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Psychological File Review

Veteran's name: John Doe

DOB: XX/XX/XXXX

File #: XXX-XX-XXXX

Report date: X/XX/XXXX

Reviewer: Todd Finnerty, Psy.D.

Records Reviewed: I have reviewed all the records provided from the Veteran's claims file. No examination was conducted for this medical opinion; I have performed a thorough review of the Veteran's medical history.

Referral Questions: The Veteran is applying for service connection for sleep apnea secondary to PTSD with alcohol abuse.

Sleep apnea analysis: Mr. Doe experiences PTSD from his military service per his VA records. The diagnostic criteria for PTSD include two separate symptoms associated with PTSD reflecting recurrent and distressing dreams as well as sleep disturbance such as difficulty falling asleep or staying asleep or restless sleep. The chronic sleep impairment from this differs from the symptoms and impairment associated with sleep apnea. Mr. Doe is also diagnosed with sleep apnea based on his 1/23/23 sleep study from Acme Sleep Medicine.

I disagree with the 6/6/26 decision to deny the Veteran's service connection for sleep apnea secondary to PTSD; the rating decision erred in finding the contract examiner's opinion sufficient and persuasive. The contract opinion should not be considered sufficient for rating purposes and relying on it was a clear and unmistakable error. The contract examiner documented using an inappropriate burden of proof that was higher than at least as likely as not; i.e., the examiner documented requiring that the medical literature demonstrate "definitive" causation rather than at least as likely as not. Merriam Webster defines the word definite as "free of all ambiguity, uncertainty, or obscurity," and yet this is not what the examiner's task was- the examiner's task was to opine on at least as likely as not. Scientific literature tends to require 95% to 100% confidence, yet the Veteran's burden of proof was 50% or greater (at least as likely as not). The examiner also did not apply this same burden of proof to the laundry list of risk factors they provided in their own opinion. For example, there is no evidence of "definitive" causation that age alone or "male gender" definitely causes sleep apnea. Simply stating some evidence relates to an "association" or a correlation is not a sufficient rationale. The Veteran's burden of proof is not 100% certainty of causation (or the 95% or higher confidence often used in research studies), it is at least as likely as not. Rationales must relate to an analysis in this context in order to be sufficient, not a requirement of closer to 100% certainty of causation. I have evaluated the medical evidence and scientific evidence related to the Veteran's case and his burden of proof has been met.

The contract examiner opined that Mr. Doe's sleep apnea was due to obesity, but argued that Mr. Doe's obesity was not due to PTSD. However, the contract examiner failed to base this evidence on a sound review

