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A Dual-System Model of Social Anxiety Disorder: The Interplay of the Social-Rank and Affiliation Biopsychosocial Systems

Eva Gilboa-Schechtman
Bar-Ilan University, Ramat Gan, Israel
ORCID: https://orcid.org/0000-0002-2831-5835, e-mail: evagilboa@gmai.com

Social Anxiety Disorder (SAD) is a highly prevalent and debilitating condition. Although effective treatments exist, their success is limited. This narrative review seeks to advance a comprehensive understanding of the types of social threat affecting individuals with SAD from the perspective of two basic biobehavioral systems: affiliation and social-rank. We argue that SAD is associated with vulnerability to events signaling loss of affiliation (exclusion) and of social-rank (defeat). Specifically, we suggest that SAD is characterized by (a) hyper-reactivity to exclusion and defeat; (b) propensity to respond to exclusion by deploying distancing and withdrawal strategies; (c) propensity to respond to defeat by deploying conflict-reducing subordination strategies; and (d) enhanced linkage of the two systems. This dual-system account may help integrate clinically significant information about SAD and offer recommendations regarding novel theory-based directions for treatments.

Keywords: dominance, belongingness, social anxiety, exclusion, defeat, depression, social stress.

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Social dangers constitute the main perils of modern life. Acceptance is never a given, prestige is never secure. Every social encounter involves a risk of rejection and a possibility

of humiliation. Whereas a certain level of anxiety in the face of exclusion and defeat is adaptive, intense anxiety can significantly impair coping with such events. *Social Anxiety Disorder* (SAD) is a condition involving marked anxiety about social or performance situations in which an individual is exposed to possible scrutiny by others with fear of public speaking being the most common concern [70]. Individuals with SAD fear acting in ways that will be humiliating, embarrassing, or will lead to rejection [6].

The Clinical Profile of Social Anxiety Disorder (SAD)

Life-time prevalence of SAD is estimated around 12% and is associated with severe psychological, interpersonal, and professional consequences resulting in quality of life impairment [3; 78]. Social anxiety (SA) ranges in severity, but even below-diagnostic levels are associated with reduced well-being [30], and lower quality of intimacy in peer, friend, and romantic relationships [106].

The clinical profile of SAD is marked by an early onset with almost 75% of the cases beginning by mid-adolescence [43]. Women are more likely to have SAD [10]. SAD is comorbid with major depressive disorder (MDD, [67]) and with substance abuse disorders [16]. Significantly, the onset of SAD precedes the onsets of both MDD and substance abuse disorders in as many as 80% of comorbid cases [83]. It is therefore unsurprising that individuals with SAD are likely to attempt suicide [47].

Multiple intervention types have shown promise in the treatment of SAD including (but not limited to) Cognitive Behavioral Therapy (CBT), acceptance and commitment therapy, interpersonal psychotherapy, and social skills training [2]. Multiple outcome studies examined the effectiveness of individual as well as group formats of CBT, documenting moderate to large effect sizes [65]. However, despite the effectiveness of CBT, many patients either do not stay in therapy (attrition rates of 5-30% [1; 51]), fail to respond to CBT (40-57% do not exhibit clinically significant symptom reduction even after completing the full course, [20]) or remain considerably symptomatic at the end of treatment (only about 1 in 5 individuals reached symptom-free functioning in a large scale community study of group CBT [9]). Moreover, even following the completion of a full course of CBT, many patients continue to report reduced well-being and satisfaction with the quality of their interpersonal relationships [20; 27].

Theoretical Accounts of SAD: Process and System Models

Cognitive models emphasize the ways in which various *processes* combine to maintain SAD. According to these models, high-SA individuals are driven by a desire to make a good impression, yet doubt their ability to achieve this goal [18; 45; 48; 66; 72]. SAD is also postulated to be associated with enhanced self-focus to internal sensations, resulting in painful self-awareness. Moreover, it is believed that SAD is perpetuated by negative cognitions and images of the self. Rapee and Heimberg also postulate that biased attention to threatening interpersonal cues contributes to the maintenance of SAD [72]. Hofmann further argues that individuals with SAD are characterized by enhanced discrepancies between actual and ideal self, and that these discrepancies may underlie their fears about their ability to maintain a desired image in the eyes of others [48]. Moscovitch stresses the importance of identifying specific self-attributes that underlie

individuals' negative self-concept, rather than focusing on the generally negative contents of their self-beliefs [66]. These theories highlight the role of multiple cognitive processes – such as attention, interpretation, and evaluation – in the maintenance of SAD.

