Social Anxiety Disorder in the Primary Care Setting

Larry Culpepper, M.D., M.P.H.

Generalized social anxiety disorder (SAD) is a disabling yet unrecognized condition for many individuals visiting primary care physicians. Social anxiety disorder carries a high risk of developing additional anxiety and mood disorders, including those with suicidal behaviors, as comorbidities, leading to a severe course. Screening and case-finding tools are available and can lead to the recognition of affected individuals. Once symptoms are recognized, an initial assessment will help to differentiate from other anxiety disorders and conditions that can be misdiagnosed as SAD. The primary care physician can manage treatment of SAD, which might require involving mental health professionals. Both pharmacotherapy, involving selective serotonin reuptake inhibitors, and psychotherapy, preferably with cognitive-behavioral therapy, can be effective. Long-term support strategies to monitor relapses or the development of additional psychiatric disorders or to provide anticipatory guidance at times of significant life transitions are additional primary care–based activities that can be helpful to the patient with SAD.

Social Anxiety Disorder (SAD), for which both genetic predisposition and neurophysiologic mechanisms are currently being elucidated, can have major disabling impacts on those affected. It also represents an initial psychiatric disability, often with an early onset for patients. About 80% of these patients will develop additional psychiatric disorders.1 Fortunately, if recognized, effective treatments can manage the symptoms and reverse or prevent further disabilities. Social anxiety disorder can be differentiated into discrete or nongeneralized SAD and generalized SAD. This article will provide an understanding of the evolution of SAD over the lifespan and ways in which the primary care physician can detect and manage it, with an emphasis on generalized SAD.

NATURAL HISTORY AND PRESENTATION OF GENERALIZED SOCIAL ANXIETY DISORDER

About half of patients diagnosed with SAD report that it has been with them for as long as they can remember.2 For the remaining population, the onset is recalled as occurring following a specific embarrassing incident—for most, during early adolescence. Behavioral inhibition is related to underlying subcortical fear circuitry and activation of the amygdala.3–5 These systems may be hypersensitive and easily conditioned to social stimuli,6 setting the stage for the development of symptoms that qualify individuals for the diagnosis of SAD, according to Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV),7 criteria (see Recognition section). Social anxiety disorder is also associated with severe defects in information processing and attentional biases that interfere with effective communication and role functioning (social, family, education, and work).8,9

Primary care physicians, especially family physicians and pediatricians, can identify families in which multiple generations have experienced SAD. Such familial burden is due to both genetic and environmental factors. Twin studies indicate that the heritability of SAD is about 50%.10 Others have found that children of parents with generalized SAD may be 10-fold more likely to develop SAD than those of unaffected controls.11 Serotonin- and dopamine-mediated mechanisms including the striatal dopamine transporter, the striatal dopamine D2 receptor, and the serotonin transporter have been identified as genetic factors in SAD.12–14 Affected parents not only might transmit SAD to their children by genetic mechanisms, they also can create family environments that put children at high risk. For instance, the Early Developmental Stages of Psychopathology Study15 found that children of parents meeting diagnostic criteria for SAD were about 5-fold more likely to develop SAD during adolescence. Independently, parental overprotection and rejection constituted additional risk factors (both with odds ratios = 1.4). Therefore, parental sensitivity to social disapproval and related anxieties can affect and influence the development of SAD.
The community-based Epidemiologic Catchment Area (ECA) study found that the full criteria for the diagnosis of SAD are first met either in early childhood (by age 5 years) or in early adolescence (around age 13 years).\textsuperscript{16} Social anxiety disorder may evolve from symptoms observable during infancy. Children demonstrating behavioral inhibition (e.g., shyness, less interaction with peers) when 4 months old were more likely to have symptoms related to SAD at follow-up assessment when 7 years old.\textsuperscript{17} Children demonstrating behavioral inhibition as early as age 2 to 6 years are over 3 times more likely to meet criteria for SAD\textsuperscript{18} and to remain at risk throughout adolescence. For those with childhood onset, the disorder is likely to persist without full recovery into adulthood. In those who have not received treatment, remission is the exception.\textsuperscript{19}

