

# **Symptom Perception, Awareness and Interpretation**

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**Abstract**

Self-reported health complaints result from a complex integration of interoceptive (bottom-up) information emerging from peripheral physiological activity with (top-down) perceptual-cognitive and affective processes. Interoceptive signals have to be sensed, perceived, attended to, appreciated and interpreted, put into language and expressed, mostly in a social context. In this chapter, we will discuss the role of perceptual and attentional processes, anticipation, symptom schemata and illness beliefs, emotion and personality, and memory processes. Their role may importantly influence the relationship between indicators of peripheral physiological activity and self-reported health complaints.

## **Interoception and symptom perception**

Although all self-reported health complaints are the result of processes in the brain, a general assumption is that they accurately reflect dysfunctional processes in the peripheral body. The afferent or sensory processes transmitting information from the body to the brain can be called interoception. However, although interoception and symptom perception are related processes, they are not the same.

### **Interoception**

Interoception is poorly defined and several definitions are used. Some equalize it rather narrowly with visceroreception (Dworkin, 2000), whereas others (Craig, 2004) also include proprioception and somatoception (the perception of stimuli interacting with the body surface). Still others conceive of interoception more broadly as a meta-representation of the body's state allowing the brain to distinguish the inner from the outer world (Mosely et al., 2012).

Recent neurobiological research has shown that sensations from within the body, such as hunger, thirst, stomach cramps, fullness, rectal urgency, temperature, itch, muscle ache, dyspnea, pain, etc. are processed in a shared evolutionarily ancient neural pathway that serves to maintain the integrity of the body or, in other words, homeostasis. Several comprehensive reviews (Craig, 2002, 2004) suggest that interoceptive pathways include a sensory branch carrying signals to the lamina I and the medullary nucleus of the solitary tract, which are (in humans) integrated in the parabrachial nucleus to travel via parallel ascending pathways to the ventromedial thalamic nuclei further to the mid/posterior insular cortex, in which a modality-selective representation is produced underlying distinctive sensations.

Via ascending afferent pathways providing a direct thalamo-cortical connection to the anterior cingulate cortex (ACC), interoceptive sensations also become endowed with affective-motivational value (e.g. unpleasantness). The interoceptive image is further re-represented in the right anterior insula, which is also involved in subjective awareness of feelings and emotions. This process of re-representing progresses from the posterior to the anterior parts implies a successively increasing “integration of homeostatic, environmental, hedonic, motivational, social and cognitive activity to produce a ‘global emotional moment’, which represents the sentient self” (Craig, 2009, p. 67). This allows for a bi-directional influence of emotion and interoception (Critchley et al., 2001; Zaki et al., 2012). For example, accurate cardiac perception is associated with more intense emotional experience (Herbert et al., 2010), and state negative affect enhances the activation of the right anterior insula and the dorsal ACC (Mayer et al., 2006). The ACC has further strong interconnections with cortical prefrontal (PFC) regions as well as strong descending projections, particularly to the periaqueductal gray (PAG) and the brainstem, allowing for top-down facilitatory and inhibitory modulation on interoception from higher cortical regions (Mayer et al., 2006). Top-down modulatory processes may go as far as to influence neural activity at the level of the spinal cord (Eippert et al., 2009).

This short neurobiological account shows that there is room for multiple top-down processes to modulate the experience of sensations from the body and different functional aspects of interoception (Garfinkel & Critchley, 2013) have been distinguished. For example, on both neurobiological and functional grounds, a distinction can be made between interoceptive accuracy, which refers to the ability to accurately perceive one’s own internal activity, and interoceptive or body awareness, which refers to the state of being cognizant, mindful of one’s own internal state (Khalsa et al., 2008; 2009; Ceunen, Van Diest & Vlaeyen, 2013). Interestingly, heightened body awareness can be both adaptive and maladaptive for

perceived health (Mehling et al., 2009), whereas evidence shows that elevated symptom reporting may be related to reduced interoceptive accuracy (see further).

