Psychosomatic symptoms as biomarkers: Transcending the psyche-soma dichotomy

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Following the advancement in understanding dynamical systems, the author presents a novel metaphor of psychosomatic symptoms as low-dimensional biomarkers. This metaphor, which transcends the old binary of psyche-soma, resonates with classical psychoanalytic concepts and with Matte-Blanco’s idea of repetition as indicative of dimensionality reduction. The relevance of this metaphor for explanation, diagnosis, and treatment is illustrated through a case study of a male patient suffering from hyperprolactinemia. (Bulletin of the Menninger Clinic, 74[1], 63-77)

Psyche and soma relationships have been a source of concern for generations of researchers and mental health practitioners. However, despite the advancements in science, these relationships are usually discussed through a limited number of old philosophical resources (e.g., dualism) that seem to be of minor use for the practitioner. This difficulty may be attributed to the limited conceptual “toolkit” available to psychosomatic research. As argued by Meissner (2008, p. 284):

\[\text{at this stage of our methodological sophistication in studying the mind-brain relation, we cannot go beyond a dual methodology, each part of which addresses its own proper domain, and only by further integration and correlation of their respective findings will we be able to achieve a more comprehensive and complete understanding of}\]

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how the brain works and what it does, and particularly how it produces mentation, both conscious and unconscious. (p. 284)

Recent advancements in understanding complex dynamical systems and the way they may enrich classical psychoanalytic ideas (e.g., Neuman, 2009a, 2009b, 2009c) allow us to transcend the “dual methodology” and to examine psyche-soma relationships in a new light. The aim of this short article is (1) to introduce the idea of dimensionality and its relevance for the study of the mind, (2) to present the metaphor of the psychosomatic symptom as a low-dimensional biomarker, and (3) to illustrate the benefits of this conceptualization through a case study. In order to avoid cognitive overload this article focuses mainly on the theoretical aspect. Future articles will further detail the practical implications.

The Mind as a High-Dimensional Space

“Dimensionality” is a central concept in diverse scientific fields, such as physics and information sciences. However, with the exception of rare cases, this concept has received minor and scant attention in the psychoanalytic literature. What is the meaning of “dimension” and why is it important to understand it? Informally, the dimension of a space is the minimum number of “coordinates” we need in order to specify a point within it. For example, if we would like to specify the position of Smiley on a plane, then we need two coordinates/dimensions: (1) height and (2) width (Figure 1).

The position of Smiley can be specified by using a vector, a directed line segment. The tail of the vector is located at the origin of the coordinate system and its tip, the terminal point, at Smiley’s location. If we add a third dimension, time, for instance, then the position of Smiley can be specified in two time points, and the vector connecting these points/vectors is the “trajectory” of the system. The idea of specifying a point through coordinates is not limited to concrete spaces or objects and may be extended to the mental realm. By using the term mental we are not obliged to use the old psyche-soma dichotomy. Mental experience is the experience that is primarily observed from the first-person perspective. That is, the defining character of the mental realm is the perspective from which it is primarily experienced. For example, wine tasting is a
mental phenomenon because it is primarily experienced from the first-person perspective. We can describe the taste of a certain wine by using one dimension—interestingness, for instance—ranging from “0” (not interesting at all) to “10” (extremely interesting). The use of a single dimension might be overly simplistic and we may want to describe the taste of a certain wine by adding a second dimension such as “aroma,” a third dimension such as “softness,” or any number of dimensions. Describing the taste of a certain wine through $n$ independent dimensions is done by specifying a point residing in the space that is constructed by the $n$ dimensions.

The mental space is too rich to be exhausted by a three-dimensional space. It is a high-dimensional space in which our experiences reside and relate in a way that is beyond our ability to visualize and fully understand. As argued by Bion (1965): “Mental space is so vast compared with any realization of three-dimensional space that the patient’s capacity for emotion is felt to be lost because emotion itself is felt to drain away and be lost in the immensity” (p. 12). In this space, emotion is “lost” because it simply cannot be represented in the high-dimensional space.

Why should we care about dimensionality and the mind as a high-dimensional mental space? Grotstein (1978) argued that “we must have a theory which can help us conceptualize the space … in which feelings and thoughts … can be examined” (p. 55). I (2009b, 2009c) have recently discussed this space. I argue that the mental space is so vast that in order for the mind to know itself, it must use

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**Figure 1.** A two-dimensional space.
low-dimensional space. In this context, psychosomatic symptoms may be interpreted as “biomarkers”—lower-dimensional signals that are projections of the higher-dimensional space. To explain this idea, I now turn to the mind as a dynamical system.

