluster A_k which equals 1 when $x \in A_k$ and 0 when $x \notin A_k$, and the function I_0 is the distribution of the disease in the very beginning, which typically can be assumed as an indicator function of a subset of $\mathcal{O}$ (see for example Figure 1).

How can we understand that the epidemic is getting serious? Very often we tend to evaluate the reality by just one number, which is very convenient as we come up with the decision depending on how this number is big or small.

In particular, whatever positive functions f and g on $\mathcal{O}$ representing two possible infection densities are, we need to choose a parameter $\varkappa$ such that

f is worse then g if and only if $\varkappa(f) > \varkappa(g)$,

so we somehow order possible infection distributions. There are two obvious choices of such a parameter: either $\varkappa$ stands for the total number of all infected people, i.e. $\varkappa(I) = \varkappa_1(I) := \int_{\mathcal{O}} I(x)\,\mathrm{d}x$, or $\varkappa$ corresponds to the density maximum, i.e. $\varkappa(I) = \varkappa_\infty(I) := \max_{x \in \mathcal{O}} I(x)$. Both approaches have their disadvantages: in the first case $\varkappa(I)$

can be incredibly big, though I can be smudged over the domain, so the infection is still not dangerous; in the second case there might be an extremely high (even infinite) pick in one point but there could be no infection outside a small surrounding of this point, and there is no need for panic yet. It turns out that there is a whole zoo of parameters which continuously interpolates between the two cases discussed above, the so-called L^p-*norms*:

$$\|f\|_p := \Big(\int_O |f(x)|^p \,\mathrm{d}x\Big)^{1/p}.$$

Notice how wonderful they are! First, they have the promised limits:

$$\|f\|_p \to \int_O f(x)\,\mathrm{d}x, \quad p\to 1, \qquad \|f\|_p \to \max_{x\in O} f(x), \quad p\to\infty.$$

Second, $\|f\|_p$ combines together caring about the infection area, as $\varkappa_1$, and taking huge picks into account, as $\varkappa_\infty$, and this dependence changes as p varies. Do you mind more about picks? Increase p! Does the infection area play a major role? Simply drop p down.

Finally, $\|f\|_p$ is a *norm*, i.e. a nonnegative function acting on functions which satisfies the following three properties:

1. *triangle inequality*: $\|f+g\|_p \le \|f\|_p + \|g\|_p$,
2. *homogeneity*: $\|af\|_p = |a|\,\|f\|_p$ for any $a\in\mathbb{R}$,
3. *positivity*: $\|f\|_p = 0$ if and only if $f=0$.

(One can think of a norm as of a *distance function*.) We will call the space of all functions f with $\|f\|_p<\infty$ the L^p *space* and denote it by $L^p(O)$. This space is a *Banach space* (i.e. a linear space with a norm which is complete, see, e.g., [11]).

Thanks to the discussion above from now on we are interested only in $\|I(t)\|_p$. Fix $2\le p<\infty$ (we omit the case of $p=\infty$ as sometimes L^∞ spaces are terrible to work with, see, e.g., [8, Example 6.1.18] and [7,8] in general, and the case of $p<2$ in order to avoid certain technical difficulties appearing later), and consider the equation (3) not pointwise in $x\in O$, but as an equation on processes *with values in* $L^p(O)$, i.e.

$$\begin{cases} \mathrm{d}I(t) = [\lambda\Delta I(t) + \alpha(I(t))]\mathrm{d}t + aI(t)\,\mathrm{d}W_t + bI(t)\,\mathrm{d}P_t \\ \qquad\qquad + \sum_{k=1}^n \mathbf{1}_{A_k}\gamma_k(I(t))\,\mathrm{d}P_t^k, \quad t\ge 0, \\ I(0) = I_0 \in L^p(O), \end{cases} \tag{4}$$

where α is a Lipschitz function which acts *from* $L^p(O)$ *to itself* and $\gamma_1,\dots,\gamma_n$ are Lipschitz functions acting from $L^p(O)$ to $\mathbb{R}$, i.e. there exist positive constants C_α and C_γ such that for all f and g from $L^p(O)$,

$$\|\alpha(f)-\alpha(g)\|_p \le C_\alpha \|f-g\|_p, \tag{5}$$

$$|\gamma_k(f)-\gamma_k(g)| \le C_\gamma\|f-g\|_p \quad \text{for any } k=1,\dots,n. \tag{6}$$

Exercise 1. Prove (5) and (6) given ζ is Lipschitz and assuming that γ_k is defined by (2) and the corresponding ψ_k is Lipschitz.