During childhood, SAD and its behavioral precursors might be expressed in primary care settings through parental concerns regarding shyness, school avoidance, clinging behaviors, social isolation, or oppositional behavior when interacting with others. Children with SAD have high levels of emotional overresponsiveness, social fear, dysphoria, social inhibition, loneliness, and general fearfulness. Moreover, they have poor social skills and often suffer from socially distressing events. Also, they are likely to cope with such events inadequately.\textsuperscript{20} Adults with SAD are more likely than controls to report traumatic childhood experiences, including separation from parents, parental marital discord, sexual abuse, familial violence, and childhood illness.\textsuperscript{21}

In those at risk, the intense social demands of adolescence, as teens become focused on their peer groups’ expectations, lead to the emergence of the full breadth of SAD symptoms. Adolescents with SAD express difficulties in initiating and maintaining relationships with the opposite sex, which may contribute to low self-esteem, demoralization, social isolation, and dysfunctional coping behaviors.\textsuperscript{19} Girls are more withdrawn and less likely to be sexually active. They are less skilled at negotiating social situations and demands and more anxious to be accepted and loved. They are also less likely to use effective contraception if sexually active. As a result, SAD is one of the early-onset psychiatric disorders that most strongly predicts adolescent pregnancy,\textsuperscript{22} and such a pregnancy might result in primary care contact at which the diagnosis could be recognized.

Adolescents also experience heightened internal expectations for performance in school or other settings. Test-taking represents one of the most feared situations for adolescents with social phobia. They are at severalfold increased risk for missing school, failing grades, being expelled, and dropping out of high school and college.\textsuperscript{23,24} These in turn may be reflected in concerns brought to the primary care physician. Social anxiety disorder is one condition that predicts nonattendance to college.\textsuperscript{25}

Adolescents suffering from SAD frequently develop dysfunctional coping strategies, such as abuse of substances including cigarettes and alcohol. Although the initiation of substance abuse usually is delayed by 1 to 2 years compared with controls, adolescents with SAD are more likely to become heavy users—and ultimately alcohol-dependent.\textsuperscript{19} These high-risk behaviors may bring adolescents to primary care attention.

During adulthood, SAD is associated not only with its direct symptoms, but with the consequences related to its long-term impact during formative school and adolescent years. These include economic and employment consequences, lower educational attainment, and less social and family support. Adults with SAD are at an increased risk of being poor (OR = 1.7) and of being in a low social class (OR = 3.1).\textsuperscript{19} The ECA study found them to be twice as likely to receive welfare or disability payments compared to those with no mental disorder (22.3% vs. 10.6%). While SAD is frequently comorbid with other psychiatric disorders, those with SAD and no psychiatric comorbidity had virtually the same rates as those with comorbidities, indicating that the financial dependency can be due to SAD alone.\textsuperscript{16}

In the ECA sample, 52% of adults with SAD were employed compared with 61% of those with no psychiatric disorder. Those with SAD who were working reported 6.9 sick days in the past 90 days compared with 3.1 days for controls.\textsuperscript{26} Another study found a 3-fold increase in unemployment and high proportions who missed work hours (8.3%) and reported impaired work performance (23.3%) in the past week.\textsuperscript{27} Not only do those with SAD have restricted job opportunities because of failure to complete high school, they are less likely to attain university or professional training (OR = 4.2).\textsuperscript{24} Inherent in the social restriction associated with SAD, patients report an increased disability in major social roles (OR = 4.2) and marked dissatisfaction with friends (OR = 5.2) and leisure activities (OR = 2.4).\textsuperscript{24} These patient characteristics can be indicators to the primary care physician to probe for SAD.

Marital status also can be compromised. The National Comorbidity Survey (NCS)\textsuperscript{1} found those with SAD to be twice as likely to remain single and 50% more likely to be separated or divorced than controls with no psychiatric disorder. As the severity of the disorder increases, so does the likelihood of single marital status.\textsuperscript{1,28} Social anxiety disorder prior to marriage also predicts marital violence, which may contribute to the intergenerational perpetuation of SAD.\textsuperscript{29}

During adulthood, individuals with SAD often develop adaptive lifestyles that provide relief from symptoms. These generally involve building an environment that keeps the individual away from frequent stresses caused by social interactions. If they marry, it often is to a childhood or family friend who then serves as an emotional buffer. They may excel at solitary occupations, including
technical fields such as computer programming. Other work adaptations might include shifting work schedules to avoid any type of socializing.