The Mind as a Dynamical System

Previously, I (2009b, 2009c) introduced the idea of the mind as a high-dimensional mental space. However, the mind is not a static space but a system that unfolds over time. It is a dynamical system. In this section, I would like to introduce the idea of the mind as a complex dynamical system.

A dynamical system is a system that unfolds over time. In a very general sense, such a system can be described by its “state variables” \( x^1(t), x^2(t), \ldots, x^d(t) \), where the superscript numbers indicate the components of the system. These state variables are the dimensions of the system. For example, the brain may be considered a dynamic system (e.g., Faure & Korn, 2001; Skarada & Freeman, 1989) where its state variables indicate active neurons. The \( d \) state variables at time \( t \) form a “vector” in a \( d \)-dimensional space that is called the “phase space.” The temporary succession of the phase space vectors forms a trajectory (phase space trajectory or orbit) (Marwan, Romano, Thierl, & Kurths, 2004), which is actually an abstract way of representing the system’s dynamics. Previously, I (2009b, 2009c) illustrated the state variables through the brain’s neurons. However, at a higher level of analysis, the state variables may correspond to semantic content that has been shown to underlie even unconscious processing (Van den Bussche, Van de Noortgate, & Reynvoet, 2009). In this context, the phase space trajectory actually represents our subjective, first-person experience as it unfolds over time, whether consciously or unconsciously. For instance, during a therapeutic session a patient usually introspects through the use of words (e.g., “painful”). These words are coordinates through which the experience is specified as a point in a high-dimensional mental space. The way this experience/point unfolds over the session may be described as the trajectory of the patient’s mental content.
In practice, the observation of a real dynamic process usually does not yield all possible state variables of the system due to (1) lack of knowledge of the system’s components and/or (2) problems of measuring the system. In other words, our ability to gain knowledge of the system’s trajectory is limited because (1) we are not familiar with all the relevant variables/dimensions constituting the phase space and/or (2) we cannot measure them all. Following the previous example, we may say that the patient is limited both in elucidating the dimensions of his or her experience and in his or her ability to “measure” them. The mind is probably limited in gaining direct access (i.e., self-observation) to its phase space trajectory because it is subject to the same problems of measurement presented above (Neuman, 2009c). In fact, there are some very good reasons to suspect that self-consciousness in its ultimate form is impossible (Neuman & Tamir, in press). In this context, the ancient Greek imperative “Know thyself!” is impossible because fully knowing my “self” is a utopian task. However, the previously mentioned constraints of knowledge and measurement of a high-dimensional dynamic system must be pragmatically addressed in order to provide the “mind” with the ability to monitor its activity. Neuman and Nave (2009) argue that the brain “solved” this problem similarly to the way physics solved the problem of measuring high-dimensional dynamic systems. This solution is detailed below.

How to understand a High-Dimensional Dynamical System?

Scientists usually face severe difficulties in studying complex systems in which variables are coupled in a synergetic way. However, we recently realized that the coupling of the variables is not only a source of difficulty but also a source of hope because the behavior of the system as a whole can be studied from the behavior of a single variable. Although the original phase space trajectory of the system cannot be structured, under several assumptions an equivalent phase space trajectory, which preserves the topological structures of the original phase space trajectory, can be reconstructed using only one observation. Physics has developed methodology for “reconstructing” the phase space trajectory of the system by measuring only one variable as it unfolds over time (Broomhead &
This logic can be illustrated using the following example of an ECG signal. The analogy to mental content is straightforward.

A one-dimensional representation of voltage as a function of time is presented in Figure 2A (Webber & Zbilut, 2005, p. 33), where the X-axis denotes time and the Y-axis the amplitude.

This signal, however, is a simple projection of the signal from a higher dimensional space, as the “ECG derives from summed cardiac potentials that move simultaneously in three dimensions (frontal, sagittal, and horizontal planes)” (Webber & Zbilut, 2005, p. 33). To represent the ECG in a three-dimensional space, it is
necessary to simultaneously record electrical potentials in three orthogonal planes from the subject” (Webber & Zbilut, 2005, p. 69). However, Takens (cited in Webber & Zbilut, 2005) has shown that “the topological features of any higher-dimensional system consisting of multiple coupled variables can be reconstructed from but a single measured variable of that system” (2005, pp. 33–34).