Now the question is: *Does* (4) *make sense?* Paper is patient, and one can write down any equation they want. But will this equation have a solution? In which sense? And is it unique, so that basing on it one can try to make forecasts?

Fortunately for us, there has been developed a massive amount of work concerning stochastic equations with values in Banach spaces. My point here is not to make an overview over all these results, but to demonstrate, so we will fix one approach, which provides us with the so-called *mild solution* (see, e.g., [3,9] and references therein):

$$\begin{aligned} I(t) &= S(\lambda t)I_0 + \int_0^t S(\lambda t-\lambda s)\,\alpha(I(s))\,\mathrm{d}s \\ &\quad + a\int_0^t S(\lambda t-\lambda s)I(s)\,\mathrm{d}W_s + b\int_0^t S(\lambda t-\lambda s)I(s)\,\mathrm{d}P_s \\ &\quad + \sum_{k=1}^n \int_0^t S(\lambda t-\lambda s)\mathbf{1}_{A_k}\gamma_k(I(s))\,\mathrm{d}P_s^k, \quad t\ge 0. \end{aligned} \tag{7}$$

Here $(S(t))_{t\ge0}$ denotes the *heat semigroup*, a family of linear maps on $L^p(O)$ which is a semigroup (i.e. $S(t+s)f = S(t)S(s)f$ for any $f\in L^p(O)$) and which is generated by the Laplacian, i.e. for any smooth $f\in L^p(O)$,

$$\lim_{\varepsilon\to0}\frac{S(t+\varepsilon)f - S(t)f}{\varepsilon} = \Delta S(t)f, \quad t\ge 0$$

(so the Laplacian is the 'derivative' of $S(t)$ in t). For example, if $O=\mathbb{R}^2$, then $S(t)$ has the following form:

$$S(t)f(x) = \frac{1}{4\pi t}\int_{\mathbb{R}^2} f(y)\,e^{-|x-y|^2/4t}\,\mathrm{d}y, \quad x\in\mathbb{R}^2. \tag{8}$$

The key tool we are going to exploit in proving that there is only one process $I(t)$ satisfying (7) is the magic *fixed point argument* (aka the *Schauder theorem*):

Theorem 1. *Let X be a Banach space with a norm $\|\cdot\|$. Assume that there is a function ϕ from X to itself and a constant $C\in[0,1)$ such that*

$$\|\phi(x)-\phi(y)\| \le C\|x-y\| \quad \textit{for any } x,y\in X. \tag{9}$$

Then there exists a unique $x\in X$ satisfying $\phi(x)=x$.

In our setting, let X be a space of all $L^p(O)$-valued processes on $[0,T]$ starting in I_0 and let ϕ be defined by

$$\begin{aligned} \phi(I)(t) &= S(\lambda t)I_0 + \int_0^t S(t-s)\,\alpha(I(s))\,\mathrm{d}s \\ &\quad + a\int_0^t S(\lambda t-\lambda s)I(s)\,\mathrm{d}W_s + b\int_0^t S(\lambda t-\lambda s)I(s)\,\mathrm{d}P_s \\ &\quad + \sum_{k=1}^n \int_0^t S(\lambda t-\lambda s)\mathbf{1}_{A_k}\gamma_k(I(s))\,\mathrm{d}P_s^k, \quad t\in[0,T]. \end{aligned} \tag{10}$$

(Such X is not a Banach space, but one can represent this as a Banach space by subtracting I_0 and setting the norm by (11).) Our aim is to find such $\|\cdot\|$ and T that (9) is satisfied (then we immediately get existence and uniqueness of I with $\phi(I)=I$, so the same would follow for the solution of the integral equation (7) on $[0,T]$!). Luckily we know the proper $\|\cdot\|$ in advance, so we choose it to be

$$\|I\|_{p,T} := \mathbb{E}\sup_{0\le t\le T}\|I(t)\|_p, \tag{11}$$

where $\mathbb{E}\xi$ means the *expectation* of a random variable ξ, i.e. the mean (or the integral) of all the values of ξ over all the randomness created by W and P (see, e.g., [1,12]).

