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PROFESSOR WIESŁAW SZLENK (1935–1995)

1. Life and activity. Wiesław Szlenk was born in Warsaw in 1935 and died in Barcelona in 1995. His university education started in 1953 at the Faculty of Mathematics, Physics and Chemistry of Warsaw University. Being a very good student he was offered a position of deputy assistant (second assistant) in 1956. In 1958 he completed his study and started a full time employment at Warsaw University. His first scientific interests were in functional analysis where he worked under the supervision of Stanisław Mazur. Szlenk's mathematical skills revealed in solving several open problems and proving new important theorems. In close cooperation with Aleksander Pełczyński he solved the Mazur problem for numerical series [20] and the problem of Singer [21]. Then he extended the Banach–Saks theorem to L^1-spaces [22]. In [23] Szlenk solved Banach and Mazur's problem 49 from the Scottish Book showing the non-existence of a universal reflexive Banach space. In the course of this work Szlenk prepared his Ph.D. dissertation "On some properties of weakly convergent sequences in Banach spaces". It was completed in 1963 under the supervision of S. Mazur.

In 1964 Mazur arranged for W. Szlenk's (and Karol Krzyżewski's) research stay at Moscow University. They had an opportunity to select from several mathematical seminars (cf. [27]). They chose the one on dynamical systems led by Yasha Sinai. It was a very important moment in Szlenk's scientific career. He decided to turn from functional analysis to dynamical systems where he achieved his most important mathematical results and showed great teaching abilities. The study he started in Moscow was continued in the following years in Warsaw. In 1967 Sinai came to Warsaw University as a visiting professor. During that visit he initiated a seminar on dynamical systems. After his departure the seminar was led by Krzyżewski and Szlenk. The seminar (still existing after more than 30 years) was the starting point of research in dynamical systems in Warsaw. Wiesław Szlenk was a leading

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figure in this activity for several years. He proved a number of important theorems (to mention just a few: on the existence of invariant measures for expanding transformations on manifolds [15]—with K. Krzyżewski, on the characteristics of entropy [17], [18]—with M. Misiurewicz, on the existence of an invariant measure for some rational transformations of the Riemann sphere [14]—with P. Grzegorczyk and F. Przytycki). In 1977 he received habilitation for the thesis "Properties of orbits of continuous functions in topological dynamical systems and their connection with invariant measures and entropy". Wiesław Szlenk was also the author of other important results in pure mathematics (see the review [19] in Polish).

Independently of his research activity Wiesław Szlenk was lecturing a lot. He attracted many gifted young mathematicians to the new scientific field, creating a very active research group. He lectured not only in Warsaw, but also at many universities abroad (in Denmark, Mexico, Spain). The result of his teaching experience was the book [25], the first monograph on dynamical systems in Polish. Its English translation [26] also got a favourable reception. Moreover, he was the author of the first high-school text-book in probability in Polish [24].

In 1979 Wieslaw Szlenk, already an associate professor, moved to the Warsaw University of Agriculture (SGGW), where he worked until 1982. During this period he developed contacts with biologists and was attracted by possible applications of mathematics in biology and medicine. He soon obtained interesting results in modelling adhesion dynamics ([8]–[10], with J. Doroszewski, J. Jakubas and K. Lewandowska), plant growth ([30]–[33], with W. Żelawski), growth of baleen whale population ([5], with F. Bofill), immune systems ([4], [6], [29], with F. Bofill, R. Quentallia, A. Borkowska and C. Vargas), and estimation of rainfalls ([11], with B. Dżura and W. Hyb). The interest in applications of mathematics lasted until his last years. His last article concerned the model of mixing in rumen [28].

In 1982, Szlenk returned to Warsaw University. From the beginning he was deeply engaged (jointly with Andrzej Palczewski) in developing a new curriculum in applied mathematics. This resulted in a substantial increase of the number of students choosing applied mathematics as their field of interest. It also helped create the Institute of Applied Mathematics and Mechanics, which greatly extended the area of research and education in mathematics.

Wiesław Szlenk was a man of action with marvellous organizational skills. This was soon recognized by his colleagues who elected him several times to various academic posts. In the years 1972–78 he was the deputy director of the Institute of Mathematics at Warsaw University. From 1979 to 1982 he was the director of the Institute of Applied Mathematics and Statistics at the Warsaw University of Agriculture. In 1993–95 he was the deputy

director of the Institute of Applied Mathematics and Mechanics at Warsaw University. Since 1974 he was a member, and from 1986 to 1993 the head of the Board of Mathematical Olympics in Poland.

I met Professor Szlenk for the first time attending his lecture on "Mathematical models in biology". The subject and the personality of the lecturer attracted me so much that I chose mathematical biology as theme of my master thesis. I was very happy when he offered me the possibility of writing a Ph.D. thesis under his supervision. During the years when I was preparing the thesis he proved to be very helpful and tutelary, always having time for discussion, encouraging to publish results, providing new scientific contacts.

In personal contact, Wiesław Szlenk was open and straightforward. He used to tell numerous anecdotes about the famous mathematicians he had met. He was also a climber and speleologist. Unfortunately I never was with him in the mountains but I have heard many stories about his mountain adventures.

Professor Szlenk was a real authority for me and for many of his collaborators. And as such he will stay in our memory.

It is to notice that some of Szlenk's collaborators prepared articles dedicated to his memory: Tomasz Nowicki [19] in Poland, Luis Alsedà [1] in Barcelona, and Karol Krzyżewski, Michał Misiurewicz and Feliks Przytycki edited papers [12] in pure mathematics dedicated to the memory of Wiesław Szlenk.

2. Activity in biomathematics. From the seventies till the end of his life, Wiesław Szlenk was attracted by the applications of mathematics to biology and medicine. From the mathematical point of view, most of the models he investigated were classical dynamical systems (both continuous and discrete).