This remarkable idea cannot be trivially translated into a physical procedure of measurement because questions concerning the measurement process (e.g., how to determine the embedding dimension) are far from trivial (e.g., Cao, 1997; Kennerl, Brown, & Abarbanel, 1992). Practically, this reconstruction can be conducted by defining a time lag (i.e., a delay), plotting the one-dimensional data against itself \( n \) times, thus creating an \( n \)-dimensional space in which recurrent points are used for diagnosing the dynamics of the system (Webber & Zbilut, 2005; see Figure 2B). In Figure 2B, we can see the three-dimensional space created by plotting the original time line against itself twice by using two delays: plus 10 ms and plus 20 ms. The points in this three-dimensional space are recurrent points, where the same (or similar) values repeat.

In this context, recurrent points of a low-dimensional signal are used in order to detect a high-dimensional pattern in the data.

Repetition and Dimensionality Reduction

The idea of detecting recurrent points for the reconstruction of a dynamic pattern in a high-dimensional phase space is not the exclusive revelation of physicists. In fact, a long time before physics discussed this issue, Matte-Blanco (1975; 1984), insightfully argued that repetition is an indication of dimensionality-reduction from a higher-dimensional mental space. Matte-Blanco realized that when projecting a high-dimensional structure into a lower-dimensional space, some information is lost and repetition is necessary to compensate for this loss. For instance, if we consider the triangle in Figure 3A, it is a geometrical figure in two dimensions. When projected into a line, the triangle may look like the line in Figure 3B.

In order to represent the triangle, we have had to represent point A, a zero-dimensional space, twice. This repetition is evident in our
mental space. For instance, Matte-Blanco (1984) considered the dream to be a “hyperspace of thought” (p. 16) where repetition is indicative of dimensionality reduction. This idea resonates with Isakower (1938, cited in Grotstein, 1978, p. 55), who argued that the dream must be explained as the projection on a screen surface. As (2009c) have explained: "The dream, as a projection of a high-dimensional mental space onto the ‘surface’ of symbolic substitute, is characterized by condensation, which is the inevitable result of dimensionality reduction." The same is true for repetition. Therefore, repetition may indicate that we are dealing with a lower-level projection of a higher-dimensional mental space. If repetition is an indication of dimensionality reduction, then by analyzing the pattern of repetition we may reconstruct the high-dimensional space from which the low-level signal has originated.

Adopting Matte-Blanco’s insight as well as Takens’ (cited in Webber & Zbilut, 2005) theorem, I would like to suggest that a psychosomatic symptom may be interpreted as a low-dimensional projection of a high-dimensional space. Through its repetition this symptom provides information for reconstructing a phase space trajectory that is equivalent to the original phase space trajectory—the “mental space”—from which it originated.

Figure 3A and B. The triangle and the triangle unfolded into a line.
Psychosomatic Symptoms as Biomarkers

Symptoms may be described as *biomarkers*. “A simple biomarker is informative when it faithfully signifies for us the complex factors from which the biomarker emerged” (Cohen, 2007, pp. 246-247). The biomarker can signal to the self or to the others (1) through a significant deviation from its base value and (2) through the temporal pattern of its appearance. For example, prolactin may be considered a biomarker. As will be later illustrated, a high level of prolactin in males may be considered a one-dimensional vector whose pattern of appearance over time may be indicative of the “mental” system’s dynamics, the “complex factors” from which it emerged.

While the early seminal studies of Breuer and Freud on hysteria (Freud & Breuer, 2000) emphasized the content of a psychosomatic symptom as a way of understanding mental *content*, the dynamic perspective presented in this article urges us to examine the way in which *the symptom unfolds over time*. Therefore, the biomarker should be studied not only metaphorically by analyzing its content (e.g., vomiting as a sign of rejection) but also as a unique functional medium that may provide us with information on the high-dimensional phase space trajectory from which the symptom emerged. In other words, we should also study the dynamics of the symptom. For example, a burst of the symptom followed by a minor relaxation may be attributed to “exogenous” variables (Crane & Sornette, 2008) such as contextual stress. In contrast, a burst of the symptom followed by a sharp relaxation may be attributed to an endogenous cause, whether “somatic” or “mental.” Another example: Through the analysis of repetition, the methodology of Recurrence Quantification Analysis (RQA; Webber & Zbilut, 2005) may provide some hints as to whether the dynamics of the system is chaotic, random, or simply deterministic. This information is highly relevant for psychosomatic diagnosis. If the repetition of the symptom is “chaotic,” then we can hypothesize the existence of a traumatic event that perturbed the system in a deterministic, albeit unpredictable, way. These ideas are illustrated in the next section.
Prolactin as Biomarker