Exercise 2. Prove that $\|\cdot\|_{p,T}$ is a norm. *Hint: think of $\mathbb{E}$ as of an integral.*

Now let us check the assumptions of Theorem 1. Let F and G be two processes with values in $L^p(O)$ such that $F(0)=G(0)=I_0$. Then by (10), (11), and by the triangle inequality

$$\begin{aligned}\|\phi(F)-\phi(G)\|_{p,T} &\leq \mathbb{E}\sup_{0\leq t\leq T}\|S(\lambda t)F(0)-S(\lambda t)G(0)\|_p\\ &+\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(\lambda t-\lambda s)(\alpha(F(s))-\alpha(G(s)))\,\mathrm{d}s\right\|_p\\ &+|a|\,\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(\lambda t-\lambda s)(F(s)-G(s))\,\mathrm{d}W_s\right\|_p\\ &+|b|\,\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(\lambda t-\lambda s)(F(s)-G(s))\,\mathrm{d}P_s\right\|_p\\ &+\sum_{k=1}^n\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(\lambda t-\lambda s)\mathbf{1}_{A_k}(\gamma_k(F(s))-\gamma_k(G(s)))\,\mathrm{d}P_s^k\right\|_p.\end{aligned}\tag{12}$$

Let us now estimate each part of the right-hand side of (12). First note that $S(t)$ is linear, consequently

$$\begin{aligned}S(\lambda t)F(0)-S(\lambda t)G(0)&=S(\lambda t)(F(0)-G(0))\\&=S(\lambda t)(I_0-I_0)=S(\lambda t)0=0.\end{aligned}$$

Thus

$$\mathbb{E}\sup_{0\leq t\leq T}\|S(\lambda t)F(0)-S(\lambda t)G(0)\|_p=\mathbb{E}\sup_{0\leq t\leq T}0=0.$$

Now note that $S(t)$ is a *contraction* on $L^p(O)$ for any $t\geq 0$, i.e.

$$\|S(t)f\|_p\leq\|f\|_p\tag{13}$$

(we leave this as an exercise). Therefore by the triangle inequality (now applied for an integral)

$$\begin{aligned}&\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(\lambda t-\lambda s)(\alpha(F(s))-\alpha(G(s)))\,\mathrm{d}s\right\|_p\\ &\leq\mathbb{E}\sup_{0\leq t\leq T}\int_0^t\|S(\lambda t-\lambda s)(\alpha(F(s))-\alpha(G(s)))\|_p\mathrm{d}s\\ &\leq\mathbb{E}\sup_{0\leq t\leq T}\int_0^t\|\alpha(F(s))-\alpha(G(s))\|_p\mathrm{d}s\\ &\overset{(*)}{\leq}C_\alpha\mathbb{E}\sup_{0\leq t\leq T}\int_0^t\|(F(s))-(G(s))\|_p\mathrm{d}s\\ &\overset{(**)}{\leq}C_\alpha\mathbb{E}\int_0^T\|(F(s))-(G(s))\|_p\mathrm{d}s\leq C_\alpha T\|F-G\|_{p,T},\end{aligned}$$

where for $(*)$ we used (5) and $(**)$ follows from the fact that any norm is nonnegative.

Next, let us move to the stochastic integral with respect to the Brownian motion. First note that thanks to the so-called *dilation trick* shown in [6] by Fröhlich and Weis we can think of $(S(t))_{t\geq 0}$ as of a *group*, not just a semigroup (though this group is different from $S(t)$ and acts on a different L^p space, but this does not play a big role for us here in this illustration) so that $S(t-s)=S(t)S(-s)$. Therefore in particular by (13) (though such a dilated $S(t)$ does not act on $L^p(O)$, it does have a bounded norm, see [6]) and by the linearity of $S(t)$,

$$\begin{aligned}&\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(\lambda t-\lambda s)(F(s)-G(s))\,\mathrm{d}W_s\right\|_p\\ &=\mathbb{E}\sup_{0\leq t\leq T}\left\|S(\lambda t)\int_0^t S(-\lambda s)(F(s)-G(s))\,\mathrm{d}W_s\right\|_p\\ &\leq\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(-\lambda s)(F(s)-G(s))\,\mathrm{d}W_s\right\|_p.\end{aligned}\tag{14}$$