2.1. *Dynamics adhesion and cell flow.* Initially, Szlenk was interested in the study of dynamics adhesion and cell flow on a glass bead column ([8]–[10]). The quantitative analysis of interactions between lymphocytes and substratum is important in view of a number of problems concerning the physiological role and kinetics of these cells *in vivo*, and in connection with the adhesion method for separation of lymphocytes T and B.

The class of models proposed by J. Doroszewski, J. Jakubas and W. Szlenk ([8], [9]) and based on experiments [7] describes the retention of lymphocytes in a glass tube. Lymphocytes of the rat thymus labeled with ^{51}Cr were suspended in a phosphate-buffered solution. The cell suspension passed through a glass column filled partly with soda-glass beads. The diameters of the beads varied from 300 to 400 μm. The concentration of cells flowing into the column was 4×10^6 cells/ml. The volume velocity of the flow was 2.3 ml/min. The temperature inside the column was between

36.8°C and 37.2°C. The pH of the medium used for perfusion was 7.0–7.2. The lengths of the columns were different in different experiments (1.2, 1.6, 2.0, 2.4, 2.8 cm). By measuring ^{51}Cr activity, the concentration of the cells flowing out of the column was determined and the fraction of cells retained on the bead bed was calculated as a function of the perfusion duration.

At the beginning of perfusion, the cell concentration in the suspension flowing out from the bead bed was low. It slowly increased to a value close to the concentration in the inflowing suspension. Therefore, the number of cells retained on the bead bed increased with the perfusion duration. The character of the curves describing the change of concentration indicated that the rate of cell retention on the bead bed decreased in time, i.e. the effectiveness of filtration in the bead layer decreased.

In the class of models of [8], [9], it is assumed that:

- On the surface of the glass, there are a finite number of hypothetical active centers which retain the flowing cells.
- In the process of retaining, one cell occupies one active center.
- The number of cells retained on the bead bed per time unit depends on the concentration of inflowing cells, the number of active centers and the adhesion properties of the cell.
- The maximal number of cells stopped on each cross-section of the column by a plane perpendicular to the axis of the tube is constant (because of the homogeneous packing of the beads).

During the perfusion, the increasing number of cells causes accumulation in the perfused layer. The accumulated cells occupy more and more active centers, and the rate of cell retention decreases, since with continuing perfusion, the number of free active centers is diminished. The amount of retained cells and the retention rate depend on the length of the layer. The concentration of cells is not uniform. It is higher in the upper part (closer to the inlet) than in the lower part.

In the particular layers of the bead bed, the phenomenon occurs similarly to that in the column as a whole. The concentration of cells flowing into the deeper-situated layers varies with time, since it is dependent not only on the flowing cell concentration, but also on the retention rate in the upper layers, i.e. on the number of cells retained there.

In a layer of very small length dx, the change of cell concentration is negligible. The process of cell retention in the bed as a whole is the result of events occurring in every layer.

Let $g = g(x,t)$ denote the suspension concentration in the layer of coordinate x (i.e. x is the distance from the entry of the column) at time t.

Let A_M denote the maximal capacity of the layer (A_M is assumed to be independent of x).

Let $A(x,t)$ denote the density of cells captured up to time t at level x.

The most general description of the above phenomenon is given by the equation

$$\frac{\partial A}{\partial t} = f(g, A_M - A, x, t).$$

Some similarities to physico-chemical aspects of the adhesion process are suggested by the equation ([8], [9])

$$\frac{\partial A}{\partial t} = \alpha n(A_M - A), \quad n = vgS, \tag{1}$$

where α is a constant, v is the mean linear velocity of the flow of cellular suspension into the packing and S is the mean area of the cross-section of the bed. If v is constant and $s(x)$ is the area of the cross-section at level x, then $S = a^{-1}\int_0^a s(x)\,dx$, where a is the length of the column.

The second equation is the transport equation

$$\frac{1}{S}\frac{\partial A}{\partial t} + \frac{\partial g}{\partial t} + v\frac{\partial g}{\partial x} = 0. \tag{2}$$

The domain for (1) and (2) is $\{x \geq 0,\ t \geq x/v\}$ and the boundary conditions are

$$g(0,t) = g_0 = \text{const} \quad \text{and} \quad A(x, x/v) = 0. \tag{3}$$

The solution of (1) and (2) with the boundary conditions (3) is

$$g = g_0\left[1 - \frac{(e^{\alpha A_M x} - 1)e^{\alpha n x/v}e^{-\alpha n t}}{1 + (e^{\alpha A_M x} - 1)e^{\alpha n x/v}e^{-\alpha n t}}\right],$$

where $g_0 = \text{const}$ is the concentration of the incoming suspension.

Let $W(x,t)$ denote the cell retention. For the column of length x the experimental results ([7], [8]) are approximated by the function

$$W(x,t) = 1 - \frac{Svg(x,t)}{Svg_0},$$

and (1) and (2) yield

$$W(x,t) = \frac{(e^{\alpha A_M x} - 1)e^{\alpha n x/v}e^{-\alpha n t}}{1 + (e^{\alpha A_M x} - 1)e^{\alpha n x/v}e^{-\alpha n t}}\ . \tag{4}$$

It occurs that, for constant bed length x, there exist two parameters, α and A_M, such that the theoretical curve fits the experimental one, but universal values of the parameters for all possible x do not exist (for example, for $x = 1.2$ and $x = 2.8$, the maximal error is not less than 0.31).

In [8], [9], the authors investigated a function which fits the experimental data [7]. They used the generalization of (4) in which the coefficient b varies with x. This concept was based on the fact that, for each given x, the

parameters α and A_M could be chosen such that they well approximated the experimental data.

