MATHEMATICA APPLICANDA
Vol. 40(1) 2012, p. 15–25

Jacek Miękisz (Warsaw)
Paulina Szymańska (Warsaw)

# On Spins and Genes

**Abstract** Many processes in natural and social sciences can be modeled by systems of interacting objects. It is usually very difficult to obtain analytic expressions describing time evolution and equilibrium behavior of such systems. Very often we rely only on computer simulations. Fortunately, in many cases one can construct useful approximation schemes and derive exact results which capture some specific features of a given process. A frequent approach is to replace interactions between objects by a mean interaction. Here we illustrate a self-consistent mean-field approximation in two examples: the Ising model of interacting spins and a simple model of a self-regulating gene.

*2010 Mathematics Subject Classification:* 00A71; 65D17; 68U10.

*Key words and phrases:* Ising model, self-regulating gene, mean-field approximation.

**1. Introduction.** Many socio-economic and biological processes can be modeled by systems of interacting objects; see for example statistical mechanics and quantitative biology archives [1]. One may then try to derive their global behavior from individual interactions between their basic entities such as protein molecules in gene regulation and signaling pathway networks, animals in ecological and evolutionary models, and people in social processes. Such an approach is fundamental in statistical physics which deals with systems of interacting particles. One can therefore try to apply methods of statistical physics to investigate population dynamics and equilibrium properties of systems of interacting proteins or people.

Although interactions in such models are usually local, they propagate in space and as a result even faraway objects are correlated. This makes the rigorous mathematical analysis of such systems very difficult if not impossible. Therefore, most of such models are investigated only by means of computer simulations. But do we really have to make such a dramatic decision either to try to prove theorems relevant for our models of real phenomena or just to simulate behavior of these models on computers? Fortunately, there is a middle ground of approximations. Approximations can be seen as abstractions. Following our heuristic understanding of the nature of a physical or biological process, we make an ansatz (a new model of reality)

and then we try to derive in a rigorous way properties of this new model and in this way increase our understanding of the process. Of course our new model is less fundamental than the original one and usually we cannot estimate an error introduced by our approximation. We may lose some important features hidden in correlations not taken into account in our approximation. Nevertheless, in many cases we may solve our new model analytically and derive valuable formulas.

We would like to present here a method of self-consistent mean-field approximation. The core idea is as follows. The force exerted on a given object, coming from its neighbors, is replaced by an unknown mean force - a mean field. Given the mean field, we can easily calculate the expected value of the state of the object in the equilibrium (a stationary state of an appropriate dynamics). Now one can compute the value of the mean field and it should be consistent with the unknown value introduced in the beginning. One may expect (on the basis of an appropriate law of large numbers) that the mean-field approximation becomes exact when the number of neighbors tends to infinity.

We illustrate the above approach in two examples: the ferromagnetic Ising model of interacting spins located on a regular lattice and a simple model of a self-repressing gene.

**2. Phase transitions in the Ising model of interacting spins.** It is well known that when one heats a magnet it loses its magnetic properties. A typical diagram of the magnetization as a function of the temperature is given in Fig. 1. We would like to derive such a behavior in some simple model. We assume that magnetization is a vector sum of small magnets associated with atoms, microscopic elements of a macroscopic magnet. On the one hand, interactions between microscopic magnets force them to be aligned so that the resulting magnetization is not zero. On the other hand, thermal motions of atoms disrupt that order. Macroscopic properties of matter are results of such a competition between energetic factors favoring an order and disruptive thermal fluctuations. In particular, it follows that magnetization is a decreasing function of the temperature which quantifies the strength of thermal fluctuations. But then one might naively expect that the magnetization should tend to zero as the temperature approaches infinity. However, we see in Fig. 1 that there is a critical temperature, called the Curie temperature, at which the magnetization vanishes. This is an important example of a phase transition. Our first exercise in a mean-field approximation will provide an explanation of such a phenomenon.

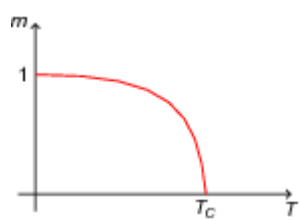


Figure 1: Magnetization per lattice site as a function of the temperature.