### **Symptom perception**

Symptom perception can be defined as the process of becoming aware of bodily dysfunction. Although symptom perception can theoretically be distinguished from symptom reporting or self-reported health complaints, these concepts are often used interchangeably. Perceived internal sensations turn into health complaints when they become endowed with negative affective value and become part of a meaning network associated with potential threat to the integrity of the body. In this respect, bodily symptoms can be conceived of as “homeostatic emotions” (Craig, 2003): they involve a sensory component serving the experience of intensity, location and other qualities of internal sensations, and an affective-motivational component providing the drive for behavioral action to promote survival. Accurate and aware perception of bodily dysfunction may, therefore, be considered the behavioral layer in a hierarchical defense system to protect the integrity of the body: the behavioral action system is engaged when automatic, more local, automatic and reflexive regulatory systems within the body fail. For example, the experience of breathlessness interrupts ongoing activities, rearranges processing priorities and is associated with a compelling drive to gasp, opening the window and/or fleeing from closed places to compensate for inaccurate automatic regulation of blood gases.

The process of becoming aware of bodily dysfunction typically includes putting perceived sensations into words, which are part of, or constitute, meaning networks. The semantics of symptom words are particularly relevant for symptom assessments. Most questionnaires use a variety of symptom words, which are clustered according to physiological systems, such as cardiorespiratory and gastrointestinal symptoms. However,

other relevant underlying aspects in symptom reporting tend to be overlooked and more sophisticated analyses of dimensions underlying somatic symptom reporting are needed (see Jasper et al., 2012). Indeed, items used in assessment instruments may differ according to other aspects as well. Some inquire about a mere sensory aspect (e.g. fast/deep breathing), while other items also probe an implicit appraisal of its unpleasantness (i.e. “breathlessness”). Both aspects can reliably be distinguished (von Leupoldt et al., 2006; Wan et al., 2009), but individuals differ in how they intuitively integrate both components (Petersen et al., 2011). Several other relevant dimensions can be distinguished, such as the extent to which a symptom word refers to a specific location in the body or to a systemic state (compare “tooth ache” with “tiredness”) and the probability of a symptom to be life threatening (compare “running nose” with “blood in stools”), etc. Few studies have looked at the role of psychological processes to select or endorse particular wordings to describe one’s own internal state (but see Van Diest et al., 2005).

Just like any other type of information entering conscious awareness, information from within the body is sensitive to modulation by functional psychological processes. The seminal work of Pennebaker (1982) on the psychology of physical symptoms introduced most processes elaborated on in later, more detailed models. These processes include attention, expectation, memory, attributions and illness beliefs, emotions and personality (Rief & Broadbent, 2007). In the following we will discuss a number of relevant psychological processes and how they influence symptom perception. Although several processes are discussed separately, it should be clear that they are mutually interacting to construct a unified somatic experience for the “sentient self”.

## **Psychological modulation of symptom perception**

## **Perception and attention**

Critical dimensions for symptom perception are intensity and location of interoceptive stimulation. A general rule seems to be that psychological modulation of bodily symptom perception is less pronounced when the interoceptive stimulation is intense and unambiguous in time (on/off) and location. In these conditions, a rather direct relationship between the interoceptive stimulation and symptom reports exists (Stegen et al., 1998; Put et al., 2004; Martin, Rothrock, Leventhal, & Leventhal, 2003). Most often, however, interoceptive information is low to mildly intense and has no clear spatial and/or temporal boundaries, particularly when the physiological dysfunction is systemic. In such conditions, attention seems to play an important role. Attention, a process involving the allocation of processing resources to stimuli, typically amplifies psychophysiological and behavioral responses to attended stimuli, whereas distraction from these stimuli reduces such responses (Bushnell et al., 1999). Attentional focus can result from local, bottom-up stimulus characteristics (e.g. novelty) or from top-down processes, such as anticipation.

Two important questions emerge as regards attention and symptom perception: is attention “directed inwards” influencing the number and intensity of perceived bodily symptoms, and is it influencing the accuracy of symptom perception (defined as the within-subject correspondence between physiological and self-reported changes)? As to the first question, direct manipulations of attention to the body and studies assessing body awareness generally show that attention focused on the body enhances the sensitivity to interoceptive stimulation and increases symptom reports. This has been documented by studies on tactile stimulation (Mirams et al., 2012), respiration (Stegen et al., 2001), pain (Villemure & Bushnell, 2009), itch (Van Laarhoven, et al., 2010), dyspnea (von Leupoldt et al., 2007), and general symptom reporting (Verkuil et al., 2007). Interestingly, not just symptom reports but also objective cough frequency is enhanced by focusing attention on internal sensations (Van

den Bergh et al., 2012). Consistent with such findings, it has been shown that performance on concurrent cognitive tasks is disrupted as a result of attentional resources being consumed by noticing interoceptive stimulation (Stegen et al., 2001; Eccleston & Crombez, 1999).