The generalization has the form

$$W(x,T) = \frac{c(x)e^{-b(x)t}}{1+c(x)e^{-b(x)t}}. \tag{5}$$

Putting

$$c(x) = (e^{\alpha A_M x} - 1)e^{\alpha g_0 S x}, \quad b(x) = \alpha g_0 v S$$

in (5) gives (4).

The differential equation of a thin layer, based on (2) and (5), is then

$$\frac{\partial A}{\partial t} = \frac{g}{g_0} b(x)(A_M - A) + gv\frac{b'(x)}{b(x)} \log \frac{g_0}{g} \tag{6}$$

with the same boundary conditions.

Fitting the curve described by (5) to the experimental one yields the functions

$$b(x) = \lambda e^{-\beta x}, \quad c(x) = \alpha(e^x - 1)e^{-\beta x}.$$

The best approximation was obtained for $\lambda = 0.31,\ \beta = 0.28,\ \alpha = 3.1$.

For $b = \text{const}$, (6) takes the form (1). It occurs (see [9]) that the function on the right-hand side of (6) depends explicitly on x, i.e.

THEOREM 1. *There exists no function $H(g, A_M - A)$ of two variables only such that*

$$\frac{\partial A}{\partial t} = H(g, A_M - A).$$

Theorem 1 provides an interesting conclusion concerning the described process. The layers in the column do not act independently, i.e. the adhesion process is not a direct sum of processes in the layers. The action of every layer depends on its place in the column.

The next model proposed by J. Doroszewski, K. Lewandowska and W. Szlenk is based on the two mechanisms involved in cell passage, i.e. labyrinth effect and random delay (see [10]). The following assumptions on the model are made:

- The structure of the labyrinth is homogeneous, i.e. it consists of a large number of similar structural units such that the border regions can be neglected. The shape of the labyrinth is cylindrical.
- There is the most probable direction of the flow in the labyrinth, parallel to the cylinder axis.
- The channel diameter (i.e. the diameter of the family of flow trajectories homotopic inside the free space in the labyrinth) is large enough such that a free flow of particles is possible.

The labyrinth bed is perfused with the fluid flowing with constant velocity. A small amount of cell suspension is injected into the perfusing fluid. The dilution of cells which pass through the labyrinth is estimated by measuring their concentration in the fluid flowing out of the bed. The cells which are retained in the labyrinth bed influence only the normalizing parameter of the dilution curve.

Assume that the bed is cut by the family of horizontal planes with constant distance between them. Let Q_i, $i = 1, \ldots, n$, denote the family of the resulting isometric parts. Let ξ denote the time of passing the column by a particle, and $\xi_i = \xi|_{Q_i}$. The authors assume that the random variables ξ_i are independent and have the same distribution. If n is large enough, then in view of the Central Limit Theorem the density $f_\xi(x)$ of the random variable ξ is close to normal distribution. Let m and σ denote its parameters. This simple mechanism of particle passage through the bed corresponds to a pure "labyrinth effect" in which the dispersion of particles has the Gaussian distribution. The experimental dilution curves [10], however, are not symmetrical, and thus they cannot be Gaussian. Therefore, another phenomenon should be taken into account. The authors of [10] suppose that the overall phenomenon is caused by two components—the labyrinth effect and a second mechanism which results in an additional delay of particles. This delay may be connected with the interactions of particles with themselves and with the labyrinth, and/or with the flow in regions near the wall. The authors assume that the particle delay is a random variable η and it corresponds to a Markov process. Let f_η denote the density of η. The authors assume

$$f_\eta(x) = c \cdot e^{-cx},$$

where c is a parameter.

Now, an interesting random variable is $\zeta = \xi + \eta$ with density $f_\zeta(t)$. It is assumed that ξ and η are independent, therefore f_ζ is the convolution of f_ξ and f_η, i.e.

$$f_\zeta(t) = \int_{-\infty}^{\infty} f_\xi(x) f_\eta(t-x)\, dx.$$

This means that

$$f_\zeta(t) = \frac{c}{\sigma\sqrt{2\pi}} e^{cd - c^2\sigma^2/2} e^{-ct} \int_{-\infty}^{t} e^{-(x-d)^2/(2\sigma^2)}\, dx,$$

where $d = c\sigma^2 + m$.

In the experiment [10], only a fraction of particles are observed flowing out of the labyrinth (because some particles may be arrested in the bed). Therefore, the authors introduce a normalizing parameter and obtain a the-

oretical curve

$$\overline{f}(t) = A \cdot f_\varsigma(t)$$

which they compare with the experimental curves. In all the cases studied (i.e. for lymphocytes, leukemic cells and a dye) the relative error is not greater than 10%, while for the Poisson model (which was used previously) it reached almost 50%.

2.2. *Plant growth.* In the eighties, the main interest of Szlenk was modelling of plant growth ([30]–[33]). In those articles, Szlenk and W. Żelawski investigated the differences between the growth of plants and other populations. The same mathematical approach is usually applied in plant or/and animal growth studies, disregarding the fact that various organisms have different life strategies, and augment their dimensions or quantities in different ways.

Following von Bertalanffy [2], the authors suggested [31] that whereas the growth of many animals is limited to the juvenile stage, the plants continue to grow during their whole life and their different parts grow independently to a large extent. Whereas animals gain their weight through the consumption of food that is taken up in the form of organic matter, the plant body is formed through some processes occurring in photosynthesizing leaves. The growth of animals, being essentially the realisation of a genetically predetermined program, can hardly be modified other than by changing the general growth rate. In plants, the genotype determines the range of phenotypical variation rather than the final size and shape of the body. The growth of a plant is the sum of growths of all its growing parts, but the role played in the total productivity by assimilatory organs themselves is a special one; the partitioning of photosynthetic products into assimilatory and non-assimilatory parts, which varies phenotypically, is of great importance for the growth of the whole plant.