These adaptive lifestyles, however, may be disrupted by normative developmental progress. Spouses might progress in their jobs, community, or social circles, resulting in expectations for the SAD-affected individual, such as participating in a business- or community-related social event. Pregnancy and parenthood might bring similar demands as well. Success at solitary work tasks or managing high workloads might lead to expectations for the individual to assume a more responsible role, such as making presentations or assuming supervisory tasks that are highly stressful for the SAD-affected worker. Consequently, these demands might lead to dysfunctional behaviors, including the use of alcohol to deal with stress-related situations. Some of these behaviors might lead to the problems that are brought to the primary care physician’s office.

In summary, patients with generalized SAD are likely to have impairments related to neurologic dysfunction associated with hyperreactivity of the amygdala to interactional cues and defects in attention and information processing. These abnormalities often take their toll beginning in early childhood through impaired educational progress and impaired development of interpersonal competencies and social and family relationships. They can lead to decreased success in the workplace and reduced economic well-being. However, because of the long-term nature of these abnormalities, patients affected usually do not seek medical attention unless the source of these impairments is probed by the clinician.

**RECOGNITION AND ASSESSMENT OF SOCIAL ANXIETY DISORDER**

In primary care and other settings, it is the unusual patient who is treated for SAD because of complaints directly related to it. The vast majority of patients with SAD perceive their condition as a social problem rather than emotional or psychiatric. In the NCS, only 5.2% of those with pure SAD thought they might have an emotional problem or might profit from mental health counseling. The NCS-Replication (NCS-R), a community-based study including adults aged 18 years or older, found that only 3.4% of adult patients with SAD reported being treated within 1 year of the disorder onset. The study also observed that for those with SAD, the median duration of delay to first treatment of any mental health problem (treatment for “problems with your emotions or nerves or your use of alcohol or drugs”) was 16 years. Those reporting childhood or adolescent onset of SAD were much less likely (OR < 0.3 in all age groups less than age 30 years), compared with older adults, to have received any treatment, as were African Americans (OR = 0.5) and Hispanics (OR = 0.7) compared with non-Hispanic whites. Of those reporting SAD (including those with psychiatric comorbidities) in the NCS-R, 45.6% were receiving treatment, with 25.3% receiving treatment in the general medical sector, 24.7% from a mental health specialist, and 13.4% from non–health care sources. However, these data are somewhat misleading because of the high rates of psychiatric comorbidity and the great likelihood that SAD is not recognized or treated directly even when a comorbid condition is. Instead, lifetime treatment rates specific to SAD appear to range from 10% to 15%.19

Thus, for patients to be treated for SAD, the primary care physician must undertake screening or case-finding activities; otherwise, the condition is very unlikely to ever be diagnosed. To screen older adolescents or adults, the Mini-Social Phobia Inventory (Mini-SPIN)30 and other instruments have been shown to be useful. The Mini-SPIN (see Table 1) is a subset of 3 items from the Social Phobia Inventory (SPIN),33 a 17-item self-administered scale for generalized SAD. Compared with the social phobia module of the Structured Clinical Interview for DSM-IV (SCID),34 the Mini-SPIN demonstrated a sensitivity of 88.7%, specificity of 90%, positive predictive value of 52.5%, and negative predictive value of 98.5% in a general managed-care population.30 Overall, the scale has 90% accuracy (efficiency) in diagnosing the presence or absence of generalized SAD.30 The International Consensus Group on Depression and Anxiety has also proposed a simple 2-question screen, although it has not yet been validated (Table 1).31

For children, a number of research instruments or scales developed for specialty settings have been found useful, including the Social Anxiety Scale (SAS), the Social Worries Questionnaire (SWQ), and the social phobia subscale of the Screen for Child Anxiety Related Emotional Disorders (SCARED).35 If parents affirm the simple statement, “My child is shy,” follow-up evaluation for potential SAD is warranted, and this simple approach might be about as accurate as more extensive initial screening.

