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COMMENTARY

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Social navigation hypothesis of depressive disorder disproven

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Abstract

Background Evolutionary psychiatry tries to explain paradoxical high psychiatric disorders and symptoms prevalence by means of evolutionary biology, but testing their hypotheses is often problematic. Social Navigation Hypothesis (SNH) belongs to the evolutionary hypotheses of depressive disorder. It assumes two mechanisms of increasing fitness by depressive signs and symptoms: social rumination function (depressed person is thinking intensely, trying to solve his/her social problems; in contrast to research demonstrating that depressed person usually manages with social challenges worse than healthy people) and social motivation function. The latter postulates that depressive behavior, by diminishing the fitness of surrounding people, forces them to help for depressed person to terminate the fitness-reducing episode. Social motivation function is discussed according to John Maynard Smith's idea of evolutionarily stable strategy. On that base two simple mathematical models are constructed.

Results SNH can theoretically describe an evolutionarily stable strategy (the precondition is the relation between the duration time of the episode and remission given by a certain formula), but the prediction of SNH (episode duration shortened with the patient's age) is contrary to epidemiological data. Presented models, based on simplistic mathematical assumptions, don't take into account kin selection and inclusive fitness.

Conclusions SNH cannot explain the high prevalence of depressive symptoms and depressive disorder. Falsification was possible only due to the mathematical formulation of the previously descriptively formulated hypothesis.

Keywords Depressive disorder, Evolutionarily stable strategy, Evolutionary psychiatry, Psychiatry

Introduction

Evolutionary psychiatry is one of three major approaches in modern psychiatry, next to cultural and computational ones [1], sometimes stated as foundational science for psychiatry [2, 3]. According to [2], one of the greatest obstacles in this field of science is the difficulty in testing hypotheses.

The current paper proposes some answer: although the evolutionary approach is different than the computational one, it sometimes needs mathematical apparatus to create testable hypotheses, e.g., linking depressive symptoms with some adaptational value in an environment of evolutionary adaptedness (mismatch account) or the modern world (persistence account) [4]. Advantages linked to the mathematical formulation of evolutionary psychiatry hypotheses can be pointed out in the case of a social navigation hypothesis (SNH).

SNH, formulated by Watson and Andrews [5], is one of the evolutionary hypotheses of depressive disorder, created to explain the great prevalence of depressive disorder and symptoms, describing the mentioned phenomena with the usage of concepts and tools of evolutionism.

Evolutionism constitutes a paradigm in biology nowadays. It originates from Charles Darwin's theory of evolution [6], which can be summarized in following presumptions: temporal variability of organism's traits, the possibility of common origin of different organisms,

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gradualism (changes rather gradual than radical, revolutionary), natural selection (particular individuals differ in fitness, i.e., quantitative representation of the ability to survival and reproduction, so specimen of greater fitness has statistically greater amount of offspring, inheriting profitable traits), population changes (evolution acts and creates adaptations by changes of traits proportion in population). Darwin's theory was modified on the basis of Mendelian genetics at the end of 1st and 2nd half of the XX century and subsequently on the ground of population genetics—in that way the modern evolutionary synthesis appeared [7]. Nowadays, evolutionism uses molecular biology, game theory, and other sciences.

Generally, natural selection diminishes the frequency of hereditary fitness-reducing traits, often causing the elimination of a given trait in a given population. Genetic factors play a significant role in the etiology of depressive disorder [8], what imply that natural selection acts on it. Patients diagnosed with depressive episodes suffer from low mood and energy, anhedonia, sleep disorders, and lack of appetite, experience difficulties in everyday activities and thinking, sometimes unable to do anything. Suicidal thoughts and tendencies might appear, which threaten the patient's life. A person suffering from a depressive episode has not only less chance for procreation and offspring but often is in life-threatening danger. To conclude, depressive episodes and their signs and symptoms diminish a patient's fitness.