Szlenk and Żelawski assumed that there are two main features dependent on the availability of such extremal factors as water, fertilizers, light, carbon dioxide, etc. These are the efficiency of assimilatory organs, and the partitioning of photosynthetic products between the two compartments, i.e. assimilatory and non-assimilatory organs.

The class of models in [30]–[33] is based on the following assumptions. Let W_n and V_n denote the dry weight of assimilatory and non-asimilatory organs, $M_n = W_n + V_n$. The organic matter, which is photosynthetically produced during the nth time unit (usually one day), is proportional to W_n with the coefficient of proportionality α_n (which represents the efficiency of assimilatory organs, see [30]) or, in the general case ([31]–[33]), $\alpha_n = \alpha(W_n)$,

where α is decreasing, continuous, and such that

$$\alpha(0) = \alpha_0 > 0, \qquad \lim_{W\to\infty} \alpha(W) = 0. \tag{7}$$

The organic matter is distributed between the assimilatory and non-assimilatory organs with certain proportions β and $1-\beta$. The coefficient β depends on the ratio $\lambda_n = W_n/V_n$, $V_n > 0$, and on a parameter $\sigma \in [0,1]$, which represents environmental conditions. In the class of models [30]–[33], $\sigma = \text{const}$, hence $\beta = \beta(\lambda)$.

$\beta \in (0,1)$ is a decreasing function of λ, and

$$\beta(0,\sigma) = 1, \qquad \lim_{\lambda\to\infty} \beta(\lambda,\sigma) = 0. \tag{8}$$

If $V_n \gg W_n$, then β is close to 1, and almost all dry substance $\alpha(W_n)W_n$ produced is used to build the assimilatory part. If $V_n \ll W_n$, then β is close to 0, and almost all material $\alpha(W_n)W_n$ is used for the extension of the non-assimilatory part.

The class of models in [30]–[33] is described by the following system of equations:

$$\begin{aligned} W_{n+1} &= W_n + \beta(\lambda_n)\alpha(W_n)W_n, \\ V_{n+1} &= V_n + [1-\beta(\lambda_n)]\alpha(W_n)W_n. \end{aligned} \tag{9}$$

Let

$$V_n = \frac{1}{1+\lambda_n} M_n, \qquad W_n = \frac{\lambda_n}{1+\lambda_n} M_n.$$

Then equations (9) take the form

$$\begin{aligned} M_{n+1} &= M_n + \alpha(\lambda_n M_n/(1+\lambda_n)) \cdot \lambda_n M_n/(1+\lambda_n) \\ \lambda_{n+1} &= \frac{1+\beta(\lambda_n)\alpha(\lambda_n M_n/(1+\lambda_n))}{1+(1-\beta(\lambda_n))\alpha(\lambda_n M_n/(1+\lambda_n))\lambda_n}\lambda_n. \end{aligned} \tag{10}$$

Let the right-hand sides of (10) be denoted by $F_1(M,\lambda)$, $F_2(M,\lambda)$, respectively. Equations (10) define a mapping F of $D = \{(M,\lambda) : M \geq 0,\ \lambda \geq 0\}$ into itself: for a point $(M,\lambda) \in D$,

$$F(M,\lambda) = (F_1(M,\lambda), F_2(M,\lambda)) \in D. \tag{11}$$

Equations (10) describe the iterations of the map F, i.e.

$$(M_{n+1}, \lambda_{n+1}) = F(M_n, \lambda_n) = F^n(M_0, \lambda_0).$$

Szlenk and Żelawski proved the following.

LEMMA 1. *The mapping F is a map of D into itself, but it is not surjective.*

One can see that there exists a unique $\overline{\lambda} > 0$ such that

$$\overline{\lambda} = \frac{\beta(\overline{\lambda})}{1-\beta(\overline{\lambda})}.$$

From now on, it is assumed that the functions α and β are of class C^1.

LEMMA 2. *There exists a number $\overline{M} > 0$ such that if $M > \overline{M}$ and $\lambda \leq \overline{\lambda}$, then $F_1(M, \lambda) \leq \overline{\lambda}$.*

THEOREM 2. *For any $M_0 > 0,\ \lambda_0 > 0$,*

$$\lim_{n\to+\infty} M_n = +\infty, \qquad \lim_{n\to+\infty} F_1(M_n, \lambda_n) = \overline{\lambda},$$

and $\overline{\lambda}$ is a unique positive fixed point of F_2.

It turns out that if M_0 is large enough, then the sequence (λ_n) is monotone. One can expect that if λ_0 is close to 0 and M_0 is small, then this sequence oscillates arround $\overline{\lambda}$.

The two extremal cases $\lambda = 0$ and $\lambda = +\infty$ are also biologically interpretable (see [33]). The case $\lambda = 0$ may correspond to the situation of a plant devoid of assimilatory organs (e.g. a deciduous tree in spring). The case $\lambda = +\infty$ may be a shoot alone without root (e.g. a leaf or a stem before rooting when vegetatively propagated). For any $M > 0$, the points $\lambda = 0$ and $\lambda = +\infty$ are repulsive, i.e. the plant tends to escape from the state where either assimilatory or non-assimilatory organs are lacking. Regeneration of the missing part is then very vigorous.

The real growth of plants may be interrupted in a catastrophic way (e.g. wind, deterioration of water regime, disease). Under certain conditions, $\alpha(W_n)$ may approach 0 causing a complete cessation of growth. However, even under the natural conditions, it is possible that α reaches 0 quickly due to the sudden deterioration of photosynthetic activity, for instance at the end of the growing season. Hence the plant growth curve is usually of sigmoidal shape.