**2.1. Mathematical magnet - the Ising model.** In the Ising model [2–4], microscopic magnets are placed at vertices of a regular lattice $\mathbf{Z}^d$ , $d \geqslant 1$, where $\mathbf{Z}$ is the set of integers. Formally, at every vertex (a lattice site) $i \in \mathbf{Z}^d$, there exists a mathematical representation of a microscopic magnet, $\sigma_i$, a variable which can

attain one of two values: +1 (a magnet directed up) and −1 (a magnet directed down). Variables $\sigma_i$ are called spins. The set of infinite configurations of our system is given by $\Omega = \{+1, -1\}^{Z^d}$, that is by the set of all functions assigning +1 or −1 to every lattice site. For a given configuration $X \in \Omega$, $X_i = \sigma_i(X)$ is called a configuration at the site $i \in \mathbf{Z}^d$. Let $\Lambda \subset \mathbf{Z}^d$ be a finite subset of sites of an infinite lattice. $\Omega_\Lambda = \{+1, -1\}^\Lambda$ is the set of configurations on $\Lambda$. Hamiltonian (an energy functional) describes the energy of configurations on $\Lambda$.

$$H_\Lambda : \Omega_\Lambda \to R \tag{1}$$

We assume that spins interact only with their nearest neighbors,

$$H_\Lambda = -\sum_{i,j \in \Lambda} \sigma_i \sigma_j - h \sum_{i \in \Lambda} \sigma_i, \tag{2}$$

where in the first sum $i$ and $j$ are nearest neighbors (for example $j$ is the up, down, left, or right neighbor of $i$ on the two-dimensional square lattice) and $h$ is an exterior magnetic field.

Hamiltonian in classical mechanics of interacting particles is a sum of the kinetic energy of particles and the potential energy of interactions between them. In the above expression we do not consider the potential energy.

Our spin system is subject to thermal fluctuations and so it is a stochastic system whose time evolution can be described by an appropriate Markov chain. The stationary state of such a Markov chain, that is a probability measure on $\Omega_\Lambda$ is interpreted as an equilibrium state of a physical system of interacting spins. All macroscopic properties, such as the energy and the magnetization of the system, are random variables defined on $\Omega_\Lambda$. We will be interested in expected values of such random variables.

We introduce the following probability mass function,

$$\rho_\Lambda^{T,h}(X) = \frac{e^{-\frac{1}{T} H_\Lambda(X)}}{Z(T, h, \Lambda)}, \tag{3}$$

where $T$ is the temperature of the system and

$$Z(T, h, \Lambda) = \sum_{X \in \Omega_\Lambda} e^{-\frac{1}{T} H_\Lambda(X)} \tag{4}$$

is a normalizing factor. $Z$ is the so-called statistical sum and $\rho_\Lambda^{T,h}$ the grand-canonical ensemble. These are basic objects of statistical physics. There are many fundamental explanations why the above probability mass function gives us stationary probabilities of our physical system. Here we will simply assume this and then draw conclusions.

Another important quantity in statistical physics is the free energy also called the thermodynamic potential,

$$F(T, h, \Lambda) = -T \ln Z(T, h, \Lambda). \tag{5}$$

It is worth mentioning here that one of the fundamental and still unsolved problems in statistical physics is to derive an analytic expression for the free energy in the three-dimensional Ising model ($d = 3$) in the thermodynamic limit, that is in the limit of the infinite $\Lambda$,

$$f(T,h) = \lim_{\Lambda \to \mathbf{Z}^3} \frac{F(T,h,\Lambda)}{|\Lambda|}. \tag{6}$$

It is a classical theorem that such a limit exists if $\Lambda$ approaches $\mathbf{Z}^d$ in some nice way. Calculating $f(T,h)$ in the one-dimensional Ising model is an easy exercise, the two-dimensional case for $h = 0$ was solved by a Nobel laureate Lars Onsager [5].

Now we define the macroscopic magnetization per lattice site,

$$M_\Lambda = \frac{1}{|\Lambda|} \sum_{i \in \Lambda} \sigma_i \tag{7}$$

To avoid unnecessary technicalities, we introduce now periodic boundary conditions, that is we turn $\Lambda$ into a d-dimensional torus. Let $m$ be the expected value of the random variable $M$ with respect to the probability mass function $\rho_\Lambda^{T,h}$, that is

$$m = \frac{\sum_{X \in \Omega_\Lambda} M_\Lambda(X) e^{-\frac{1}{T} H_\Lambda(X)}}{Z(T,h,\Lambda)}. \tag{8}$$

We take into account periodic boundary conditions and write the above expression as

$$m = \frac{\sum_{X \in \Omega_\Lambda} \sigma_o e^{\frac{1}{T}(\sum_{i,j \in \Lambda} \sigma_i \sigma_j + h \sum_{i \in \Lambda} \sigma_i)}}{Z(T,h,\Lambda)}, \tag{9}$$

where $o$ is any fixed site, $o \in \Lambda$.