Another set of models examines SAD from the perspective of basic biobehavioral systems, highlighting the possible ways in which such systems may malfunction to produce the clinical profile of SAD. Two such prominent systems are the affiliation (aka belongingness) and social-rank (aka power, dominance) systems. Some models postulate that SAD is only associated with one of the two, in particular, the social-rank system [46; 105]. Hermans and van Honk propose that the adaptive function of SA is rooted in ancient communicative systems that regulate social order and inhibit inappropriate and antisocial behaviors [46]. Weeks and colleagues elaborate this line of thinking, suggesting that the tendency to avoid evaluations and to exhibit submissive behaviors may be helpful in coping with social threats by dodging conflicts with powerful others [105].

Other models argue that SA needs to be considered from the perspectives of both systems [36; 99]. Trower and Gilbert propose that SA individuals tend to over-utilize the social-rank and under-utilize the affiliation systems [99]. They further argue that SA individuals are attuned to cues of dominance often at the expense of signals of affiliation. Further elaborating the dual-system account, Gilboa-Schechtman and colleagues emphasize the combined role of the social-rank and affiliation systems in the etiology and maintenance of SAD [4; 34; 35; 42]. They propose that while social cautiousness may be advantageous in unstable hierarchies, it may backfire in moderately benevolent and cohesive social groups.

Purpose and Structure of the Review

Despite the major advantages achieved in the understanding of SAD under the theoretical guidance of both the process and the system-models, gaps in existing conceptualization and knowledge loom large. *First*, a robust literature documents the deficits in affiliative relationships associated with SAD [5]. Yet, many state-of-the-art approaches, such as CBT, do not examine the outcome of treatment in terms of reduction of these deficits. The restrictive focus on social avoidance and submissiveness features of SAD in CBT may contribute to the partial success of these treatments. The importance of addressing multiple interpersonal deficits in SAD is underscored by studies highlighting the partial independence of affiliative and social-rank biobehavioral systems [17; 44; 61]. *Second*, existing perspectives do not offer an integrative framework for the broad clinical picture of SAD – including developmental trajectory, gender differences, and patterns of comorbidity. *Third*, we advance a view of SAD as based on the dysregulation of two specific systems, rather than on a set of symptoms. Such a vision is consistent with recent calls to conceptualize psychological disorders in terms of basic neuropsychological mechanisms (RDoC, [54]). *Finally*, conceptualizing SAD through the lenses of the two biobehavioral systems highlights a functionalist account of SAD, and has the potential to synthesize diverse levels of analyses: from subjective self-repot, to cognitive, endocrine, and neural indices. Looking at both the whole and the parts (i.e., systems and processes) can be a powerful method of advancing knowledge. A system-based account with links to specific processes can integrate the currently expansive, yet disparate, literature on SAD.

This review highlights the organizing properties of biobehavioral social systems as a set of mechanisms that have a common functional theme and act as a coordinated set of neural, hormonal, cognitive, and behavioral processes [31; 80; 81]. This overarching structure is used to synthesize and integrate a large number of *process*-based findings into a more cohesive whole. The current review also highlights the potentially impaired *linkage* between the two systems [36].

We first present data on patterns of reactions of high- and low-SA individuals to challenges in the affiliation (exclusion) and social-rank (defeat) domains. We then discuss the possibility of enhanced coupling of the systems in SAD. Next, we explore the ways in which this account helps integrate clinically significant information about SAD. We conclude by reviewing established and novel theory-based directions for treatments of SAD, and sketch directions for future research.