Assume that $\alpha(W) = \text{const}$ and

$$\beta(\lambda_n) = \frac{\sigma}{\sigma + (1-\sigma)\lambda_n}, \quad 0 < \sigma < 1. \tag{12}$$

Then

$$\overline{\lambda} = \sqrt{\frac{\sigma}{1-\sigma}}.$$

Usually, the assimilation rate α is < 1, and in this case, the following corollary of Theorem 2 holds:

COROLLARY 1. *If $\alpha < 1$, then for every initial value λ_0, $\lim_{n\to\infty} \lambda_n = \overline{\lambda}$.*

The ratio $\overline{\lambda}$ is experimentally measurable. It is the ratio towards which the plant adjusts itself during the process of adaptive growth. The growth under constant conditions for some time leads to the stabilization of the proportion of assimilatory and non-assimilatory organs.

Considering the experimental data [30], it occurs (due to self-shading effects and an increasing ballast of non-photosynthesizing tissue) that the coefficient α decreases in an inverse proportion to the size of assimilatory organs ([31]–[33]):

$$\alpha(W) = \frac{a_0}{W + a}, \quad \alpha_n = \alpha(W_n), \tag{13}$$

where a_0, a are some constants. Due to the daily variation of the environment, the coefficient a, reducing the potential unit leaf rate α, is introduced to the model. Mathematically, α_n is a stochastic process.

In [32] the following is proved.

THEOREM 3. *If α is of the form* (13) *and β is of class C^1 and satisfies conditions* (8), *then there exist constants P, Q, R such that*

$$\lim_{n\to+\infty}(M_n - (Pn + Q\ln n + R)) = 0.$$

This means that for large n, the total mass of the plant increases almost as a linear function $(Pn + R)$. The total error of approximation tends to 0 as n tends to $+\infty$. If one considers the growth curves of wood accumulation in trunks of large trees, one can see that being at the beginning exponential, they become gradually more and more similar to linear ones.

Let $W_{\alpha/2}$ denote the dry weight of assimilatory organs for $\alpha_n = \alpha_0/2$. Then

$$\alpha_n = \frac{\alpha_0 W_{\alpha/2}}{W_{\alpha/2} + W_n},$$

and equations (9), for β defined by (12), take the form

$$\begin{aligned} W_{n+1} &= W_n + \frac{W_{\alpha/2} d\alpha_0 \sigma V_n W_n}{(W_{\alpha/2} + W_n)[\sigma V_n + (1-\sigma)W_n]}, \\ V_{n+1} &= V_n + \frac{W_{\alpha/2} d\alpha_0 (1-\sigma) W_n^2}{(W_{\alpha/2} + W_n)[\sigma V_n + (1-\sigma)W_n]}, \end{aligned} \tag{14}$$

which is the form of the model used in [31] to compare with experiments [30].

The experimental data [30], obtained during the first growing season of Scots pine, were used to test the model. The initial values W_0 and V_0 where taken from experimental data when the cotyledons and first primary needles were already formed. The parameter σ was calculated as $\sigma = \overline{\lambda}^2/(1+\lambda^2)$, where $\overline{\lambda}$ was established at the end of the growing season. The parameter α_0 was first estimated from the linear regression between $1/\alpha$ and W (for all periods when such a relation did hold with correlation coefficient $r > 0.98$) and then fitted more precisely by trial-and-error, simultaneously with $W_{\alpha/2}$. The parameter d was assumed to be either 1 (for laboratory experiments) or $\sin\frac{\pi}{2}[(185-n)/125]$ (for experiments at natural day length and light intensity). The approximation of growth curves to the experimental points,

representing mean values of numerous plants, was fully satisfactory, not only under laboratory conditions but also under uniform weather conditions.

2.3. *Immunology.* One of the latest research fields of Szlenk was modelling in immunology ([4], [6], [29]). He was interested in Marchuk's model, which is a system of four differential equations with a time delay

$$\begin{cases} \dot{V} = (\beta - \gamma F)V, \\ \dot{C} = \alpha\xi(m)V(t-\tau)F(t-\tau) - \mu_c(C - C^*), \\ \dot{F} = \varrho C - (\mu_f + \eta\gamma V)F, \\ \dot{m} = \sigma V - \mu_m m \quad \text{for } m \le 1, \end{cases} \tag{15}$$

where $V(t)$, $C(t)$, $F(t)$ are the antigen, plasma cell and antibody concentrations at time t; $m(t)$ is a characteristic of the damage of the organ-target; the function ξ has the following properties: ξ is 1 for $m < m^*$ (where m^* is some level of the damage), and is linearly decreasing to 0 for $m \in (m^*, 1]$; the parameter $\tau = \text{const}$ denotes the delay of immune reactions.

Szlenk and C. Vargas (see [29]) analysed a simple form of Marchuk's model. They assumed that the damage of the organ-target is small, $m < m^*$. In this case the model reduces to three equations in V, C, F. They also assumed that the delay is negligible. These assumptions lead to the following model:

$$\begin{cases} \dot{V} = (\beta - \gamma F)V, \\ \dot{C} = \alpha V F - \mu_c(C - C^*), \\ \dot{F} = \varrho C - (\mu_f + \eta\gamma V)F, \end{cases} \tag{16}$$

with all coefficients positive.

Following Marchuk (see [16]), they discussed the model with initial condition $X_0 = (V_0, C^*, F^*)$, where C^* is the physiological level of plasma cells and $F^* = \varrho C^*/\mu_f$ is the physiological level of antibodies. This type of initial conditions may describe the situation of a healthy organism (which is expressed by the physiological level of plasma cells and antibodies) infected at time 0 by a small dose of the antigen ($V_0 > 0$).