The presence of the sum $\sum_{i,j} \sigma_i \sigma_j$ in the above formula prevents us from expressing $m$ in terms of elementary functions. Now comes the most important step in our calculations. We introduce a mean-field ansatz and replace random variables $\sigma_j$ by their unknown expected value $m$. Every spin interacts with his $2d$ nearest neighbors and hence we replace $\sum_{i,j} \sigma_i \sigma_j$ by $\sum_{i \in \Lambda} 2dm\sigma_i$ [2, 4]. Now the exponent both in the numerator and in the denominator factorizes and after simple algebraic manipulations we get

$$m = \tanh \frac{2dm + h}{T}. \tag{10}$$

Observe that the external magnetic field $h$ is modified by the mean field $2dm$ which can be interpreted as an effective field coming from averaged interactions felt by a given spin. We are especially interested in the case of the zero external field, $h = 0$, that is in the case of a spontaneous magnetization. In such a case, to get a self-consistent $m$, we have to solve the following equation,

$$m = \tanh \frac{2dm}{T}. \tag{11}$$

It can be solved graphically. We look for intersections of the graph of the function $f(m) = \tanh \frac{2dm}{T}$ and the diagonal $y = m$, see Fig. 2. It is easy to see that for big temperatures, that is for $T > 2d$, there exists the unique solution $m = 0$ of (11). However, if $T < T_C = 2d$, then there are three solutions: $m = 0$, and $m = \pm m_0(T)$ for some positive $m_0(T)$. $T_C$ is the critical Curie temperature at which a phase transition takes place. Fig. 1 follows directly from Fig. 2 - we have achieved our goal.

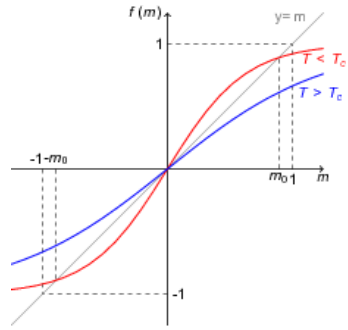


Figure 2: Graphical solution of the mean-field equation (11).

One can prove that for $T < T_C$ the solution $m = 0$ is thermodynamically unstable. The presence of two solutions $m = \pm m_0(T)$ is an effect of the symmetry of the Hamiltonian (in the case of the zero external field, $h = 0$) with respect to the spin flip $\sigma_i \to -\sigma_i$. It means that at any temperature below the critical one, there coexist two equilibrium macroscopic states. The situation is similar to the coexistence of ice and water at the zero Celsius temperature. However, such a mixed state is highly unstable and we usually end up in one of the two so-called pure states.

**3. A simple stochastic model of gene regulation.** One of the fundamental processes taking part in living cells is regulation of the gene expression. It enables cells to differentiate and adapt to a changing environment. Gene expression is a complex process involving many biochemical reactions with proteins being final products. Produced proteins may in turn enhance or repress the expression of other proteins. They may also regulate their own expression.

Here we will show how the mean-field approximation, described in the previous section, might be used for analyzing a simple model of a self-repressing gene.

**3.1. Birth and death process - the simplest model of gene expression.** Birth and death processes describe the stochastic evolution of the number of certain living objects, particles or molecules taking part in chemical reactions. It is a standard assumption in such processes that the probability of any reaction to take place in a sufficiently small time interval $\Delta t$ is proportional to its length, that is it has the form $r\Delta t + o(\Delta t)$, where $r$ is the intensity or the rate of the reaction and $o(\Delta t)$ is a quantity of a lower order than $\Delta t$. We also assume that the probability does not explicitly depend on time and that events in non-overlapping time intervals are independent. It is a standard theorem that there exists a stochastic process satisfying such conditions.