In addition to screening at periodic health assessment visits, SAD might be uncovered in patients by undertaking case-finding interviews of those who have risk factors that should elevate suspicion of SAD. These include the characteristics discussed in the preceding section. Since about
Table 2. DSM-IV Criteria for Social Anxiety Disorder (Abbreviated)\(^a\)

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<tr>
<th>Persistent fear of 1 or more social or performance situations in which the person is exposed to unfamiliar people or to possible scrutiny by others. The individual fears he or she will act in a way (or show anxiety symptoms) that will be embarrassing and humiliating.</th>
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<td>Exposure to the feared situation almost invariably provokes anxiety, which may take the form of a situational bound or situationally predisposed panic attack.</td>
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<td>The person recognizes that this fear is unreasonable or excessive.</td>
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<td>The avoidance, anxious anticipation, or distress in the feared social or performance situation(s) interferes significantly with the person’s normal routine, occupational (academic) functioning, social activities or relationships, or there is marked distress about having the phobia.</td>
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<tr>
<td>In individuals under age 18 years, the duration is at least 6 months.</td>
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<tr>
<td>The fear or avoidance is not due to direct physiologic effects of a substance (e.g., drugs, medications) or a general medical condition.</td>
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<td>The disturbance is not due to another mental disorder.</td>
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\(^a\)Adapted with permission from the American Psychiatric Association.\(^7\)

80% of those with SAD will develop at least 1 psychiatric comorbidity by adulthood, and 60% of children suffer from a second concurrent disorder,\(^20\) such case finding should extend to patients already recognized as suffering from an anxiety disorder, depressive disorder, or substance abuse. For these patients, case finding might involve the use of Mini-SPIN questions or a targeted interviewing approach that is appropriate to the symptoms or life circumstances of the individual patient. An additional group at high risk for SAD is composed of individuals presenting somatic symptoms commonly expressed during socially stressful situations. These range from blushing, profuse sweating, and palpitations to abdominal or chest pains and even full panic attacks.

Screening and case finding will help to identify a significant number of affected individuals with functional impairments who might benefit from treatment. In 1 family practice, 511 English-speaking adults presenting for routine medical care participated in a 2-stage screening for SAD, the result of which was verified by a structured diagnostic interview.\(^36\) Seven percent were confirmed as suffering from SAD. Over half (58.3%) of these had major depression, 30.6% had generalized anxiety disorder, and 27.8% had panic disorder. Those with SAD reported more impairment in all functional domains and made greater use of health care resources than patients without psychiatric disorders. However, less than 20% were receiving appropriate treatment.\(^36\) Others have found primary care recognition and treatment to be even less frequent; for instance, in 1 study of a large managed-care population, only 1 of 200 patients with SAD was receiving treatment for it.\(^37\)

To establish the diagnosis in those identified by screening or case finding, information regarding the DSM-IV criteria for SAD should be gathered. These criteria are presented in Table 2. Central to the diagnosis is the recognition of the patient’s intense fear of doing or saying something embarrassing or humiliating in social situations. Individuals often go into such situations with the expectation that they will present themselves poorly (“I will be boring and inappropriate”), and they overestimate the likelihood of negative outcomes (“My boss will think I’m inept”) and exaggerate the consequences of such negative outcomes (“I’ll lose my job and my friends”) in comparison to individuals with other anxiety disorders or nonanxious controls. They also are more likely to interpret such occasions as indicative of personal shortcomings of a profound and enduring nature.\(^38\)

Although SAD has been viewed as reflecting an extreme degree of shyness, shyness tends to be less stable and minimally impairing, while SAD is enduring and can be very disabling. To differentiate SAD from shyness, key criteria should be explored\(^39\):

- Fear of acting in a humiliating or embarrassing manner can be explored by asking, “What are your specific fears about (feared situation)? Are you concerned that you might do something embarrassing or humiliating? Are you concerned that others will notice your sweating hands, trembling, etc.?”
- Avoidance of social situations can be assessed by asking, “Have you found yourself avoiding situations because of your anxiety (worry, shyness, self-consciousness)? Have you missed work (or other function) to avoid such situations?”
- Other serious role impairment or dysfunction can be probed by asking, “What sorts of things do you think you’ve missed out on as a result of your social anxiety (self-consciousness, shyness)? Do you think your work or family or personal life have been affected?”
- Dysfunctional coping can be assessed by asking about drug or alcohol use to feel comfortable in social situations.\(^39\)