As a result, natural selection should reduce the prevalence of depressive episodes, signs, and symptoms. However, this conclusion is inconsistent with the great prevalence of depressive episodes observed in different societies with a frequency of several dozen or so percent [9] and the growing negative effects of depressive disorder [10]. In that way a paradoxical deduction can be made: some non-obvious mechanism should exist, which at least compensates for the negative influence of depressive signs, symptoms, and episodes on fitness and in the effect of which fitness does not reduce its prevalence. Evolutionary hypotheses of depressive disorders try to explain that mechanism. Apart from SNH, these hypotheses include immunologic hypothesis (PATHOS-D, depressive signs and symptoms as a part of immunological response to infection, [11]), attachment hypothesis (reaction parallel to the behavior of infant left alone by his/her mother, hidden and waiting for her return, [12]), social position or social status hypotheses (avoidance of competition with persons having higher social position in group, [13]) and incentive hypothesis (fear, anticipation of depressive signs or symptoms prevent person from action, which can diminish his/her fitness, [14]). Some approaches within evolutionary hypotheses treat the depressive episode as if it increases fitness, the others recognize it as an effect of improper regulation of mechanisms increasing fitness.

Watson and Andrews' social navigation hypothesis

Watson and Andrews in their very interesting work [5] pointed out a very strong connection between the occurrence of depressive disorders and social problems. Accordingly, the authors tried to find the fitness-increasing mechanism of depressive signs and symptoms in social relations. They described two different social functions of depressive signs and symptoms with regard to their severity.

In the case of mild depressive signs and symptoms, not matching the criteria of major depressive disorder (MDD), the authors proposed social rumination function (SRf). They stated social problems are very absorbing, demanding elaborate analysis, but a person experiencing them, like every other one, has a limited amount of energy available. According to this view depressed person loses the ability to feel pleasure while performing other activities, cannot perform them and does not have enough energy to do so, sleeps shortly, and eats small amounts of food because he/she is absorbed in intensive intellectual work to resolve existing social problems.

However, as Varga [4] pointed out, sometimes there is no social problem triggering depressive episode or the existing trouble sometimes involves moral, cultural, or religious issues and cannot be executed by a calculative choice between options, and hardly need a few months of thinking about them, as usually, the episode lasts. What is more, rumination usually impairs problem-solving and exacerbates the episode.

In other papers two more reasons why this argument cannot explain the high prevalence of depressive disorder can be found. Firstly, the authors of SNH stated that SRf is relevant to mild states, not classified as MDD. Of course, it is not easy to draw a line between a mild decline of mood or energy and a more severe clinical state, meeting the criteria of a mood disorder, but—what constitutes the second reason—the more severe the depressive signs and symptoms are, the weaker the cognitive functions. Attention, ability to learn, memory, linguistic fluency [15], and the theory of mind [16] work in a depressed person worse than in a healthy one. Depressed people experience greater difficulties in noticing assistance offered by surrounding people [17], which seems to be meaningful in the context of the proposed social function of depressive signs or symptoms. Watson and Andrews did not cite in their publication any work giving evidence of postulated greater solving problem ability in depressed persons and Varga [4] stated there is no such paper. Nowadays such experiments exist (for example depressed persons have a

greater inclination to reject unjust offers in ultimatum games, [18]), but depressed persons' greater problem-solving abilities were observed only in the case of particularly scheduled experiments (if the researcher has created the situation, in which majority of participants exhibit decision bias connected with a too optimistic assessment of the situation, depressed person, perceiving world and future in a more pessimistic way, will tend to construct a more adequate representation of situation). In every other situation, depressiveness worsens the ability to cope with problems. Two preceding arguments make explaining of above-mentioned paradox by SRf impossible.

The second explanation proposed by Watson and Andrews, social motivation function (SMf), concerns mainly major depression. It describes depressive signs and symptoms in a manner similar to older concepts of crying for help. It is based on the fact that people live in society, owing its functioning mostly to the cooperation of individuals, depending on each other. As a result—if one person finds himself/herself in an unprofitable situation or need of a change in his/her social niche, surrounding people might not be interested in assisting him/her in these alterations, because these changes might be connected with loss of benefits obtained from the previous relationship. Surroundings might not respond to the cry for help, and what is more—they might even (not necessarily consciously) act against changing one's unprofitable situation. If the relationship connecting two people provides two-sided benefits, alteration of that relationship's character by one of them might deprive the second person of these profits. Lower energy, slower thinking, and difficulties in everyday activities might be a handicap of one's functioning and—in the small community (such as ones in which humanity lived through thousands of years before present times—the environment of evolutionary adaptedness)—this might impair the functioning of all group (including surrounding people). What is more, low mood spreads among the people closest to a depressed person. As a result, their fitness diminishes. Nowadays, it is easy to terminate harmful relationships by separation, changing one's job, or moving to another place. In the society of hunter-gatherers living in an environment of evolutionary adaptedness that was not possible. Lonely individuals had to die. Hunting for big game requires the cooperation of men and—in the case of (very often) failure—providing food by cooperating and gathering women. Change of social group was dangerous because neighboring groups often were hostile—nowadays hostility towards other nations or other sport team's fans still exists. Likely, violent aggression between groups of the closest human relative, the common chimpanzee, is observed.