Now, as we have a proper stochastic integral (we call it *proper* as it has the form $\int F(s)\,\mathrm{d}W_s$ but not $\int F(t,s)\,\mathrm{d}W_s$, so it is a *local martingale*, see, e.g., [13]), thanks to the work [13] of van Neerven, Veraar, and Weis on stochastic integration in Banach spaces (see also [14]) and using (13) we can estimate (14) in the following way exploiting the *square function*:

$$\begin{aligned}&\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(-\lambda s)(F(s)-G(s))\,\mathrm{d}W_s\right\|_p\\ &\leq C_p\mathbb{E}\sup_{0\leq t\leq T}\left\|\left(\int_0^t|S(-\lambda s)(F(s)-G(s))|^2\mathrm{d}s\right)^{1/2}\right\|_p\\ &=C_p\left\|\left(\int_0^T|S(-\lambda s)(F(s)-G(s))|^2\mathrm{d}s\right)^{1/2}\right\|_p,\end{aligned}$$

for some fixed constant C_p, where the latter integrals are taken pointwise in O. By Hölder's inequality, the Fubini theorem, and (13) we get

$$\begin{aligned}&\left\|\left(\int_0^T|S(-\lambda s)(F(s)-G(s))|^2\mathrm{d}s\right)^{1/2}\right\|_p\\ &\leq\mathbb{E}\left\|T^{\frac{p-2}{2p}}\left(\int_0^T|S(-\lambda s)(F(s)-G(s))|^p\mathrm{d}s\right)^{1/p}\right\|_p\\ &=T^{\frac{p-2}{2p}}\mathbb{E}\left\|\left(\int_0^T|S(-\lambda s)(F(s)-G(s))|^p\mathrm{d}s\right)^{1/p}\right\|_p\\ &=T^{\frac{p-2}{2p}}\mathbb{E}\left(\int_0^T\|S(-\lambda s)(F(s)-G(s))\|_p^p\mathrm{d}s\right)^{1/p}\\ &\leq T^{\frac{p-2}{2p}}\mathbb{E}\left(\int_0^T\|F(s)-G(s)\|_p^p\mathrm{d}s\right)^{1/p}\\ &\leq T^{\frac{p-2}{2p}}T^{\frac{1}{p}}\|F-G\|_{p,T}=T^{\frac{1}{2}}\|F-G\|_{p,T}.\end{aligned}$$

Consequently, if we sum everything up

$$\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(\lambda t-\lambda s)(F(s)-G(s))\,\mathrm{d}W_s\right\|_p\leq C_pT^{\frac{1}{2}}\|F-G\|_{p,T}.$$

Now we move to our last Poisson ingredients of (12). First similarly to (14) we notice that

$$\begin{aligned}&\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(\lambda t-\lambda s)(F(s)-G(s))\,\mathrm{d}P_s\right\|_p\\ &\leq\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(-\lambda s)(F(s)-G(s))\,\mathrm{d}P_s\right\|_p.\end{aligned}$$

Thanks to the work of Dirksen [5, Theorem 1.1] we can estimate such a stochastic integral with respect to the Poisson noise by

$$\begin{aligned}&\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(-\lambda s)(F(s)-G(s))\,\mathrm{d}P_s\right\|_p\\ &\leq\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(-\lambda s)(F(s)-G(s))\,\mathrm{d}(P_s-s)\right\|_p\\ &\quad+\mathbb{E}\sup_{0\leq t\leq T}\left\|\int_0^t S(-\lambda s)(F(s)-G(s))\,\mathrm{d}s\right\|_p\\ &\leq C_p\mathbb{E}\sup_{0\leq t\leq T}\int_0^t\|S(-\lambda s)(F(s)-G(s))\|_p\mathrm{d}s\\ &\quad+\mathbb{E}\sup_{0\leq t\leq T}\int_0^t\|S(-\lambda s)(F(s)-G(s))\|_p\mathrm{d}s\\ &\overset{(*)}{\leq}(1+C_p)\mathbb{E}\sup_{0\leq t\leq T}\int_0^t\|F(s)-G(s)\|_p\mathrm{d}s\\ &=(1+C_p)\mathbb{E}\int_0^T\|F(s)-G(s)\|_p\mathrm{d}s\\ &\leq(1+C_p)T\|F-G\|_{p,T},\end{aligned}$$

where $t\mapsto P_t-t$ is called a *compensated Poisson process* which is called so as

$$\mathbb{E}P_t-t=0,\quad t\geq 0,$$

C_p is a constant depending only on p, and $(*)$ follows from the dilated analogue of (13), see [6]. (Integrals with respect to P_t-t