As argued by Balon (2009), sexual dysfunction “seems to be a prime example of the interdependence of psychosocial and biologic/physiologic factors (p. 69). However, the “psychosomatic medicine literature is almost devoid of publications addressing human sexuality” (p. 7). This avoidance may be attributed to the tendency of medicine to seek somatic correlates for sexual dysfunction and for the tendency of conventional psychotherapy to seek mental correlates (e.g., stress) for sexual dysfunction. What is “in between” the soma and the psyche remains, as Balon argues, “a neglected area.” Following Balon, I will use the case of hyperprolactinemia to illustrate this theorization.

Prolactin (PRL) is a polypeptide hormone that is actually a cytokine (Goffin, Binart, Touraine, & Kelly, 2002, p. 48). This fact cannot be underestimated as cytokines are actually signs intensively communicated, for example, by the immune system (Neuman, 2008). In this sense, prolactin cannot be considered simply a functional signal per se but as a sign with “up to 300 separate functions or molecules” (Goffin et al., 2002, p. 51) that are interpreted in context. As such, it is a perfect candidate for functioning as a psychosomatic biomarker.

Normal prolactin levels in men are approximately less than 20 ng/mL (or 375 mU/L (Todd, 2005). Abnormal levels of prolactin (i.e., hyperprolactinemia) in men are characterized by several symptoms of hypogonadism such as loss of libido, impotence, and infertility (Gilliam, Molitch, Lombardi, & Colao, 2006; Todd, 2005). For men, hypogonadism is a term that describes failure of the testes to produce testosterone. There are different possible sources of this failure, such as a dysfunction of the testes or the brain areas that control the production of the testosterone (e.g., the pituitary gland). Although hyperprolactinemia is associated with symptoms of hypogonadism, it does not necessarily have to be associated with significantly lower levels of testosterone, as will be illustrated through the clinical case.

In a case of a burst of significantly higher levels of prolactin, we may suspect, at least as a relevant hypothesis, that the prolactin functions as a psychosomatic symptom/biomarker.
The “instinct” of endocrinologists is to attribute hyperprolactinemia to somatic sources (e.g., adenoma). It is suspected to result from a prolactinoma, which is the most frequent pituitary tumor and is usually treated through dopamine agonist therapy, with cabergoline as the most successful treatment (Gilliam et al., 2006).

In males, the major function of prolactin is *inhibition of the sex drive*. Therefore, when encountering a male patient suffering from hyperprolactinemia, a physician with a psychosomatic orientation should be inclined toward a different path of diagnosis. Following the dynamic perspective presented in this article, the first two questions the physician should ask in the case of a male patient with hyperprolactinemia is (1) why this patient should inhibit his sex drive, and (2) why this will for inhibition cannot be (a) resolved or (b) consciously experienced. On the other hand, a psychotherapist encountering a male patient who complains of a decrease of his libido should ask whether biomarkers such as hyperprolactinemia are evident. Moreover, in order to transcend the soma-psyche dichotomy, the physician or the psychotherapist should not simply seek the source of the problem either in the somatic or the mental realm, but should examine the way in which the behavior of the low-dimensional signal is indicative of the high-dimensional space from which it has originated. Tagging this dimension as “somatic” or “mental” is irrelevant but only the way in which a higher-order dimensional system behaves. The high-dimensional space is probably composed from different dimensions: “somatic,” “social,” “psychological,” etc. However, tagging these dimensions is of no interest but only the dynamics that take place at the high-dimensional space.

These questions address the function of the prolactin as a low-dimensional biomarker of a high-dimensional trajectory and bring us to the realm of psychosomatics. The second task of the physician/therapist is to understand the repetitive pattern of the prolactin appearance over time and by using this pattern to learn about the phase space trajectory from which it emerged. These minimal guidelines are illustrated in the following case. It must be noted, though, that currently there is no therapeutic approach that results
from these guidelines. The development of this approach may be the aim of future studies.