Criticism

SNH became the object of criticism [4, 19], partially contradicted by other authors [20]. SNH opponents pointed out that the occurrence of MDD only in part of the population makes it impossible to consider it an adaptation, which—according to the mentioned author—should be observed in every member of a given population. What is more, depressed people are perceived as unattractive partners, so relations with them might be terminated more often. A response to the first argument is that some features are expressed only in specific conditions. Furthermore, every feature appears firstly in one individual and needs a period of time to spread throughout the whole population (or to be superseded).

The second of the mentioned arguments is more interesting, but it was analyzed very cursorily in scientific literature. According to SNH, a depressed person reduces the fitness of people he/she is in relation with, forcing them to help him/her. Should such extortion be favored by natural selection (acting in this case on an individual level, because a depressed person's behavior has a deleterious effect on adjacent people and all groups), it has to cause obtaining help. But this description omits the second person in the relationship, who can give aid or break up relation and who is a subject of evolution too. He or she has a choice: help or not and this choice has an effect on his/her fitness. Giving aid is also analyzed in evolutionary categories and in this analysis, there is an important role of the concept of evolutionarily stable strategy (ESS).

Evolutionarily stable strategies

The idea of ESS derives from the works of John Maynard Smith, [21] p. 2, who applied game theory to describe the evolution of interactions between organisms (or units of other biological organization levels). EES is a form of Nash equilibrium, which means strategy, which, if fixed in a given population, cannot be displaced by new traits caused by mutation (unless environmental conditions change). It means that natural selection does not cause fixation of strategies guarantying the best fitness for the population or individuals, but fixation of ESS, e.g., group composed of absolute altruists (individuals helping others regardless of situation) would have greater fitness (considering group level of natural selection) than a group of competing individuals. Every individual in every situation would receive aid, which would increase his/her fitness and the overall fitness of the group. But consider the appearance of one competing individual in this absolute altruists' group—the newcomer would abuse their altruism easily and reach greater fitness (despite the fact of a decrease in overall group fitness).

The competition trait would be inherited by the offspring of new individuals and would spread through the population in the next generations. As a result, the trait of absolute altruism would be lost—because it is not ESS. Describing analogous situations, evolutionary biology ascribes to individuals' strategies some payoffs (understood as fitness change, positive, negative, or equal to 0). Sometimes it allows us to assess which strategy is ESS or to which value the frequencies of particular strategies, realized by individuals alternately, will tend. This approach is especially useful in the case of frequency-dependent fitness [21].

Depressive episode model

The concept of ESS can be applied to the description of the behavior of a person in relation to a depressed individual. The simplest case is the relation between a healthy person (let he/she be named person 2) and a person suffering from a depressive episode (person 1).

To apply the ESS concept to analyze social motivation function Maynard Smith's idea needs to be modified. Originally fitness w after a single behavior (realizing given strategy) is given by formula:

$$w = w_0 + E, \tag{1}$$

where w_0 is initial fitness and E (or Δw) is the payoff ascribed to a strategy. However, the answer of person 2 to the behavior of person 1 can be hardly described as a single behavior bearing a single payoff, it is rather a continuously realized strategy, dissolved in some time. It cannot be ascribed to a single payoff, because the payoff, which is fitness change, depends on depressive episode characteristics—mainly how long the episode lasts and maybe how severe the episode is. However, the discussed reasoning could be applied in the case of (1) the replacement of fixed payoff values by functions of time in which the given strategy is realized and (2) the introduction of additional values obtained by dividing fitness change by time in which the change happens:

$$\omega = \Delta w/t \tag{2}$$

The introduced value ω represents the growth or descent of fitness per time and is linked to the effectiveness of the realized strategy. Then fitness change or total payoff is given by formula $\Delta w = \omega t$, which means linear function, whose argument is time in which the given strategy is realized (this assumption is a simplification but using exact formulae $\omega = dw/dt$ and $\Delta w = \int \omega(t)dt$ would made presented model too complicated, see *Limitations* section).