Here we are interested in proteins. In our case there are two reactions: production of a protein molecule directly from the DNA (two fundamental biochemical processes, transcription and translation, are lumped together into one process) and its degradation [6], see Fig. 3. Let $n$ be a number of protein molecules in the cell. We assume that probabilities of reactions in a small time interval $\Delta t$ are:

- production, $n \to n + 1$, : $k_0\Delta t + o(\Delta t)$
- degradation, $n \to n - 1$ : $\gamma n\Delta t + o(\Delta t)$

- more than one reaction, $o(\Delta t)$

$$\mathrm{DNA} \xrightarrow{k_0} P \xrightarrow{\gamma} \emptyset$$

Figure 3: The simplest model of gene expression, $k_0$ is the production rate and $\gamma$ is the degradation rate.

Let $f(n,t)$ be the probability that there are $n$ molecules in the cell at time $t$. Now we can write the following standard system of differential equations, the so-called Master equation [7]:

$$\frac{df(n,t)}{dt} = k_0[f(n-1,t) - f(n,t)] + \gamma[(n+1)f(n+1,t) - nf(n,t)], \tag{12}$$

where we assume that $f(n,t) = 0$ for $n < 0$.

The Master equation describes the conservation of the probability (in the discrete time set-up it is an example of the high-school total probability formula). We are especially interested in the stationary point of (12), the so-called stationary state $f(n)$, that is we set $\frac{df(n,t)}{dt} = 0$. The resulting system of recurrence equations can be easily solved. It is well known that the stationary probability distribution is Poissonian, that is

$$f(n) = e^{-\frac{k_0}{\gamma}} \frac{(\frac{k_0}{\gamma})^n}{n!}. \tag{13}$$

Other solvable models of gene expression can be found in [8–13]. However, in most cases exact solutions are not possible and we have to use approximations. Some regulatory gene networks were analyzed by appropriate linearization scheme in [14].

We will now present a general generating function approach used later in the case of a self-repressing gene.

We define the generating function $F(z,t)$ corresponding to $f(n,t), n \geqslant 0$,

$$F(z,t) = \sum_{n=0}^{+\infty} z^n f(n,t). \tag{14}$$

Generating functions generate moments of probability distributions,

$$\frac{dF(z,t)}{dz}_{|z=1} = \langle n \rangle; \quad \frac{d^2F(z,t)}{dz^2}_{|z=1} = \langle n(n-1) \rangle, \tag{15}$$

where by $\langle x \rangle$ we denote the expected value of the random variable $x$ with respect to the probability distribution $f(n,t)$. We differentiate (14) with respect to $t$, use (12) and get

$$\frac{\partial}{\partial t} F(z,t) = (z-1)[k_0 F(z,t) - \gamma \frac{\partial F(z,t)}{\partial z}]. \tag{16}$$

Now we differentiate (16) once and twice with respect to $z$, use (15) and get

$$\frac{d\langle n\rangle}{dt} = k_0 - \gamma\langle n\rangle, \tag{17}$$
$$\frac{d\langle n(n-1)\rangle}{dt} = 2(k_0\langle n\rangle - \gamma\langle n(n-1)\rangle).$$

In the stationary state we obtain

$$\langle n\rangle = \frac{k_0}{\gamma}, \tag{18}$$
$$\langle n(n-1)\rangle = \frac{k_0}{\gamma}\langle n\rangle,$$

which gives us the following formula for the variance in the stationary state,

$$var(n) = \langle n^2\rangle - (\langle n\rangle)^2 = \langle n\rangle = \frac{k_0}{\gamma}. \tag{19}$$

We note that the variance is equal to the expected value which is not surprising because we mentioned before that the birth and death process has the Poisson stationary distribution.

**3.2. Self-repressing gene – a mean-field approximation at work.** It happens frequently that proteins regulate their own production. We will discuss here the repression - protein molecules may bind to a certain promoter region of their own DNA and thus decrease or completely stop the transcription. Therefore we will consider a stochastic model, where the gene (DNA) can be in two discrete states: unbound (on) denoted by 0 or bound (off) denoted by 1. In the generic case, the transcription rates for the on- and off-states are given by $k_0$ and $k_1$ respectively, but we set $k_1 = 0$ as it is often done, the protein degradation rate is denoted as before by $\gamma$, see Fig. 4. We consider a monomer binding and thus we assume that the binding rate is given by $\beta n$, the rate of switching the gene on (unbinding) is denoted by $\alpha$.