SAD and the Affiliation System: Dealing with Exclusion

Over the course of evolution, conspecifics depended on each other, with those able to garner social support increasing their chances of survival. Such natural selection pressures led to the evolution of a biobehavioral system of affiliation, which continuously monitors for inclusionary status, and uses this information to guide behavior [12; 86]. Affiliation behavioral system emerges early in the developmental sequence, operates automatically and fluently, is attuned to specific social cues such as touch, gaze, and vocalization, and involves specific endocrine pathways [31; 37; 38; 97]. The brain circuitry dedicated to the affiliation biobehavioral system integrates sub-cortical survival-related networks with insula-cingulate and frontotempoparietal networks [26]. Social exclusion may be elicited by discrete cues (e.g., looking away) or by complex events, such as social exclusion [109].

Social exclusion has been found to affect behavioral, physiological, motivational, endocrine, and neural responses [102]. For example, excluded individuals are more sensitive to emotional tone and more accurate in distinguishing between real and posed smiles [14; 69]. Moreover, exclusion appears to be associated with an enhanced activation of the noradrenergic component of the sympathetic nervous system and with greater activity in dACC and anterior insula [26; 110]. Importantly, individuals differ in their regulation of responses to exclusion: whereas some attempt to initiate or enhance pro-social behaviors, others withdraw from interpersonal contact [93]. These regulatory differences are related to dispositional traits of the excluded individuals (targets), the relationship between excluders and target (in-group, out-group), and perceived expectancies of relationship repair and/or opportunities for reaffiliation [19; 89; 109].

Interpersonal rejection and social exclusion are common occurrences in the daily lives of high-SA individuals [32]. Yet, high-SAs are found to exhibit a more intense, less self-regulated, and more protracted response to exclusion than do low-SA individuals [36]. Whereas a common response to exclusion in non-SAs includes an enhanced attempt to reconnect with others, high-SA individuals appear to “down-regulate” the system: they exhibit a drop in progesterone [64], selectively attend to threat rather than to affiliation cues [94], and do not enhance the positive evaluation of new potential interaction partners [62]. In sum, the functioning of subjective, cognitive, and hormonal components suggest

that high-SA individuals tend to withdraw from affiliative opportunities following exclusion events.

SAD and the Social-Rank System: Dealing with Defeat

Group living confers evolutionary advantages, but the primary cost of such an arrangement is the need to compete with conspecifics for important resources [7; 57; 100]. The social-rank biobehavioral system monitors the relative social standing of conspecifics, and uses this information to guide behavior [87]. It also emerges early in the developmental sequence, operates automatically and fluently, is attuned to nonverbal signals, and involves specific endocrine pathways [38]. Data from human and animal studies suggest that specific brain circuits specialize in processing social-rank related information [8]. For humans, losing a competition, being publicly criticized and failing to achieve the expected degree of social influence is experienced as social defeat [95; 111].

Social defeat is among the most stressful events for humans and animals alike [56], affecting response layers from subjective to cognitive, expressive, endocrine, and neural [24]. Specifically, loss of social-rank is associated with increases in feelings of shame, sensitivity to cues of dominance, blood pressure, cortisol levels, and inflammatory responses [56]. Defeat may elicit distinct reactions ranging from submission, subordination and ingratiation [63; 108] to expressions of dominance and aggression [112].

A robust body of literature suggests that SAD is associated with submissive response to dominance cues [33]. For example, Maner and colleagues found that high-SA, but not low-SA, men exhibited a drop in testosterone following defeat [63]. Similarly, high-SA individuals demonstrated an increase in pitch and a decrease in postural expansion when competing over female attention [103]. High-SA individuals also exhibited more signals of low-dominance during social tasks, such as rigidness, fidgeting and gaze avoidance [104]. Combined, evidence encompassing subjective, expressive, and hormonal data suggests that SA is associated with a down-regulation of social-rank system, although this relationship is stronger in men than in women.

Defeat-Exclusion linkage in SAD

In everyday life exclusion and defeat may co-occur [109]. Targets of exclusion may feel not only disliked but also demoted and disrespected [76; 107]. It appears that high-SA individuals are more inclined than low-SA individuals to interpret exclusion as significant threat to their relational value and self-esteem [41]. These interpretations may, in turn, trigger a pattern of cognitions and behaviors which are akin to social defeat [73].