Then, if person 2 in relation to person 1 behaves towards him/her in a way described by SMH, person 2 must choose from 2 strategies:

- Firstly, person 2 can give in and help, which exposes him/her to bear the costs throughout the whole time of depressive episode (t_{ep}). He or she is entitled to expect a continuation of relation in remission (let signify the time of relation lasting after remission by t_r).
- On the other hand, person 2 can end the relation—he/she would not be exposed to help-associated fitness decrease, but life in desolation links to poorer fitness than living in relation (he/she is not entitled to expect continuation of relation in the remission).

Having assigned ω_P to the aiming help strategy, ω_r to staying in relation (with an undepressed person), and ω_s to solitary life (e.g., after the termination of relation), one can describe fitness changes in mentioned conditions by inequality:

$$\omega_r > \omega_s > \omega_p \tag{3}$$

The strategy of helping during depressive episodes and continuing relation in remission is connected with fitness change amounting to:

$$\Delta w_P = \omega_p t_{ep} + \omega_r t_r \tag{4}$$

When the fitness change in the case of ending relation amounts to

$$\Delta w_s = \omega_s t_{ep} + \omega_s t_r \tag{5}$$

Now the question asked above, why should person 2 help the depressed person rather than terminate the relation, can be answered strictly: natural selection promotes helping if $\Delta w_P > \Delta w_s$; it promotes termination of relation when $\Delta w_P < \Delta w_s$; and in the case of equality of Δw_P and Δw_s choice of first or the second strategy is a neutral trait (does not affect fitness). Considering the last, boundary situation and substituting in the equality $\Delta w_P = \Delta w_s$ formulae (4) and (5), one can obtain $\omega_P t_{ep} + \omega_r t_r = \omega_s t_{ep} + \omega_s t_r$, and from it:

$$t_{ep} = t_r * (\omega_r - \omega_s)/(\omega_s - \omega_P) \tag{6}$$

Interpretation

Equation (6) represents some relation between the time of depressive episode duration and the time of relationship lasting after alleviating depressive symptoms. It describes the condition under which the choice of helping strategy or termination strategy is neutral. In the case of the faster ending of depressive episodes natural selection promotes helping, in the case of longer depressive episodes it promotes ending the relationship.

However, John Maynard Smith has already noticed that none of "pure" strategies might turn out evolutionarily

stable strategy. ESS is often proved to be a mixed strategy, named Observer strategy. An individual realizing this strategy estimates the situation and—depending on his/her estimation—realizes sometimes the former strategy, sometimes the latter, e.g., males competing for territory or females tending to combat with smaller specimens and avoiding conflict with bigger ones. In the case described by SNH natural selection promotes help in relatively short-lasting depressive episodes followed by long-lasting relationships in remission. Termination of relation is promoted when the depressive episode lasts a long time and the following relation in remission is short. Predicting the time of the episode is useful, but not necessary after taking into consideration the possibility of terminating the relationship in the course of a depressive episode if it protracts (but it means loss of costs that have been already invested in the relation by person 2). Now let us consider the time after breaking the relationship. As mentioned above, in the environment of evolutionary adaptedness people terminated relations by changing groups very rarely, only in extraordinary conditions (however, fitness loss caused by the behavior of other group members can be acknowledged as such a condition). On the other hand, mortality was significant, and lifespan was much lower than nowadays. Relation was terminated usually by the death of one of the people in relation. The health state of a given person was often difficult to assess, so the best predictor of the risk of being closer to death was one's advanced age. The probability that depressed person 1 will be able to pay off debt in remission after a depressive episode decreases with the increase of person 1 age. Since the rough estimation of the same group members' age is not especially difficult, natural selection should promote dependence of strategy chosen by person 2 (aiding help or finishing relation) on the age of person 1. What is more, from the obtained formula one can infer that if person 1 is at an advanced age, he or she cannot extort on surroundings aiming help by long-lasting depressive episode—the older person 1 is and the longer his/her depressive episode lasts, the bigger is the probability of terminating relation by person 2. As a result, if a depressive episode serves to achieve profit by forcing help from a person in relation, as SNH postulates, the duration time of the depressive episode will negatively correlate with the age of the depressed person. Such correlation will not be expected only in the case of ω value not independent of the time of the episode and predicted time of relation in the future.