Equations (16) have one or two stationary states. The first one exists for any positive values of the parameters, and is equal to $(0, C^*, F^*)$ (i.e. it describes the healthy state). In the case of small physiological level of antibodies and large stimulation coefficient α or large physiological level of antibodies and small coefficient α, there exists a second stationary state

$$\overline{X} = \left(\frac{\mu_c\mu_f(\beta - \gamma F^*)}{\beta(\alpha\varrho - \mu_c\eta\gamma)}, \frac{\alpha\beta\mu_f - \eta\gamma^2\mu_c C^*}{\gamma(\alpha\varrho - \eta\gamma\mu_c)}, \frac{\beta}{\gamma} \right),$$

which describes the chronic form of the disease.

In [29], two important theorems are proved, concerning the cases of large and small physiological level of antibodies.

THEOREM 4. *Let* $\alpha\varrho > (\mu_c+\beta)\eta\gamma$ *and* $\gamma F^* > \beta$. *Then for every* $V_0 \geq 0$, *the solution* $X(t)$ *of* (16) *has a limit and*

$$\lim_{t\to+\infty} X(t) = (0, C^*, F^*).$$

Theorem 4 means that every solution of (16) tends to the unique (in this case) stationary state of the system.

In the case described in Theorem 4, if there exists a point t_0 such that $F(t_0) > F^*$, then there exists $\widehat{t} > t_0$ such that F has a local maximum at $\widehat{t}$. One can show that

$$V(\widehat{t}) < \frac{\mu_c\mu_f}{\kappa - \eta\beta\gamma + \eta\gamma^2 F(\widehat{t})},$$

where

$$\kappa = \alpha\varrho - \eta\gamma\mu_c.$$

It also turns out that the function $C(t)$ decays to C^* faster than the exponential function, and the function $F(t)$ converges to F^* exponentially, i.e. there exist some constants $K_1,\ K_2,\ K_3$ such that

$$C(t) - C^* \leq K_1 e^{-\mu_c t}$$

and

$$K_2 e^{-\mu_c t} \leq |F(t) - F^*| \leq K_3 e^{-\mu_c t},$$

for sufficiently large t.

The characteristic equation, for the point $\overline{X}$, is

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0,$$

where

$$\begin{aligned} a_1 &= \mu_c + \mu_f + \eta\gamma\overline{V}, \\ a_2 &= \mu_c\mu_f(1 - d/\overline{F}) - \eta\gamma^2\overline{V}\,\overline{F}, \\ a_3 &= \gamma d\mu_c\mu_f, \end{aligned} \tag{17}$$

and $\overline{X} = (\overline{V}, \overline{C}, \overline{F}),\ d = \overline{F} - F^*$.

It is easy to see that one characteristic value is real and negative, two other may be real or complex.

THEOREM 5. *Let* $\alpha\varrho > (\mu_c + \beta)\eta\gamma$ *and* $\gamma F^* < \beta$. *If the point* $\overline{X}$ *is either asymptotically stable, or there are two complex characteristic values with positive real parts, then each solution* $X(t)$ *of* (16) *is bounded and has time-average value* $\overline{X}$, *i.e.*

$$\lim_{t\to+\infty} \frac{1}{t}\int_0^t X(s)\,ds = \overline{X}.$$

Both Theorems 4 and 5 describe the case of an efficient immune system ($\alpha\varrho > \eta\gamma\mu_c$), which reacts immediately ($\tau = 0$). In Theorem 4, the

physiological level of antibodies is high ($F^* > \beta/\gamma$), and then the organism recovers even if the initial level of antigens is high. In Theorem 5, the physiological level of antibodies is low ($F^* < \beta/\gamma$), and then the organism cannot recover, even if the initial level of antigens is very low.

Next, Szlenk in cooperation with A. Borkowska analysed the special case of immune reactions, i.e. reactions after a series of vaccinations (against hepatitis B). Basing on the same ideas as in Marchuk's model, and taking into account some effects connected with the high density of antibodies in such a situation, they proposed [6] a simple model of the antibody ($F(t)$) decline after vaccination:

$$\dot{F}(t) = -A(F(t) - D) - B(F(t) - D)^2, \quad B \ll A. \tag{18}$$

The first term on the right-hand side of (18) corresponds to the mortality of antibodies, the second one is caused by mutual collisions of antibodies, due to their high concentration after a series of vaccinations.

Equation (18) has the solution

$$F(t) = \frac{D + L(A - BD)e^{-At}}{1 - BLe^{-At}}, \quad L = \frac{F_0 - D}{A + B(F_0 - D)},$$

where F_0 is the initial level of antibodies. This solution was used to predict the rate of decrease of antibody concentration in the organism after a series of vaccinations, for experimental data published by Gesemann and Scheiermann [13].

Also Marchuk's model in the case of vaccination, i.e. for $\beta = 0$, was investigated by Szlenk with coworkers—F. Bofill and R. Quentallia ([4]). In [4], it is proved that for an efficient immune system ($\alpha\varrho > \eta\gamma\mu_c$), there are only the following types of the solution behavior:

- F has two extremal values, C has one maximum,
- C has two extremal values, F has one maximum,
- F has one minimum, C has one maximum,
- F is decreasing, C has one maximum,
- F is increasing, C has two extremal values.

It is obvious that $\lim_{t\to+\infty} F(t) = F^*$ and $\lim_{t\to+\infty} C(t) = C^*$. Such types of behavior of the concentration $F(t)$ are observed after a series of vaccinations. The model explains the two different types of behavior observed in experiments (e.g. [3]): the first when the concentration constantly decreases, and the second when $F(t)$ has extremal values.

2.4. *Baleen whales.* Szlenk also studied other biological processes in single articles. In [5] Szlenk and F. Bofill proposed a simplification of the evolution model of the baleen whale population. The International Whaling

Commission used the following model:

$$x_{n+1} = (1-\mu)x_n + R(x_{n-\tau}), \quad n = 0, 1, 2, \ldots,$$

where x_n is the population of whales at time n, μ is the probability that an individual does not survive to the next time moment, τ is the time interval in which a newly born individual becomes sexually mature ($\tau = 7$ years), and $R(x) = (1-\mu)^\tau x[p + a[1 - (x/K)^r]]$, where p, a, r, K are some constants.