The Clinical Case

P is a 37-year-old man, married and working as a computer scientist. He consulted his family physician with complaints of fatigue and loss of libido. The family physician has more than 20 years of experience and a background in alternative medicine. Following three meetings with P. and a series of blood tests, the physician noticed that the testosterone level was within the norm but identified hyperprolactinemia and suggested pituitary hyperfunction as a primary diagnosis. A computed tomography scan did not reveal signs of microadenoma, although this possibility cannot be dismissed. P was sent to an expert endocrinologist for further examination. The expert is working in a regional university hospital and has over 30 years of experience. The expert’s report is as follows:

This 37 year old man is seen because of fatigue, muscle weakness compared to his usual state of health, all during the past 4 months. His testes were brought down surgically at age 6 and he is azoospermic. He appears well—I did not examine his genitalia. Note one complaint is loss of libido. Lab shows PRL 34-40, LH 11.3, FSH 68 and total testost 2.80 (LLN 2.5), estradiol 11. CT as expected a normal sella. IMP primary hypogonadism, the minimal hyperprolactinemia may be related, I doubt, but cannot exclude a microadenomea but at this point would not request an MRI or start cabergoline. I would repeat his total and free testosterone, FSH LH and PRL and then start Tesoviron Depot 150 mg every two weeks and after 6 weeks raise the dose to 200 mg every 2 weeks, check his total testosterone once at midpoint in one week after the injection and just before the next due injection. I would also check his CBC after 3 months.

It must be noted that P’s level of prolactin is significantly high. However, the expert described the hyperprolactinemia as “minimal” because the symptoms were relatively mild and were attributed to hypogonadism. One can argue with this decision.

At no point in his diagnosis was P asked for the reason he had to inhibit his sexual drive. On the other hand, a psychotherapist
working from a mentalistic perspective would probably have ignored the existence of a biomarker during his diagnosis. Following the expert’s opinion, the general practitioner recommended testosterone therapy. P received testosterone gel in a daily dose of 50 mg but refused to continue the treatment because a side effect of mild testicular pain was evident, probably due to reduction of testosterone production in the testes.

Consulting a therapist working with the previously discussed dynamical suggestions, it was found that for several years P had extreme difficulties in his marriage. These difficulties resulted in a complete cessation of sexual activity. As a religious person highly devoted to certain standards of moral behavior, he refused the possibility of having sex outside his marriage or even masturbation as ways of alleviating his sex drive. Based on this finding, it seemed reasonable that the hyperprolactinemia was a psychosomatic biomarker signaling the acute stress he experienced and his unconscious attempt to inhibit his sex drive through interference with PRL production cycles. Tracing the trajectory of his symptom, it was found that P had experienced an incremental decrease in his libido for a couple of years, a decrease that reached a certain low-level plateau just before he consulted his physician. Therefore, a reasonable hypothesis is that a minor endogenous hypogonadism interacted with a specific and extremely stressful life event. However, it seems that the key to the problem was not purely “somatic” or “mentalistic” but psychosomatic. Measuring the effect of the intervention with P is beyond the scope of this article and actually impossible because he did not continue any kind of treatment. His case is used for illustrative purposes only.

Discussion

As suggested by Oken (2000), “Each ‘disease’ in each person reflects an ideographic process comprised of these complex transactions among internal and external variables that only partly reflects the nomothetic stereotype” (p. 172). Oken’s use of the term ideographic is important because psychology experiences severe theoretical and practical difficulties in measuring ideographic processes (Molennar, 2004). The ideographic process described by Oken is...
comprehensible as the phase space trajectory of the individual, and it can be reconstructed and studied through its lower-dimensional manifestation—the psychosomatic symptom. This suggestion resonates both with classical psychoanalytic ideas and with our modern understanding of dynamical systems.

The “conversion” of a stressful mental event into a somatic symptom was identified by Darwin in the context of animal behavior and by Breuer and Freud in the context of human psychology (Freud & Breuer, 2000). This conversion is the driving force behind psychosomatic symptoms, and it can be metaphorically interpreted as the projection of a mental space trajectory into a low-dimensional biomarker. This perspective transcends the old mind-body dichotomy and urges us to examine psychosomatic symptoms in a new light. Therefore, the article concludes with an invitation for further research and inquiry through this novel metaphor.

References


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