Recurrent depressive disorder model

The model described above represents the relationship between two people, one of them suffering from a depressive episode. However, in the real world, nearly every person keeps relationships with more than one person and depressed people might be diagnosed not only with a depressive episode but also with a recurrent depressive disorder. The depressive episode model has to be modified to describe such a population. Having assumed the relation of person 1 with n surrounding people (all of them affect the fitness of person 1) and introducing the new symbol Ω on total fitness change in time, one can obtain:

$$\Delta w = \Omega t = \Sigma_{i=1}^{n} \omega_i t \tag{7}$$

Among that n people signs or symptoms allowing for diagnosing depressive episodes or recurrent depressive disorder will be observed at any time in cn, where c is morbidity. The proportion of individuals in remission in the group of people suffering at any time from a depressive episode of average duration time t_{ep} and with average remission duration time t_{rem} is expressed by fraction $t_{\mathrm{rem}}/(t_{\mathrm{rem}}+t_{\mathrm{ep}})$, when the proportion of individuals currently suffering from the depressive episode is characterized by $t_{\mathrm{ep}}/(t_{\mathrm{rem}}+t_{\mathrm{ep}})$. Amongst the latter group probability of receiving help (what means maintaining relation) is p and the probability of receiving no help (in other words, terminating the relation, which will not be renewed in remission as unserviceable for person 1) is equal to $1\text{-}p$. The estimation is rough, but it allows describing fitness change by a somewhat simpler formula, invariable in the time:

$$\Omega = (1-c)n\omega_r + cnt_{ep}/(t_{rem}+t_{ep}) * (p\omega_p + (1-p)\omega_s) + cnt_{rem}/(t_{rem}+t_{ep}) * (p\omega_r + (1-p)\omega_s) \tag{8}$$

The first addend represents a relation with healthy individuals, the second—with people suffering from depressive episodes in the course of recurrent depressive disorder, and the third term—individuals in remission in the course of recurrent depressive disorder. This formula can be reduced to

$$\Omega/cn = (1/c-1)\omega_r + p * (\omega_p t_{ep} + \omega_r\ t_{rem})/(t_{rem}+t_{ep}) + (1-p)\omega_s \tag{9}$$

To simplify the formula was divided bilaterally by cn to move the repeating multiplier to the less expanded left side of the equation. Consecutive addends represent healthy individuals, suffering individuals staying in

relation, and people after the termination of relation. By transforming the equation another time one can get a formula describing fitness change as a linear function of the probability of maintaining relation:

$$\Omega/cn = ((\omega_p\ t_{ep} + \omega_r\ t_{rem})/(t_{rem}\ +\ t_{ep}) - \omega_s) * p\ +\ ((1/c - 1)\omega_r\ +\ \omega_s) \tag{10}$$

Fitness change increases with the growth of the probability of maintaining relationships in depressive episodes. And from the foregoing formula (10): fitness change increases with the growth of relation maintaining probability, if factor $(\omega_p\ t_{ep} + \omega_r\ t_{rem})/(t_{rem} + t_{ep})$-$\omega_s$, multiplied by p, is positive. Whereas this factor equals 0 (boundary situation), fitness change does not depend on p:

$$(\omega_p\ t_{ep}\ +\ \omega_r\ t_{rem})/(t_{rem}\ +\ t_{ep}) - \omega s = 0 \tag{11}$$

This formula can be transformed to

$$t_{ep} = t_{rem} * (\omega_r\ -\ \omega_s)/(\omega_s - \omega_p) \tag{12}$$

Formula (12) has the same form as formula (6) of the above-mentioned depressive episode model. As a result, this model can be converted into a depressive episode model by changing the average depressive episode duration time into the only depressive episode duration time and the average time of remission between depressive episodes into the duration of relation in remission after finishing the only episode and by assuming only one relation between suffering person 1 ($c=1$) and person 2 ($n=1$). The question of how it affects the conclusions from the depressive episode model has to be answered. The possibility of predicting the duration of relation with person 1 by person 2 has been already pointed out. In the case of remission between depressive episodes, there is not such a possibility. However, some fraction of the relation proceeds towards the depressive episode model, ending with the death of person 1 (or 2, and sometimes the age of person 1 makes the expectation of death a reliable prediction), and as a result, conclusions from the analysis of depressive episode model are still valid, but take less perceptible effect.