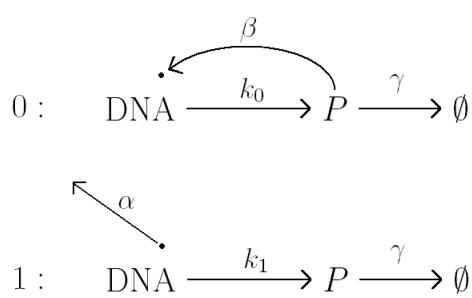


Figure 4: Self-repressing gene, the unbinding and binding rates are denoted by $\alpha$ and $\beta$, respectively, production rates by $k_i$, depending on the state of the gene, and the degradation rate by $\gamma$.

We introduce two probability mass functions, $f_i(n)$, $i \in \{0,1\}$ - a joint probability that there are $n$ protein molecules in the system and the gene (DNA) is in the state $i$. Now we can write the Master equation,

$$\begin{aligned}\frac{d}{dt}f_0(n,t) =& k_0[f_0(n-1) - f_0(n)] + \gamma[(n+1)f_0(n+1) - nf_0(n)] - \beta n f_0(n) + \alpha f_1(n),\\ \frac{d}{dt}f_1(n,t) =& \gamma[nf_1(n+1) - (n-1)f_1(n)] + \beta n f_0(n) - \alpha f_1(n),\end{aligned} \tag{20}$$

for $n \geqslant 1$, for $n = 0$ we have $\frac{d}{dt}f_0(0,t) = -k_0 f_0(0) + \gamma f_0(1)$ and $f_1(0,t) = 0$.

In the above equations we assumed that a bound protein molecule cannot degrade. Similar equations were considered in [12], where the formula for the stationary distribution of the number of protein molecules was derived in terms of the Kummer functions. Our goal here is to find an explicit expression for the variance of the number of protein molecules in the stationary state.

Let $A_0$ and $A_1$ be probabilities (frequencies) that the gene is unbound or bound respectively and the system is in the stationary state (i.e. $A_i = \sum_{n=0}^{+\infty} f_i(n), i = 0, 1$). Expressions $\langle n\rangle_i = \sum_{n=0}^{+\infty} n f_i(n)$ are expected numbers of protein molecules with respect to the probability mass functions $f_i(n)$. Obviously $\langle n\rangle = \langle n\rangle_0 + \langle n\rangle_1$. We proceed now exactly in the same way as in the previous section. We introduce two generating functions:

$$\begin{aligned}F_0(z,t) &= \sum_{n=0}^{+\infty} z^n f_0(n,t),\\ F_1(z,t) &= \sum_{n=0}^{+\infty} z^n f_1(n,t).\end{aligned} \tag{21}$$

and get the following system of algebraic equations for the moments in the stationary state:

$$\begin{cases} A_0 + A_1 = 1\\ \beta\langle n\rangle_0 - \alpha A_1 = 0\\ k_0 A_0 - \gamma\langle n\rangle_0 - \beta\langle n^2\rangle_0 + \alpha\langle n\rangle_1 = 0\\ \gamma A_1 - \gamma\langle n\rangle_1 + \beta\langle n^2\rangle_0 - \alpha\langle n\rangle_1 = 0\\ 2k_0\langle n\rangle_0 + 2\gamma\langle n\rangle_0 - 2\gamma\langle n^2\rangle_0 - \beta\langle n^2(n-1)\rangle_0 + \alpha\langle n(n-1)\rangle_1 = 0\\ -2\gamma A_1 + 4\gamma\langle n\rangle_1 - 2\gamma\langle n^2\rangle_1 + \beta\langle n^2(n-1)\rangle_0 - \alpha\langle n(n-1)\rangle_1 = 0 \end{cases} \tag{22}$$