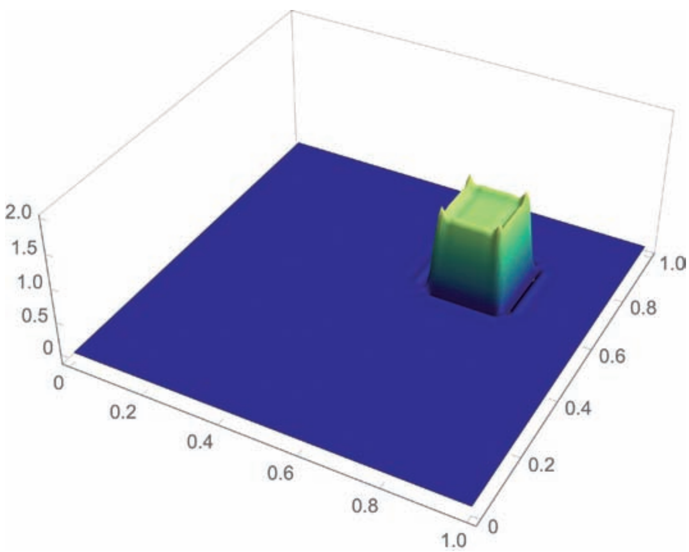

Figure 1 Here and later $O = [0,1]^2$ is periodic, $I(t) = \mathbf{1}_{[0.6,0.8]\times[0.6,0.8]}$ for $t = 0$.

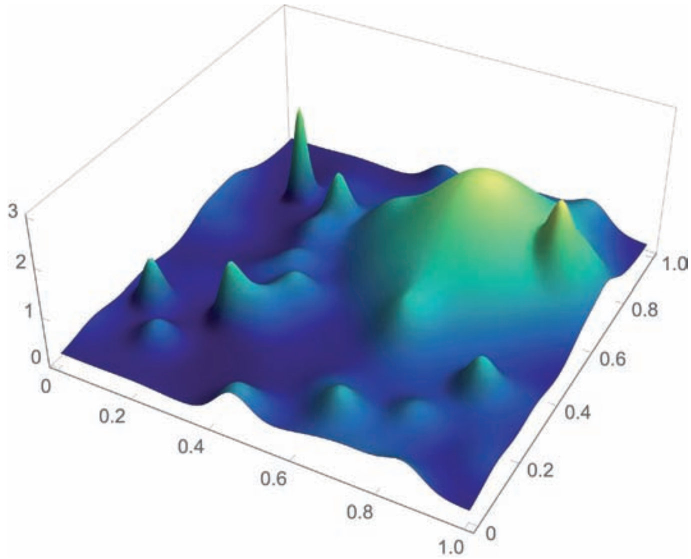

Figure 2 $I(t)$ for $t = 0.6$.

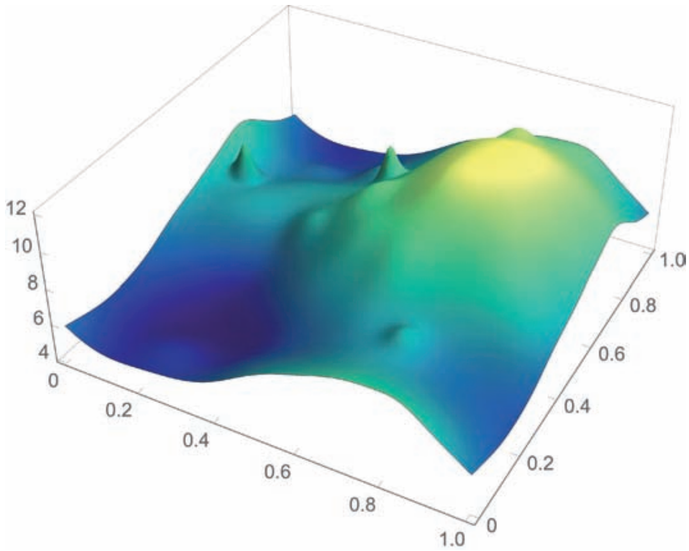

Figure 3 $I(t)$ for $t = 1.2$.