The DSM-IV criteria require that symptoms lead to “interference or marked distress,” a more stringent requirement than that used by the third edition of DSM (DSM-III) (“significant distress”). While 18.7% of 1 patient group met the criteria for SAD using a threshold of “moderate interference or distress,” this decreased to 7.1% for “marked interference or distress” (the DSM-IV requirement) and to 1.9% for “marked interference.”\(^40\) Thus, the primary care physician must exert judgment and engage the patient in deciding whether the functional impairments, life restrictions, and psychic discomfort experienced by the patient are ones for which the patient is willing to consider treatment.

In addition to the assessment of SAD, the primary care physician should determine the presence of other psychi-
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The NCS found that 81% of patients with SAD had at least 1 comorbidity, with 14.1% reporting 2 other disorders and 48.0% reporting 3 or more disorders. These included 57% with anxiety disorder, 37% with major depression, 23% with alcohol dependence, and 15% with other drug dependence. Unfortunately, suicidal ideation and behaviors are a considerable risk in patients with SAD, and this risk is heightened further in SAD patients with psychiatric comorbidities. Therefore, questioning patients about suicidality should be a routine part of a patient assessment. Sexual function should be assessed since decreased sexual arousal, poor sexual performance, marked avoidance, and less satisfaction from sexual activity are more frequent in those with SAD compared with controls.

The differential diagnosis for SAD should include other anxiety disorders and, in the case of panic attacks or other significant physical symptoms, possibly investigation of potential cardiac or other somatic conditions. In generalized anxiety disorder, patients might worry about social events and the difficulties they might encounter (e.g., the weather might be bad, the caterer will not come, or guests might not arrive on time). However, in SAD, the attention is primarily focused on their own performance during the event and how others will perceive them (“I’ll embarrass my spouse in front of his boss”). In panic disorder, the focus of concern is the panic attack itself, and the possibility of additional attacks, while in SAD the panic attack is secondary, caused by the anxiety over social expectations. Agoraphobia involves the fear of being in settings perceived as unsafe and is likely to lead to a panic attack. In posttraumatic stress disorder, social withdrawal might be related to concern that events might recall a past trauma or be unsafe.

A number of body-focused disorders may mimic SAD. Body dysmorphic disorder involves an obsessive perception that one is ugly and often involves compulsive compensatory behaviors. Olfactory reference syndrome often includes social avoidance and shame because of concern over imagined offensive body odors. Avoidant personality disorder as a diagnostic entity overlaps with SAD; however, those with the former exhibit more pervasive avoidant behaviors in a broader spectrum of situations. The distinction between avoidant personality disorder and SAD is important, since treatments are available that have been demonstrated as effective for SAD, and the prognosis for significant improvement in SAD is much better.

Depression is frequently comorbid with SAD and tends to have atypical features including increased appetite and increased sleeping. Of note, atypical depression often includes heightened sensitivity regarding rejection. The onset of depression places the patient at an increased risk of subsequent alcohol abuse disorders. Social anxiety disorder conveys a 3.5-fold increase in the risk of developing major depression and precedes depression in 75% of cases by at least a year. Social anxiety disorder also increases the severity of depression, with a 2.3-fold increased risk of persistence or new episodes of depression and a 6-fold increased risk of suicidality.

Establishing the proper diagnosis is critical to understanding the patient, focusing the patient education, engaging the patient in pharmacotherapy or psychotherapy, and establishing the proper focus of psychotherapy.

### PRIMARY CARE MANAGEMENT OF SOCIAL ANXIETY DISORDER

Given the complexity of SAD, establishing a therapeutic alliance is particularly critical to the successful management of SAD. Initial care might require response to a crisis in the patient’s life through counseling and support. As any crisis is stabilized, management using effective strategies in care of chronic disease can be helpful, beginning with patient and family education, agreement on patient-specific goals, treatment, follow-up, and monitoring. These should encompass SAD, as well as any other comorbidities.