When exclusion connotes defeat, low-SAs often attempt to reinstate their social value via aggression [23], conspicuous consumption [59], or pursuit of risky financial opportunities [25]. High-SA individuals, however, appear to respond to exclusion by a mixture of social withdrawal and submission, such as decrease in vocal confidence [33; 62] and submissive interpersonal tactics [77]. In a daily diary study, perception of low-affiliative behaviors in a communication partner were linked to submissive behaviors more strongly in individuals diagnosed with SAD than in non-clinical controls [79]. Combined, these data suggest that SA is associated with a tendency to interpret exclusion as a threat to both affiliation and social standing, and to forgo strategies geared to bolster them. Figure

graphically depicts the proposed differences in the reactions of high- and low-SA individuals to two common social challenges of defeat and exclusion.

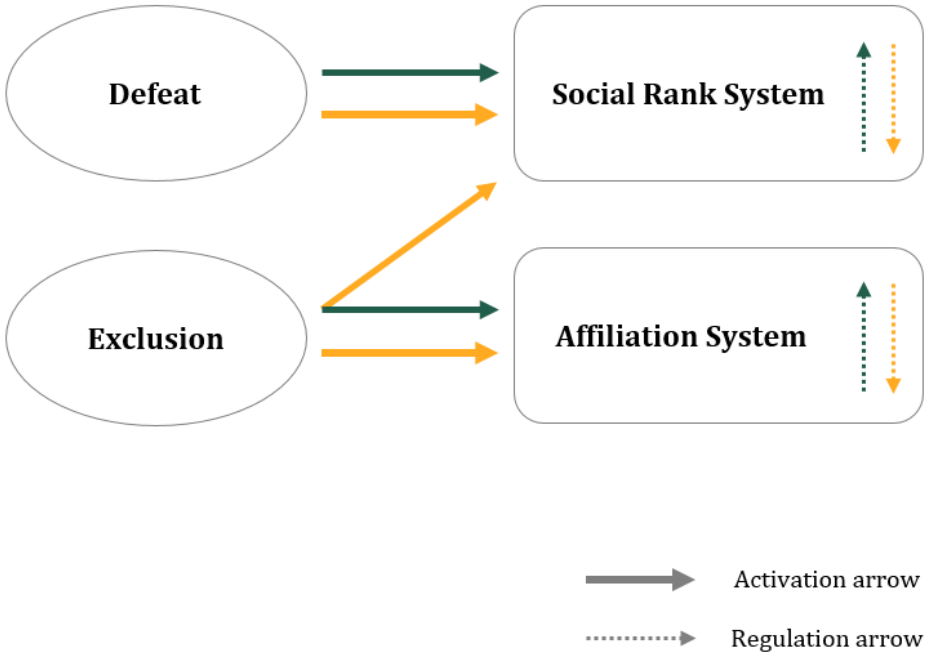

Figure. A Dual-System Model of SAD

Clinical Profile of SAD from the Perspective of the Dual-System Model

Given its early onset, SAD has been called an "adolescent disorder" [43]. Adolescence is a period of neural plasticity, of enhanced emphasis on formation of social bonds and establishment of social status [22]. Accordingly, adolescents are especially vulnerable to both exclusion and defeat, and exhibit enhanced reactivity on affective, neural, and hormonal measures [40; 50; 84; 92]. The increased onset of SAD during adolescence may be attributable to the heightened functioning of both the affiliation and social-rank biobehavioral systems during this period. Indeed, prevention and intervention efforts may be especially targeted to this sensitive period.

Gender affects the severity, expression, developmental trajectory, and interpersonal costs incurred by individuals with SAD [21]. Increase in the onset of SAD during adolescence is particularly pronounced for girls, with early sexual maturation presenting a particular risk factor for this gender [15]. Men are more likely to incur the interpersonal cost of social defeat leading to less frequent opportunities to establish intimate bonds whereas women enlist social bonds to form exclusionary alliances [13]. Specifically, men with SAD appear to entail greater costs to intimate relationship than do women, possibly due to their enhanced submissive strategies in response to social setbacks [113]. Consistent with our dual-system account, these findings paint a gender-specific pattern of vulnerabilities for affiliation and social-rank threats in SAD.