Szlenk and Bofill assumed that $\tau = 1$ and replaced $R(x)$ by the function

$$\phi_\lambda(x) = \begin{cases} \lambda x & \text{for } x \in [0, 1/2], \\ \lambda(1-x) & \text{for } x \in (1/2, 1], \end{cases}$$

with parameters $\lambda \in (0, 2]$, $\mu = \lambda/2$.

Then the corresponding process is expressed by the dynamical system $T_\lambda : Q \to Q$, where $Q = [0, 1]^2$ is the phase space and

$$T_\lambda(x, y) = (y, \phi_\lambda(x) + (1 - \lambda/2)y).$$

The dynamics of the system (Q, T) depends on λ, and may be regular or random. The system has two fixed points: $X_1 = (0, 0)$ and $X_2 = (2/3, 2/3)$. It is easy to see that X_1 is a saddle point, and X_2 is a focus, stable for $\lambda < 1$ and unstable for $\lambda > 1$. The following theorems about the regularity of the dynamical system were presented in [5]:

THEOREM 6. *If $0 < \lambda < 1$, then for every point $Y \neq (0, 0)$, the limit $\lim_{n\to\infty} T^n Y$ exists, and*

$$\lim_{n\to\infty} T^n Y = X_2.$$

THEOREM 7. *If $\lambda \in (4^{1/3}, 2)$, then the corresponding dynamical system with its absolutely continuous invariant measure μ_λ is exact, which corresponds to random behavior.*

2.5. *Rainfall estimation.* Some methods of rainfall estimation were proposed by W. Szlenk, B. Dżura and W. Hyb in [11]. The first one is referred to as the PBAM (piecewise biharmonic approximation method), where the area considered is divided into rectangles, and the approximation function is a polynomial of the third degree. Another one is referred to as the PLAM (piecewise linear approximation method), where the area is divided into triangles, and the approximation function is linear.

The rainfall function is the map

$$\text{(point of the area, time period)} \mapsto \text{value of rainfall.}$$

Such a function was found in [11], as a function minimizing some functional. More precisely, let D denote a nonempty, open, convex, bounded subset of $\mathbb{R}^k$ ($k = 1, 2, 3$) with piecewise continuous boundary, and $\overline{D}$ be

the closure of D. Let $W^{2,2}(D)$ denote the Sobolev space of all real functions which are square integrable in D, and which have square integrable generalized derivatives of the first and second order in D.

Let $z_i \in D$ and $f_i \geq 0,\ i = 1, \ldots, m$,

$$A = \{f \in W^{2,2}(D) : f(z_i) = f_i,\ i = 1, \ldots, m\},$$

$$B = \{f \in A : f(r) \geq 0 \text{ for } r \in D\},$$

$$|\text{grad } f|^2 = \sum_{j=1}^{k} \left(\frac{\partial f}{\partial x_j}\right)^2, \quad |d^2 f|^2 = \sum_{i,j=1}^{k} \left(\frac{\partial^2 f}{\partial x_i \partial x_j}\right)^2.$$

Let $P_a : W^{2,2}(D) \to \mathbb{R},\ a \in [0,1]$, denote the class of functionals such that

$$P_a(f) = a \int\limits_D |\text{grad } f(x)|^2\, dx + (1-a) \int\limits_d |d^2 f(x)|^2\, dx \quad \text{for } f \in W^{2,2}(D).$$

Definition 1. A surface $\{f(r) : r \in D\}$ is called *minimally folded* if the following conditions hold:

- $f \in W^{2,2}(D)$,
- $f(z_i) = f_i,\ i = 1, \ldots, k$,
- there exists some $a \in [0,1]$ such that f minimizes the functional P_a.

Such an f is called a *minimizing function*. It is known that for $k = 2$ and $a = 0$ such a function is biharmonic, i.e. $\sum_{i,j=1}^{2} \partial^4 f / \partial x_i^2 \partial x_j^2 = 0$. For example, it can be a polynomial of degree at most three.

Now, let D represent a part of the river basin, $z_i \in D$ represent the points of a rainfall station network in D and f_i be the value of rainfall at z_i, $i = 1, \ldots, m$. Assume that $D \subset \mathbb{R}^2$ and $z_i = (x_i, y_i)$.

In the case of the PBAM, D is divided into rectangles in the following way:

- the edges are parallel to the axes,
- any rectangle contains from 3 to 9 of the points z_i.

We look for an estimator of the form

$$f(x,y) = a_0 x^3 + a_1 x^2 y + a_2 x y^2 + a_3 y^3 + b_0 x^2 + b_1 xy + b_2 y^2 + c_1 x + c_2 y + d.$$

Let $(x_0, y_0) \in D$. One wants to calculate $f(x_0, y_0)$. Let $z_i = (x_i, y_i),\ i = 1, \ldots, k$, belong to the rectangle containing (x_0, y_0). To find the polynomial f such that $f(z_i) = f_i,\ i = 1, \ldots, k$, and f minimizes P_a in the class of polynomials of degree at most three, for fixed $a \in [0,1]$, one applies the method of Lagrange multipliers and one obtains a very complicated system of linear equations.