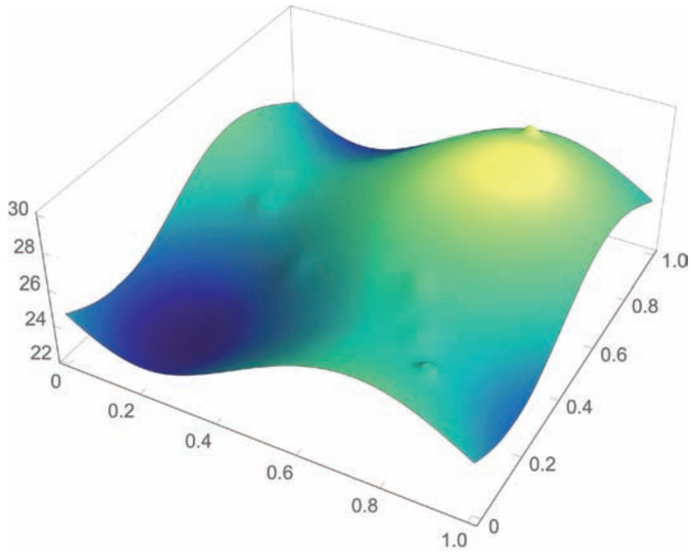

Figure 4 $I(t)$ for $t = 1.8$.

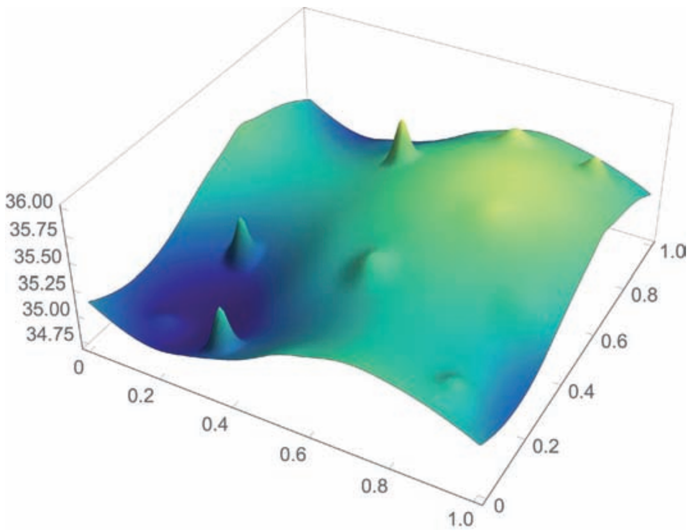

Figure 5 $I(t)$ for $t = 2.4$.

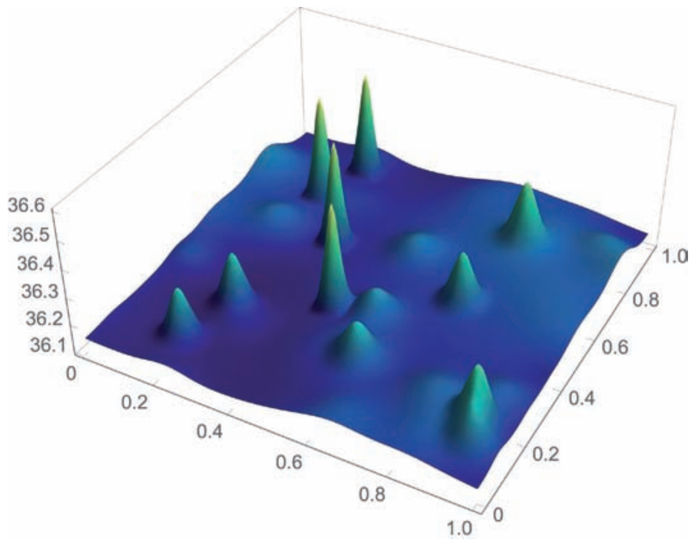

Figure 6 $I(t)$ for $t = 3$.