The above system of equations is not closed (unlike in the case of unregulated gene expression analyzed in the previous section), equations for lower-order moments involve higher-order moments. Several concepts and techniques were developed to close such hierarchical systems of equations [15–17]. To deal with interactions present in auto-regulatory genetic systems, a mean-field approximation was introduced recently in [18]. Here we use it to close the system of equations (22). The idea is exactly the same as in the ferromagnetic Ising model. Namely, we replace $n$ in the switching term in (20) by its unknown expected value, that is instead of $\beta n f_0(n)$ we write $\beta\frac{\langle n\rangle_0}{A_0} f_0(n)$. This leads to a closed system of equations. Instead of (22) we get

$$\begin{cases} A_0 + A_1 = 1 \\ \beta\langle n\rangle_0 - \alpha A_1 = 0 \\ k_0 A_0 - \gamma\langle n\rangle_0 - \beta\frac{\langle n\rangle_0}{A_0}\langle n\rangle_0 + \alpha\langle n\rangle_1 = 0 \\ \gamma A_1 - \gamma\langle n\rangle_1 + \beta\frac{\langle n\rangle_0}{A_0}\langle n\rangle_0 - \alpha\langle n\rangle_1 = 0 \\ 2k_0\langle n\rangle_0 - 2\gamma\langle n(n-1)\rangle_0 - \beta\frac{\langle n\rangle_0}{A_0}\langle n(n-1)\rangle_0 + \alpha\langle n(n-1)\rangle_1 = 0 \\ -2\gamma A_1 + 2\gamma\langle n\rangle_1 - 2\gamma\langle n(n-1)\rangle_1 + \beta\frac{\langle n\rangle_0}{A_0}\langle n(n-1)\rangle_0 - \alpha\langle n(n-1)\rangle_1 = 0 \end{cases} \tag{23}$$

which we solve and obtain the self-consistent value for $\langle n\rangle_0$, and finally the expression for the variance $var(n)$ in the stationary state. In Fig. 5 we plot $var(n)$ as a function of $\log\omega$, where $\omega = \frac{\alpha}{\gamma}$ is the so-called adiabaticity parameter describing how fast is the gene switching with respect to the protein degradation.

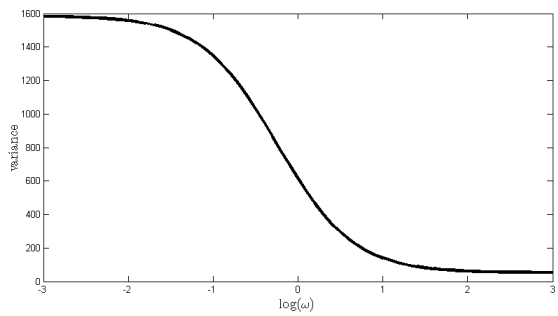


Figure 5: Variance of the number of proteins produced in the self-repressing system as a function of $\log(\omega)$, $\omega = \alpha/\gamma$.

We see that the variance is a decreasing function of the frequency of gene switching. We recently extended the mean-field approach to a model with two gene copies [19, 20].

The validity of the mean-field approximation was checked in two extreme cases: slow gene switching (where to get analytic results we used the conditional variance) and fast gene switching (where we used the so-called adiabatic approximation). We found that in both cases, the stationary variance of the number of protein molecules coincides with the mean-field approximation [20].

**Acknowledgments.** Paulina Szymańska was supported by the EU through the European Social Fund, contract number UDA-POKL.04.01.01-00-072/09-00.

## References

[1] Statistical Mechanics and Quantitative Biology on xxx.lanl.gov

[2] K. Huang, *Statistical Mechanics*, Wiley (1963).

[3] C. J. Thompson, *Mathematical Statistical Mechanics*, Princeton University Press (1972).

[4] S.-K. Ma, *Statistical Mechanics*, World Scientific, (1985).

[5] L. Onsager, *Crystal statistics. I. A two-dimensional model with an order-disorder transition*, Phys. Rev. 65: 117–149 (1944).

[6] T. Kepler and T. Elston, *Stochasticity in transcriptional regulation: origins, consequences, and mathematical representations*, Biophys. J. 81: 3116-3136 (2001).

[7] N. G. van Kampen, *Stochastic processes in physics and chemistry*, 2nd ed. Amsterdam: Elsevier (1997).

[8] M. Thattai and A. van Oudenaarden, *Intrinsic noise in gene regulatory networks*, Proc. Natl. Acad. Sci. USA. 98: 8614–8619 (2001).

[9] P. S. Swain, M. B. Elowitz, and E. D. Siggia, *Intrinsic and extrinsic contributions to stochasticity in gene expression*, Proc. Natl. Acad. Sci. USA. 99: 12795–12800 (2002).

[10] J. Paulsson, *Summing up the noise in gene networks*, Nature 427: 415–418 (2004).