The high comorbidity between SAD and MDD is not surprising given the sensitivity to exclusion and defeat common to both conditions. Indeed, enhanced sensitivity to social rejection is at the core of several models of depression [68; 88]. Moreover, maladaptive

tendency to withdraw in response to social rejection by strangers is also common to both conditions [52; 55]. Some patterns of reaction to exclusion, however, appear to be disorder-specific: depressed individuals seek closeness and reassurance from their significant others whereas high-SA individuals appear to ward off closeness by decreasing self-disclosure [106].

SAD is also highly comorbid with substance abuse disorders, especially alcohol [16]. High-SA individuals appear to use alcohol to self-medicate, allegedly seeking to ease the anxiety in anticipation of, and during, social interactions. Recent research linked social exclusion to drinking [11; 71], and to disinhibited behavior following drinking [90]. In summary, SAD individuals' alcohol consumption may be used not only to attenuate the cognitive and motivational aspects of anticipatory anxiety, but also the pain of social setbacks.

State-of-the-Art Treatments and Promising Treatment Targets

CBT is the most well-research treatment for SAD. A typical course of therapy begins with psychoeducation regarding the condition, and includes a rationale for the proposed intervention. Next, the majority of sessions are devoted to a combination of exposure ("behavioral experiments") and cognitive restructuring. At the onset of this sequence of sessions, a personalized plan of exposures is constructed collaboratively by the client and the therapist. The importance of reduction of self-focused attention and decrease in safety-seeking behaviors is emphasized. Exposure in SAD constitutes a deliberate and systematic series of actions geared to approach socially threatening situations such as social gathering or job interviews. Exposure experiences have a clear set of goals in which the person's expectations are put to test. At the end of exposure "data" collected during the experience are processed, and anxiety-related and negative self-relevant beliefs are challenged. Typically, the emphasis in exposure is to increase the patient's presence and assertiveness in social interactions. CBT thus focuses on counteracting the down-regulation of the social-rank system. Treatment is typically terminated once the pre-determined sequence of exposures has been completed, and concludes with an assessment of the progress made.

Although CBT has shown consistent effectiveness in alleviating SAD severity, many patients still fail to respond or exhibit only partial recovery. Such failures may be due, at least in part, to a rather partial view of vulnerability in SAD, highlighting deficits associated with the functioning of the social-rank system, and focusing almost exclusively on cognitive and expressive components. According to the presented conceptualization, simultaneously targeting *multiple* components of response to social challenges is key to enhancing the efficacy of SAD treatments.

Some novel interventions propose to specifically target cognitive, endocrine, and neural components of reactions to social threats and setbacks. Cognitive bias modification programs (CBMs) seek to train individuals to attend affiliative stimuli to or interpret ambiguous social events in a benign manner. For example, interpretation training reduced SA-severity possibly by training individuals to re-appraise exclusion events, and attentional procedure were used to implicitly train individuals to re-focus on affiliative stimuli [58; 96]. Attentional CBT were found to attenuate anxious response to subsequent stressor, as well as to enhance positive affect [82].

Embodiment-based interventions target the expressive component by modifying facial and bodily expressions related to the production of affiliative and dominant non-verbal signals [38]. In non-clinical populations such interventions lead to congruent changes in affect and cognitive processing. For example, adopting powerful postures and lowering one's pitch reduced interpersonal fearfulness [53; 91].

Based on the compromised endocrine response to exclusion, translational pharmacological methods have begun to enhance existing therapeutic procedures via administration of pro-affiliatory hormones such as oxytocin [29]. In addition, based on the findings of reduced testosterone levels in SAD [39] and the fact that testosterone administration can shift social avoidance to social approach [98], suggestions are raised for the inclusion of testosterone administration as an enhancer to boost the effects of exposure therapy in SAD [28].