Limitations

- Presented models assumed a simple relation between the time of realizing a given strategy and fitness change in this time, $\Delta w = \omega t$. Actually, ω does not have to be constant. The exact formulae considering it are: $\omega_t = dw/dt$ and $\Delta w = \int \omega_t(t)dt$. Fitness change calculating requires in such conditions integrating (calculating integral from the time when realizing strategy had started to the time it ended), which is not possible without accurate knowledge of function $\omega_t(t)$ and very difficult apart from few preferable theoretical cases. Hence, let us assume that the foregoing models are based on the approximated ω definition: $\omega = \Delta w/t = (\int \omega_t(t)dt)/t$. In the result of such simplification actually exact linear relation of t_{ep} and t_{rem} is not expected, using such constructed model one should predict only the correlation between t_{ep} and t_{rem} (and lack of such correlation would falsify models and in consequence SNH).
- Presented models do not concern kin selection and inclusive fitness. Describing these phenomena requires different mathematical apparatus, which cannot be reconciled with the simplicity of presented models.

Clinical data and conclusions

The described model can be used to falsify or corroborate SNH. What is more, data enabling testing of the hypothesis had been already published.

Research on a huge group of Chinese women performed by Yang et colleagues [22] showed a connection between the early onset of major depression disorder and longer duration, more numerous episodes, and longer duration of longest lasting episodes. Differences in symptom severity between early onset and other than early-onset major depression were not observed. Depressive episodes characterized by longer duration, but no greater severity and greater risk of relapse had been described earlier in early-onset MDD [23, 24]. According to other work [25] in persons who contact illness earlier suicidal thoughts and suicidal attempts were found more frequently. The heterogeneity of MDD depending on the age of onset was even proposed [23]. These data correspond with the implication of the discussed models partially. Episodes of greater duration at a young age would affect research results similarly; people of younger age (what means: of a greater chance for obtaining help) could actually force help more intensely in the case of reluctant surroundings. On the other hand, according to the foregoing argumentation depressive episodes of aging person should shorten, which was not observed. What is more, depressive episodes in patients of advanced age are characterized by longer duration and worse response to therapy [26].

These observations and the abovementioned SNH models are contradictory. Prolonged depressive episodes in older people cannot be reconciled with and cannot be explained by SNH, it cannot increase fitness

in mechanisms postulated by Watson and Andrews. As a result, SNH is not likely to become a theory explaining the origin of all or even the majority of depressive episodes. SNH can be formulated as a falsifiable hypothesis, consistent with the modern theory of evolution. Based on SNH one can describe theoretically evolutionarily stable strategies, towards which natural selection would modify the behavior of population members. Actually, some depressive episodes (especially at a young age) can increase fitness, but others cannot, contrary to SNH. Fitness increase at a young age can be associated with an evolutionary understanding of Bowlby's attachment theory [12]—depressive signs increase the fitness of a child left by a mother because it is safer to stay in a shelter and wait for the mother than engage in any activity lonely. The appearance of depressive signs or symptoms in older patients is explained by erroneous, excessive work of regulatory mechanisms, beneficial in other conditions—evolution does not create ideal solutions. Mechanisms saving life at the beginning of ontogenesis but failing later, sometimes after successful reproduction, increase fitness. SNH can be formulated in the same way—recognize the majority of depressive episodes as the effect of dysregulation of usually beneficial mechanisms. However, SNH does not allow ascribing positive adaptation value to depressive episodes in general, which is postulated by Watson and Andrews. Even not allowing to explain paradoxical high depressive disorder prevalence, Watson and Andrews' proposition remains an important and interesting voice in scientific debate and it belongs to the most inspiring concepts, which, although falsified, contributed to introducing a new method, in this case analysis of evolutionarily stable strategies, into evolutionary psychiatry.

SNH after its original formulation was not popular. The authors formulated in a descriptive, non-mathematical way, what made falsification or corroboration of the hypothesis impossible. Only strict mathematical formulation enabled it. In conclusion, the same should be stated about other evolutionary hypotheses in psychiatry—only mathematically formulated ones can be tested, what makes them valuable. Accordingly, there is no evolutionary psychiatry without modern evolutionary synthesis and appropriate mathematical apparatus.

Abbreviations

SNH	Social Navigation Hypothesis
MDD	Major depressive disorder
SRf	Social rumination function
SMf	Social motivation function
ESS	Evolutionarily stable strategy

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