Conversely, distraction from bodily sensations generally reduces the perceived intensity of interoceptive stimulation and symptom reports (Pennebaker & Lightner, 1980; Coen et al., 2008; Accarino et al., 1997). The role of attention for symptom reports has been captured in the “cue-competition hypothesis” (Pennebaker, 1982), assuming that the amount of interoceptive information reaching awareness is a function of the ratio between the amount of interoceptive and exteroceptive information. Findings that persons living in boring environments tend to report more symptoms than persons living in rich and stimulating environments are in line with this hypothesis.

As to the second question, evidence suggests that an interoceptive attentional focus is not necessarily associated with better interoceptive accuracy, but rather with a tendency to overestimate the intensity of somatic sensations. In a study applying low intensity tactile stimuli, it was shown that a more liberal response criterion was used when attention was directed inwards, leading to more false positives (Mirams et al., 2012). Some groups, such as persons with high trait negative affectivity (NA) who tend to focus attention inwards during a physiological challenge (Stegen et al., 2001), report more symptoms which are less closely related to the induced physiological changes (Van den Bergh et al., 2004; Bogaerts et al., 2005). Also persons with somatoform disorders, who are typically preoccupied with sensations in the body, reported more symptoms as shown by their interoceptive accuracy to be lower (Schaefer et al., 2012) compared to healthy controls.

An as yet unresolved question is whether the emotional significance of an interoceptive sensation interacts with its sensory processing. Substantial evidence shows that the amygdala serves as a “neurological hub” swiftly assessing the emotional value of

exteroceptive sensory events and subsequently enhancing perceptual processing in early sensory pathways through connections with sensory areas in more distant cortical regions (Pourtois et al., 2013). Claims are made that similar attentional prioritization and subsequent enhancement of processing occurs for potentially noxious stimuli in the “peripersonal space” (the body and the space surrounding it) (Legrain et al., 2011), but it is not known whether similar mechanisms operate when processing stimuli originating from within the body.

### **Anticipation, symptom schemata and illness beliefs**

A wealth of evidence on placebo and nocebo documents the dramatic effects of expectations on symptom perception and reporting. Anticipation can be induced either by classical conditioning, verbal instructions and all kinds of contextual factors. It should be noted that conditioned placebo and nocebo effects do not necessarily depend on conscious expectation (Benedetti, 2013). Effects can impact the intensity, but also determine the sheer presence/absence of symptoms. Particularly pain analgesia has been thoroughly investigated and a so-called descending pain modulatory network has been described, involving prefrontal (DLPFC), limbic (ACC, amygdala), midbrain (PAG) and even spinal cord areas. Knowledge about the mechanisms involved in placebo and nocebo phenomena related to other sensations than pain is more sketchy, but sufficient to document the pervasive effects of anticipation on neurotransmitter function, including the endogenous opioid system, and on autonomic, endocrine, immune and motor functions (Finniss et al., 2010; Enck et al., 2013). Such findings show that symptom perception involves a bi-directional process without a marked boundary between central and peripheral processes. Bottom-up processes relay information of peripheral physiological activity to the brain, but cognitive processes (critically requiring the involvement of prefrontal areas, such as the dorsolateral PFC) in turn substantially influence activity in other central neurobiological and in peripheral physiological systems. These

specific systems may differ according to the type of placebo or nocebo response (Benedetti, 2013).

Symptom schemata typically result from repeated symptom episodes. It is assumed that the person's history with symptoms episodes is recorded in memory and represented in the form of schemata, which capture the commonalities among different symptom episodes. Because some people had more episodes than others, while repeated symptom episodes can result from the same or from different health problems, symptom schemata can differ in complexity and coherence (Petersen et al., 2011). Symptom schemata act like perceptual categories: When activated or primed, a readiness to perceive an interoceptive stimulus configuration in a particular way is facilitated. The benefit is that less information has to be checked and controlled for a symptom experience to emerge, speeding up the perceptual process, but the ease of perception may come at a cost showing up as an elevated probability towards biased perception. For example, the slightest change in respiratory effort may be noticed by an "experienced" asthma patient, whereas the same change may not reach awareness of the not-yet diagnosed or novice asthma patient. Conversely, an asthma patient who is concerned about potential attacks may easily misperceive respiratory distress caused by stress-induced hyperventilation as signs of an impending attack. Even simple magnitude judgments of respiratory resistance and related affective and behavioral responses are affected by primed perceptual categories of high or low respiratory effort (Petersen et al., in press).