Excitatory brain stimulation to enhance control over automatic social avoidance has been proposed [101]. Indeed, an anodal transcranial direct stimulation over an area which is postulated to be responsible for the regulation of social pain (rVLPFC) reduced distress following exclusion [74]. The same procedure was also found to reduce the relationship between social exclusion and aggression [75]. Such stimulation techniques may prove helpful in a range of clinical conditions involving hypersensitivity to exclusion.

The need for personalized interventions has been emphasized by authors reviewing state-of-the-art treatments for SAD [49]. Patient-specific conceptualization of regulatory repertoire in the face of social threats may prove valuable to clinicians in designing focused, individually-tailored intervention. The understanding of the range and diversity of this repertoire may indicate foci where treatment effectiveness can be enhanced [66].

Future Directions

The present model provides but a first step in the creation of a comprehensive account of SAD in which system-level disturbances are linked to specific processes. Several directions for future research are apparent. First, data regarding individual differences in response to exclusion and defeat are only beginning to accumulate. Second, the causal status of responses to exclusion and defeat in the etiology and maintenance of SAD is yet unclear (see, however, [60]). Third, a more extensive effort needs to be dedicated to understand the impact of favorable social events (e.g., inclusion, social ascent) on SAD [36]. Finally, more research is needed to examine the *flexibility* of responses to exclusion and defeat, namely, the ability to select an appropriate, context-specific, regulatory response strategy. Clearly, such flexibility is likely to determine adaptive functioning [85].

Concluding Remarks

We argued that individuals with SAD are characterized by a heightened sensitivity to social challenges, by a propensity to respond to these challenges by down-regulating the triggering system, and by an enhanced coupling between the affiliative and the social-rank systems. Exploring the mechanisms used to maintain a stable, yet flexible balance between the need to belong and the need to matter can help understand and treat SAD.

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Двухсистемная модель социального тревожного расстройства: взаимодействие факторов биосоциальных систем социального положения и чувства принадлежности

Ива Гильбоа-Шехтман
Университет Бар-Илан, Рамат-Ган, Израиль
ORCID: https://orcid.org/0000-0002-2831-5835; e-mail: evagilboa@gmai.com

Социальное тревожное расстройство (Social Anxiety Disorder, СТР) – широко распространенное состояние, нарушающее функционирование индивида. Хотя существуют разные методы терапии СТР, их эффективность ограничена. Данный аналитический обзор направлен на углубление всестороннего понимания типов социальных угроз, влияющих на людей с социальным тревожным расстройством, с точки зрения двух основных биоповеденческих систем – принадлежности и социального положения. Авторы утверждают, что социальное тревожное расстройство связано с восприимчивостью к событиям, свидетельствующим о потере принадлежности (отчуждение) и социального положения (социальное поражение). В частности, предполагается, что социальное тревожное расстройство характеризуется: (а) гиперреактивностью к отчуждению и социальному поражению; (б) склонностью реагировать на отчуждение путем развертывания стратегий дистанцирования и ухода; (в) использованием стратегий подчинения, снижающих конфронтацию в ситуациях социального поражения и (г) усилением связи двух систем. Приведенная выше двухсистемная модель может помочь объединить клинически значимую информацию о социальном тревожном расстройстве может стать основой для рекомендаций относительно новых направлений терапии.

Ключевые слова: доминирование, принадлежность, социальная тревожность, отчуждение, поражение, депрессия, социальный стресс.

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Information about the author

Eva Gilboa-Schechtman, PhD, Professor, Director of Clinical Training, Head of Emotional Processing Laboratory, Department of Psychology and Gonda Multidisciplinary Brain Center, Bar-Ilan University, Ramat Gan, Israel, ORCID: https://orcid.org/0000-0002-2831-5835; e-mail: evagilboa@gmai.com

Информация об авторе

Ива Гильбоа-Шехтман, PhD, профессор, руководитель программы по клинической подготовке, руководитель лаборатории эмоционального процессинга, департамент психологии и Мультидисциплинарный центр изучения мозга Гонда, Университет Бар-Илан, Рамат-Ган, Израиль, ORCID: https://orcid.org/0000-0002-2831-5835; e-mail: evagilboa@gmai.com

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