Patients whose disorders have affected their relationships since childhood may not perceive the possible benefits of or be willing to commit to therapy, particularly psychotherapeutic interventions. Motivational interviewing, including identifying specific achievable goals important to the patient, can be helpful, possibly integrated into the patient education. Such goals can be updated as therapy progresses and symptoms abate.

Therapy might include pharmacotherapy or psychotherapy, especially cognitive-behavioral therapy, or both. Both are reviewed in depth in accompanying articles.

While antidepressants, benzodiazepines, and anticonvulsants have been shown to be effective in SAD, the selective serotonin reuptake inhibitors (SSRIs) are recognized as appropriate initial therapy. This recommendation is justified by the broad range of effectiveness of the SSRIs not only in SAD but also in comorbidities such as mood and anxiety disorders or body dysmorphic disorder. Selecting an SSRI with an intermediate half-life will prevent withdrawal effects if the medication is skipped or not available. In addition, it is important to select an SSRI (such as escitalopram, citalopram, or sertraline) with minimal side effects and low risk for drug-drug interactions through inhibition of the cytochrome P450 (CYP450) 2D6 isozyme or other pathways.

The benzodiazepines are effective in SAD, with both alprazolam and clonazepam having supportive clinical trial data in SAD, including one study of long-term treatment. In some patients, initial therapy with both an SSRI and a benzodiazepine might be helpful, particularly if the patient is in crisis or coping with a particularly stressful set of social demands. Tapering the benzodiazepine after the first weeks of use but retaining it as an available rescue medication in case of stressful situations might be helpful. How-
ever, some controversy exists regarding the symptoms for years, with resultant significant restriction in their lives. Especially for those who developed SAD as children, the benefits of pharmacotherapy can include a marked reduction in symptoms in social situations that were previously intensely dysphoric. However, such individuals often need help in learning new behaviors and expanding their social repertoire once SAD symptoms have been controlled. Individuals with SAD have sometimes been noted to be socially awkward, with their symptoms leading to self-fulfilling behaviors that reinforce their isolation.

Psychotherapy (especially cognitive-behavioral therapy) can be of great benefit both to initially control symptoms (for some patients, as an alternative to pharmacotherapy) and to gain a broadened set of functional competencies and, hopefully, improved quality of life. Consequently, a key role for the primary care physician is to encourage patients to seek psychotherapy and to direct them to a mental health specialist with expertise in SAD.

In a long-term setting, as patients improve through therapeutic interventions, the role of the primary care physician remains very important in monitoring and helping the patient to maintain and extend the goals achieved. Vigilance, not only for relapse of SAD symptoms but for the development of other psychiatric disorders, can lead to early treatment and minimization of functional decline. Monitoring and anticipatory counseling can be very helpful at times of normative life-stage transitions, such as moving from high school to college or work, marriage and childbirth, or progression of job responsibilities, including work demands to relocate.

The intriguing possibility that early recognition and intervention for SAD might prevent the progression to secondary development of additional anxiety and depressive disorders has been supported by observations regarding early intervention for generalized anxiety disorder. One group has estimated that 10% of mood disorders might be prevented by early intervention for SAD.

**CONCLUSION**

Given the complexity of diagnosing SAD, establishing a therapeutic alliance between the patient and the primary care physician is particularly critical to successful management and treatment. Patients with SAD symptoms since childhood may not perceive the seriousness of their condition or commit to therapy, both psychological and pharmacologic. Thus, care of this chronic disease should include patient and family education and treatment goals. Cognitive-behavioral therapy can be of great benefit, mak-

**Drug names:** alprazolam (Xanax, Niravam, and others), citalopram (Celexa and others), clonazepam (Klonopin and others), escitalopram (Lexapro), sertraline (Zoloft).

**Disclosure of off-label usage:** The author has determined that, to the best of his knowledge, alprazolam, citalopram, clonazepam, and escitalopram are not approved by the U.S. Food and Drug Administration for the treatment of social anxiety disorder.

**REFERENCES**

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