Just like the perception of a chair is automatic, categorical, meaningful and constructive, so is symptom perception the result of similar basic and automatic perceptual-cognitive processes. These involve a process of mapping sensory evidence on perceptual hypotheses, representing some kind of implicit reasoning. This was captured by Brown's model when postulating a primary attentional system, PAS (Brown, 2004). In line with research on perception, it hypothesizes that sensory stimuli are activating perceptual

hypotheses in a feed-forward process and that actual information is gradually mapped onto them, giving rise to awareness of only this perceptual hypothesis that obtains the highest level of evidence (e.g. becomes a percept). The higher the activation state of symptom schemata, the less evidence from peripheral stimulation is needed in order to result in percepts.

Ultimately, percepts may emerge without peripheral input at all, such as in placebo or nocebo symptoms, which then can be characterized as “somatovisceral illusions”. For example, when participants were given an air mixture to breathe for a number of times, consisting of a harmless odor and CO<sub>2</sub>-enriched air causing bodily symptoms, elevated symptom reports emerged upon subsequently breathing a mixture of the harmless odor only. These nocebo symptoms were similar to the symptoms originally induced by CO<sub>2</sub>-inhalation and apparently resulted from automatically activated symptom schemata biasing the subject’s perception of his/her somatic state (Van den Bergh et al., 1997; 1998).

However, once symptoms are felt, people may contemplate upon them, link them to an illness label, infer potential causes, anticipate about their consequences and worry and ruminate about them. In other words, they develop an illness theory, elaborate on it and behave accordingly. The interpretation of palpitations as a symptom of an acute heart condition will prompt completely different behaviors compared to believing that they result from drinking too much coffee. This secondary process may, however, prime perceptual categories and modify the amount of evidence needed for a somatic percept to emerge, with potentially beneficial or disadvantageous outcomes. In extreme cases, chronic activation of perceptual hypotheses, possibly as a result of catastrophizing, worrying and ruminating, may contribute to somatovisceral illusions, underlying medically unexplained symptoms (see further).

### **Emotion and personality**

A robust association between symptom reporting and trait Negative Affectivity (NA) is consistently found ( $r = .40$  to  $.50$ ). Trait NA is a broad and stable disposition to appraise situations as more threatening and to experience negative mood states and emotions (Watson & Clark, 1984). It can be described as an over-reactive evaluative system combined with poor or deficient emotion regulation when processing emotional stimuli (Yiend, 2010). It appears to have a genetic basis, is associated with distinct brain circuit function and neurotransmitter activity (Hariri, 2009) and is considered a vulnerability factor for emotional disorders, such as anxiety and depression (Lonigan & Vasey, 2009).

The association with symptom reporting reflects the effect of NA upon symptom reporting rather than vice versa (Watson & Pennebaker, 1989). The association appears in non-consulting healthy persons (Van Diest et al., 2005), in primary care patients where medically unexplained symptoms co-occur with elevated anxiety and depression levels (Kroenke, 2003), and in patients with functional syndromes showing elevated psychiatric comorbidity (Wessely et al., 1999). In addition, also in known diseases a substantial correlation between symptom-reports and NA exists (Janssens et al., 2009), reflecting a tendency to over-report symptoms in high NA persons. Closer inspection suggests that the depressive component of NA is mainly associated with “over-reporting” of past symptoms, whereas the anxiety component is more related to excessive reporting of concurrent symptoms (Suls & Howren, 2012). Also, not all symptoms are associated with NA. Van Diest et al. (2005) investigated the relationship between 73 individual symptoms and NA in a large healthy sample and found a wide variability ( $r$ 's  $.0$  to  $.45$ ), which could be explained by two factors, namely severity (whether a symptom was potentially life-threatening) and “somatic versus psychological” (whether a symptom was vague and possibly associated with anxiety; e.g. compare “stuffed nose” versus “loosing contact with reality”).