It is possible to obtain a negative value of f; then the PBAM is modified so that

$$f_0 = \max\{f(x_0, y_0), 0\}.$$

In the case of the PLAM, D is represented by the triangulation with z_i as vertices, and the estimator has the form

$$f(x, y) = ax + by + c.$$

Let $z_0 = (x_0, y_0) \in D$, and z_1, z_2, z_3 be the vertices of the triangle which contains z_0. One finds f such that $f(z_i) = f_i$, $i = 1, 2, 3$. It is easy to see that

$$f_0 = w_1 f_1 + w_2 f_2 + w_3 f_3,$$

where w_j, $j = 1, 2, 3$, are the barycentric coordinates of z_0 in the triangle $z_1 z_2 z_3$. Note that

$$w_1 = \frac{P_1}{P}, \qquad w_2 = \frac{P_2}{P}, \qquad w_3 = 1 - (w_1 + w_2),$$

where P_1, P_2, P are the areas of the triangles $z_2 z_3 z_0$, $z_1 z_3 z_0$, $z_1 z_2 z_3$.

The PBAM and PLAM have been verified empirically, using the rainfall data from the Noteć river basin [11], and the autoverification method, which consists in isolating one by one the stations from the network, approximating the rainfall at a particular station by the given method, and comparing the result with the actual data. It turned out that the linear method is better in general, but in the case of continuous and abundant rainfall, the biharmonic method is more accurate.

2.6. *Mixing in rumen.* The last work of Szlenk was modelling the mixing mechanism in the rumen ([28]). In the Department of Animal Physiology of the Warsaw University of Agriculture, the following phenomenon was observed in the digestive process of sheep. Each sheep ate two types of food (referred to as A and B). Every five minutes a sample of food was taken from a fixed location in the rumen. After 1 hour the samples were found to be composed almost entirely of either A or B. This means that the two substances practically did not mix. In [28], Szlenk tried to answer the question: what was the mixing mechanism in the rumen?

As the model of mixing, Szlenk proposed the following dynamical system. Let $Q = [0, 1] \times [0, 1]$ be the phase space (corresponding to the rumen), $\lambda \in (0, 2]$ is a given number, $Q_0' = [0, \lambda^{-1}] \times [0, 1]$, $Q_0'' = (\lambda^{-1}, 1) \times [0, 1]$. A map T_0 was defined as follows:

$$T_0(p) = \begin{pmatrix} \lambda & 0 \\ 0 & \lambda^{-1} \end{pmatrix} \begin{pmatrix} x \\ y \end{pmatrix} = \begin{pmatrix} \lambda x \\ \lambda^{-1} y \end{pmatrix} \in Q \quad \text{for } p = (x, y) \in Q_0', \tag{19}$$

and

$$T_0(p) = \begin{pmatrix} 1-y \\ x \end{pmatrix} \quad \text{for } p \in Q_0'' \text{ (i.e. } x > \lambda^{-1}). \tag{20}$$

T_0 on Q_0'' was constructed geometrically.

Using the dynamical system (Q, T_0), Szlenk defined another dynamical system. Let $R = [0,1] \times [0, \lambda^{-1}]$. For each $p \in Q$, one of the points

$$T_0(p),\ T_0^2(p),\ T_0^3(p)$$

belongs to R. Let $i = i(p)$ denote the smallest integer such that $T^i(p) \in R$, $i = 1, 2, 3$. A new map $T : R \to R$ is defined as $T(p) = T^i(p)$ for $i = i(p),\ p \in R$. Explicitly,

$$T(x,y) = \begin{cases} (\lambda x, \lambda^{-1} y) & \text{if } x \le \lambda^{-1} \ (i(p) = 1), \\ (\lambda(1-y), \lambda^{-1} x) & \text{if } x > \lambda^{-1} \text{ and } y > 1 - \lambda^{-1} \ (i(p) = 2), \\ (\lambda(1-x), \lambda^{-1}(1-y)) & \text{if } x > \lambda^{-1} \text{ and } y \le 1 - \lambda^{-1} \ (i(p) = 3). \end{cases}$$

THEOREM 8. *Let $\lambda \in (2^{1/(k+2)}, 2^{1/(k+1)})$ for some integer $k \ge 1$. Then there exists a set of rectangles Q_i, $i = 1, \dots, 4k$, such that $T^{4k}|_{Q_i} = \mathrm{id}$. Therefore, the map T does not mix points in*

$$U = \bigcup_{i=1}^{4k} Q_i.$$

The general description of the dynamics of (R, T) is the following: for a given n one can split the space R into a finite number of rectangles P_j such that $T^n|_{P_j}$ has the form

$$T^n(p) = (A_j^n - \lambda^{-k_n} x, B_j^n - \lambda^{k_n} y)$$

or

$$T^n(p) = (A_j^n - \lambda^{-k_n} y, B_j^n - \lambda^{k_n} x),$$

where A_j^n, B_j^n are some numbers associated with the rectangle P_j, and k_n is an integer depending on n, which can oscillate between $-\infty$ and $+\infty$.

Let λ be the solution of the equation

$$\lambda^{r+2} - \lambda^{r+1} - 1 = 0, \tag{21}$$

for some natural r. Equation (21) has exactly one root in $(1, 2)$, and implies that

$$\lambda - 1 = \frac{1}{\lambda^{r+1}}, \qquad 1 - \frac{1}{\lambda} = \frac{1}{\lambda^{r+2}}.$$

Let

$$E = \left[\left(0, \frac{1}{\lambda^{r+1}}\right) \times \left(0, \frac{1}{\lambda}\right) \right] \cup \bigcup_{i=1}^{r+1} \left[\left(\frac{1}{\lambda^{r-i}}, \frac{1}{\lambda^{r-i+1}} \right) \times \left(0, \frac{1}{\lambda^{i+1}}\right) \right].$$

THEOREM 9. *There are at least two invariant sets, U and E, for the dynamical system (R,T). The dynamical system (U,T) is periodic, i.e. there exists m such that $T^m = \mathrm{id}$, and the system (E,T) is random, i.e. its trajectories behave as realizations of a stochastic process.*

The mixing process described by the model is different in different parts of the rumen. Coexistence of a periodic process and a random one seems to be strange, but it occurs to be the reality of some physiological processes.

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