[11] J. Paulsson, *Models of stochastic gene expression*, Phys. Life Rev. 2: 157–175 (2005).

[12] J. E. Hornos, D. Schultz, G. C. Innocentini, J. Wang, A. M. Walczak, J. N. Onuchic, and P. G. Wolynes, *Self-regulating gene: An exact solution*, Phys. Rev. E 72: 051907 (2005).

[13] P. Paszek, *Modeling stochasticity in gene regulation: characterization in the terms of the underlying distribution function*, Bull. Math. Biol. 69: 1567–1601 (2007).

[14] M. Komorowski, J. Miękisz, and A. Kierzek, *Translational repression contributes greater noise to gene expression than transcriptional repression*, Biophys. J. 96: 372–384 (2009).

[15] I. Nasell, *An extension of the moment closure method*, Theor. Pop. Biol. 64: 233–239 (2003).

[16] B. Barzel and O. Biham, *Binomial moment equations for stochastic reaction systems*, Phys. Rev. Lett. 106: 150602 (2011).

[17] B. Barzel, O. Biham, and R. Kupferman, *Analysis of the multiplane method for stochastic simulations of reaction networks with fluctuations*, Multiscale Model. Sim. 6: 963–982 (2011).

[18] J. Ohkubo, *Approximation scheme based on effective interactions for stochastic gene regulation*, Phys. Rev. E 83: 041915 (2010).

[19] P. Szymańska, *Modeling of self-regulating gene*, Master thesis, University of Warsaw (2011).

[20] J. Miękisz and P. Szymańska, *Gene expression in self-regulating systems with many alleles*, preprint (2012).

## O spinach i genach

**Streszczenie.** Naszym celem jest zrozumienie i przewidywanie zachowania się układów wielu oddziałujących obiektów, takich jak cząstki i spiny w fizyce statystycznej czy geny i białka w biologii molekularnej. Jako matematycy pragniemy udowadniać twierdzenia i wyprowadzać analityczne wzory. Bardzo szybko okazuje się, że w istotnych zastosowaniach jest to niemożliwe. Co robić? Część z nas ucieka w wyrafinowane symulacje komputerowe. Czy nie ma innej drogi? Czy jesteśmy ograniczeni do wyboru pomiędzy Matematyką i Mathematicą? Na pomoc przychodzi metoda samouzgodnionego pola średniego. Ferromagnetyczny model Isinga i samoregulujący się gen zilustrują nam tę niezwykle uniwersalną metodę otrzymywania przybliżonych rozwiązań analitycznych.

**Słowa kluczowe:** model Isinga, temperatura krytyczna, autoregulacja genu, przybliżenie pola średniego.

*Jacek Miękisz* was hiking Tatra Mountains at the age of minus 6 months and then he was born in Wrocław in 1956. He investigated interacting suspensions in ferrofluids and received Master degree in physics from Wrocław Technical University in 1979. Then he moved to Blacksburg, Virginia Tech, studied interacting spins in Ising models of ferromagnetism and got PhD in mathematics in 1984. He spent postdoctoral years at the University of Texas at Austin, in Louvain-la-Neuve and Leuven, studying interacting particles in lattice-gas models of quasicrystals. Now he works at the Institute of Applied Mathematics and Mechanics of the University of Warsaw and deals with interacting agents in evolutionary games and interacting proteins in genetic regulatory networks, sometimes with time delays.

*Paulina Szymańska* graduated from University of Warsaw in 2011, her master thesis in Mathematics under the supervision of Prof. Jacek Miękisz, entitled *Modeling of self-regulating gene* won the second prize in Competition for Best Students Work In Probability and Applied Mathematics, organized by the Polish Mathematical Society. Currently she is a PhD student at the College of Inter-Faculty Individual Studies in Mathematics and Natural Sciences of the University of Warsaw, working on stochastic models of gene expression and skiing in Tatra Mountains in her free time.

Jacek Miękisz
University of Warsaw
Institute of Applied Mathematics, Banacha 2, 02-097 Warsaw, Poland
*E-mail:* miekisz@mimuw.edu.pl
*URL:* www.mimuw.edu.pl/ miekisz

Paulina Szymańska
University of Warsaw
College of Inter-Faculty Individual Studies in Mathematics and Natural Sciences
*E-mail:* p.szymanska@mimuw.edu.pl

(Received: 4th of July 2012)