Interestingly, also state negative affect impacts symptom perception. When presenting unpleasant emotional cues during experimentally induced bodily sensations, like pain (de Wied & Verbaten, 2001; Meagher et al., 2001), dyspnea (Von Leupoldt, Mertz, Kegat, Burmester & Dahme, 2006) or esophageal stimulation (Philips et al., 2003), symptom reports are more elevated. Even very short presentations of unpleasant pictures can result in increased symptom reports, particularly in persons with high habitual symptom levels, and/or high negative affect (Bogaerts et al., 2005; 2008; 2010; Constantinou et al., 2013) and in patients with functional syndromes (Montoya et al., 2005).

Several hypotheses have been advanced to understand this relationship. One idea is that persons with NA and/or during negative affective states have higher levels of sympathetic activity, which would translate into elevated self-reported symptoms. However, several extensive laboratory and ambulatory studies have not been able to document significant differences in a wide variety of peripheral physiological stress or arousal indicators (Houtveen & Van Doornen, 2007). Recent studies are inspired by the hypothesis that inflammatory load and immune-related mechanisms are involved and that the relationship between negative emotional states and elevated symptom reports results from stress-related sensitization of the brain-immune communication, but only scanty evidence exists so far in support of this idea (Lacourt et al., 2013; Lacourt, 2013).

Another idea emphasizes attentional mechanisms leading to lower perceptual thresholds for (normal) physiological arousal responses to enter awareness. A set of studies by Gendolla et al. (2005) suggested that self-focused attention in conjunction with negative mood is critical for elevated symptom reports to occur. The interpretation advanced for these findings is that attentional focus on one's own negative mood would activate symptom schemata biasing reports of one's somatic state. However, self-focused attention seems not critical: simply viewing pictures with negative valence also elicits elevated symptom reports in high

NA persons (Bogaerts et al., 2010; Constantinou et al., 2013). Apparently, simply processing negative cues, either in the internal or the external world, triggers symptom schemata and makes people prone to biased symptom perception, particularly in persons with high NA.

## **Memory**

Symptom assessments during diagnosis and treatment evaluations importantly rely on memory information. “How did you feel” is an often heard question in the doctor’s office, but also screening and assessment instruments measuring symptoms in general or related to a particular disease typically use retrospective frequency estimates over a long, often unspecified time window. Questionnaires typically require the respondent to rate a set of symptoms along intuitive standards such as seldom, frequent, almost daily, etc. (Zijlema et al., 2013). In such conditions, symptom reports are mainly based on memory of somatic episodes. The importance of symptom assessment being memory-based is often overlooked despite the fact that memory is not just storing an experience, but is actively reconstructing it. Only when actual symptoms are assessed (how do you feel now?), either during doctor visits or with (often disease-specific) questionnaires, momentary information is collected. Evidence suggests that even then, there is no clear one-to-one relationship between physiological responses and self-reported complaints (Walentinowicz et al., 2013). Increasingly, time- or event-related multiple momentary assessment is used to assess symptoms in daily life (Shiffman et al., 2008), allowing for interesting comparisons between concurrent and retrospective ratings spanning the same symptom episodes and assessment period.

The majority of findings suggest a consistent overestimation of past symptoms as measured with global retrospective symptom ratings (Stone et al., 2004; Giske et al., 2010; Linton & Melin 1982; Broderick et al. 2008). However, several factors relating to characteristics of the symptom episodes, the duration of the recall period, the context during

recall and individual differences seem to moderate this overestimation, often resulting in a complex picture. For example, retrospective overestimation of pain was greater with lower initial pain, while high initial pain was associated with underestimation (Feine et al., 1998). Also the variability of symptoms is an important variable (Stone et al., 2005): greater momentary variability was associated with greater discrepancy between momentary and retrospective ratings and higher retrospective ratings overall. Overestimation typically also increases with longer recall periods (Broderick et al., 2008), although in a study recording physical symptoms in daily life in students, a gradual increase in overestimation of experienced symptoms with longer time frames was observed only among high, but not low habitual symptom reporters (Houtveen & Oei, 2007). Furthermore, actual state during recall plays a role: lower current pain intensity at the moment of recall was associated with underestimation of recalled pain, whereas higher current pain was associated with overestimation (Smith and Safer, 1993; Lefebvre & Keefe, 2002; Gendreau et al., 2003),