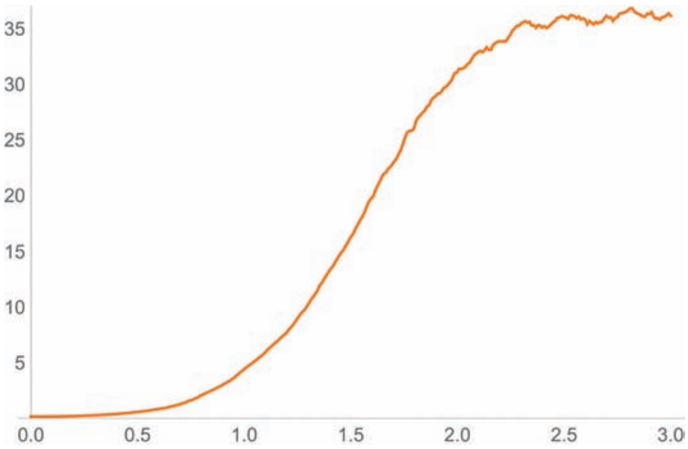

Figure 7 Evolution of $\| I(t) \|_2$, $0 \leq t \leq 3$.

and more general compensated Poisson random measures have been studied by Dirksen in [5].) Similarly, exploiting (6) one can show that

$$\mathbb{E} \sup_{0 \leq t \leq T} \left\| \int_0^t S(\lambda t - \lambda s) \mathbf{1}_{A_k} (\gamma_k(F(s)) - \gamma_k(G(s))) \, \mathrm{d}P_s^k \right\|_p$$
$$\leq (1 + C_p) C_\gamma T \| F - G \|_{p,T}.$$

If we sum up all the estimates, we get

$$\| \phi(I) - \phi(G) \|_{p,T}$$
$$\leq \left(C_\alpha T + |a| C_p T^{\frac{1}{2}} + |b| (1 + C_p) T + (1 + C_p) C_\gamma T \right) \| F - G \|_{p,T},$$

so if we choose T small enough, the conditions of Theorem 1 are satisfied, and thus there is only one I so that (4) holds on $[0,T]$. Iterating the procedure (first constructing the solution on $[0,T]$, then starting in T on the segment $[T,2T]$, et cetera), we obtain a unique solution of (4) on the whole $\mathbb{R}_+$.

Now, when we have that (4) has a unique solution (again, *in some sense*), we are able to ask further questions, such as

- For which t either $\| I(t) \|_p$ or $\mathbb{E} \| I(t) \|_p$ becomes small/big?
- What happens if we change the coefficients of (4)? What additional terms corresponding to the fight against the disease would with a high chance vanish $\| I(t) \|_p$ in time?
- Can we simulate the solution of (4) (see Figures 1–7)? Will this simulation converge to the solution? In which sense?
- How (4) and its solution are related to the real epidemic dynamics?
- What happens if we change (3) and (4) to the full SIR (or any other appropriate) model (likewise in [10])? Can we then obtain the existence and uniqueness of the solution using the tools exploited above?
- Do we have convergence of the disease distribution in some sense (see, e.g., Figures 1–7)?
- et cetera.

These questions are extremely challenging on their own and they require additional rigorous research, but at least we can guarantee that they are well-posed.

Example 3. Figures 1–7 present a simulation of the solution of (3) for $\mathcal{O} = [0,1]^2$ being periodic (in this case the heat kernel has a simple form), $\lambda = 0.015$, $\zeta(x) = \max\{3.7 - 0.1x, \min\{-2, 0.1x - 6.5\}\}$, $a = 50$, $b = 5$, $(A_n)_{n=1}^{2500}$ is a partition of $\mathcal{O}$ into 2500 equal squares with the side length 0.02, $\gamma_k(x) = \min\{x, 4/(x + 0.1)\}$, and intensities of P^k are not Lebesgue measures, but $0.08\lambda_{\mathbb{R}_+}$. The simulation is made on the mesh of 50×50 points with the time differences of 0.01. Figures 1–6 show the graph of the infection density for times $t = 0, 0.6, 1.2, 1.8, 2.4, 3$, and Figure 7 demonstrates the evolution of $| I(t) |_2$ over $t \in [0,3]$.

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