A well-investigated memory bias is the so-called peak-end effect in retrospective evaluations. This refers to the observation that not all constituent elements of an experience are equally important when representing it in memory: the intensity of an experience at both the peak and the end receive relatively more weight as these episodes convey the most relevant information. The effect in symptom studies shows up as lower retrospective discomfort ratings when for example pain or dyspnea ended gradually at a lower discomfort level rather than abruptly at the peak, despite an equal level of peak discomfort and a longer duration of total discomfort (Kahneman et al., 1993; Bogaerts et al., 2012). Interestingly, patients with medically unexplained symptoms do not show this effect, suggesting substantial differences in the way they represent symptom episodes in memory (Bogaerts et al., 2012).

In general, it can be concluded that research on memory for symptoms needs more attention in view of its importance for diagnostic and therapeutic assessment of self-reported health.

### **Medically unexplained symptoms: a symptom perception pathology?**

For a substantial part of the patients consulting medical doctors, no physiological dysfunction can be related to their health complaints. Hence, the latter are often called medically unexplained symptoms (MUS). The share of MUS in primary care consultations is estimated to range from 20% up to 50%, while prevalence rates in secondary care are even higher (Nimnuan et al., 2001). Typical symptoms are fatigue, weakness, headache, muscle aches, nausea and other gastrointestinal complaints, joint pain, palpitations, chest pain, dyspnea, dizziness, etc. (Barsky & Borus, 1999). In a clinical context, the symptoms often appear as functional syndromes, such as chronic fatigue, fibromyalgia, irritable bowel disease, multiple chemical sensitivity, etc. Despite specific diagnostic criteria for such syndromes, overlap and comorbidity between the different categories is large, leading to a debate between “lumpers” (who consider the different syndromes as basically identical) and “splitters” (who emphasize the different specificities of the syndromes) (Wessely et al., 1999; Lacourt et al., 2013; White, 2010).

MUS challenge the traditional disease model, which assumes a direct relationship between a physiological dysfunction and self-reported complaints. The absence of such a relationship is the source of both frustrated medical doctors and patients, it feeds somatization processes in the patient and leads to overuse of health care resources and puts a substantial burden on the health care system (Barsky et al., 2005).

Several strategies are followed in search of an explanation. One strategy is motivated by the assumption that as yet unknown (stress-related?) dysfunction in peripheral physiological systems and/or in their interaction with the brain, such as the immune-to-brain communication, is critical and that symptom reports are a true reflection of such dysfunctions. Several reviews revealed evidence in support of this strategy, but the critical involvement of specific physiological systems is overall not considered convincing enough because of the lack of evidence documenting the specificity, consistency and/or causal direction of the findings (Rief & Barsky, 2005). Other strategies rely on the idea that the critical mechanisms are centrally mediated distortions in the perception of one's bodily state. While substantial evidence is consistently showing distortions in perceptual-cognitive mechanisms of symptom perception, cause-effect relationships remain often unclear and the critical hypothesized mechanisms remain untested (Rief & Broadbent, 2007).

Both strategies are neither mutually exclusive nor theoretically incompatible. The important advances in placebo research of the recent decade illustrate how deeply psychological variables, such as learning, anticipation and social context are intertwined with central and peripheral physiological mechanisms (Benedetti, 2013). Such advances are likely the prelude of a paradigm shift in which the opposition between psychological and physiological processes in the study of MUS is considered elusive and will be replaced by research and theorizing on MUS as a nocebo phenomenon, emerging from an intricately intertwined mind-body system.

## **Conclusion**

Several processes modulate the relationship between peripheral physiologic dysfunction and self-reported symptoms. In extreme cases there is no link at all. This is

insufficiently recognized in a biomedical disease model, which assumes a direct relationship between peripheral dysfunction and symptom reports. A more advanced symptom model (Kroenke & Harris, 2001), in which both peripheral and central/psychological processes are included in both a diagnostic and therapeutic step, is needed to further improve health care.

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#### Cross references

14017. Doctor-Patient interaction In The West: Psychosocial Aspects

14059. Explanatory style and health

14069. Chronic pain: models and treatment approaches

14105. Illness Behavior and Care Seeking

14138. Health Risk Perception

14145. Interoception