of the psychological science. The examiner stated that weight gain is “multifactorial, and related to many factors” [*sic*] (here the examiner’s rhetoric gets out of control as he essentially just said the same thing twice- of course multifactorial means many factors). However, the examiner’s task was to do more than offer a vague list that was not necessarily related to the Veteran’s own medical evidence, it was to address this based on factors that appear in the Veteran’s own medical evidence (including factors such as the psychological issues which can at least as likely as not impact diet and exercise). The examiner noted that this list of factors includes “genetics, diet choices, exercise patterns, etc.” The examiner noted “genetics” yet did not describe any specific gene or gene combination that causes obesity, nor did he identify any specific gene or gene combination that the Veteran actually has been shown to possess that causes obesity. There was no indication of any gene in the Veteran’s medical evidence that would cause sleep apnea. The examiner noted diet “choices” and exercise patterns, but failed to address the fact that these can be heavily impacted by Mr. Doe’s mental health difficulties. Later in this opinion I cite extensive scientific research supporting that it is at least as likely as not that his PTSD impacted his eating behaviors and activity levels and at least as likely as not caused his obesity. Diet “choices” and exercise patterns are not simply driven by “choices,” these are behaviors that are heavily impacted by mental health symptoms. Given that psychology is often noted to be the study of human behavior and that I am an expert in behavioral health, I am the most qualified expert to comment on these behaviors. The examiner’s stigma-driven, biased opinion reflects that he clearly lacked the relevant expertise in mental health conditions to address this question. He focused on the Veteran’s “poor” diet “choices” and “lack” of exercise which likely “contributed in a significant way” to his obesity. I agree that his behavior contributed to his obesity; what is more his behavioral health condition at least as likely as not caused these behaviors. The non-expert C&P examiner is blaming the Veteran for his mental health symptoms. Instead of recognizing his food intake behaviors and activity levels for what they are—driven by mental health symptoms—the non-expert, adversarial C&P examiner is blaming the Veteran for his “choices” that he considers “poor,” and calling the Veteran out for what the examiner clearly views as character issues. This is a stigma-driven and fallacious rationale which reflects that the examiner is unlikely to fairly consider obesity as an intermediate step. The examiner also documented using a requirement that to consider obesity as an intermediate step the Veteran should be “physically precluded from all forms of exercise.” However, it is not the examiner’s job to re-wrote policy or re-adjudicate court findings. The Veteran’s PTSD can clearly impact his food choices and motivation to engage in activities, something that can at least as likely as not lead to weight gain. The examiner is attempting to singlehandedly re-write VA policy and re-adjudicate legal findings related to obesity as an intermediate step, yet this is not acceptable. The examiner’s bias related to mental health conditions clearly reflects a significant lack of expertise in behavioral health. The examiner failed to even address the extensive scientific evidence that reflects that his mental health conditions at least as likely as not impact both dietary choices and activity levels. If we were to extend the examiner’s non-expert reasoning to other mental health symptoms one would expect that the examiner would view the impact of mental health symptoms as a choice that should not be rated, since, for example, it didn’t preclude all forms of physically interacting with others or all forms of concentrating or all forms of dealing with stress.

The examiner has placed an inappropriate burden on the Veteran which is not in keeping with the court’s determination that obesity can be used as an intermediate step. The VA cannot simply sidestep its court mandated duties by relying on non-expert, stigma-driven, adversarial opinions from examiners who are clearly not willing to consider obesity as an intermediate step. The opinion should be disregarded as not sufficient for rating purposes and relying on it was a clear and unmistakable error. It is at least as likely as not that his mental health difficulties caused the obesity which the examiner agrees caused his sleep apnea.

The VA relied upon an opinion from the contract examiner that centered on a straw man logical fallacy. The contract examiner argued that sleep apnea and PTSD were “separate entities entirely.” This is an absurd,

fallacious argument. No one argued that sleep apnea and PTSD were the same entity. Sleep apnea is, of course, a separate entity from mental health disorders, but that does not mean that one disorder cannot lead to and/or substantially aggravate the other.

The VA's decision relied upon an opinion that PTSD and sleep apnea were not related, yet there is ample scientific evidence refuting this unsupported statement. It is notable that this statement is even refuted by the VA's own website. The VA's own website indicates sleep apnea has a "relationship with PTSD" (ex: <https://www.research.va.gov/topics/respiratory.cfm#research8>). There is ample evidence of multiple risk factors which show an association with sleep apnea-- one of these factors— at least as likely as not on par with the others or with stronger association than the others is PTSD; we also know the direction of the relationship as by definition PTSD is caused by trauma and PTSD is not caused by sleep apnea. These associated factors (ex: weight gain) can significantly contribute to the development of sleep apnea and/or substantially aggravate sleep apnea. There is scientific evidence that PTSD can also at least as likely as not cause sleep apnea through multiple pathways. Some C&P examiners perform a cursory review of data related to sleep apnea using resources such as UpToDate; UpToDate does not reflect an adequate literature review related to the evidence associated with sleep apnea being secondary to PTSD. It is not peer-reviewed research literature, it is geared toward giving a basic overview of the characteristics of sleep apnea to non-experts, and it does not address the growing body of primary source journal articles that are specifically related to sleep apnea secondary to PTSD. Citing sources such as UpToDate—or no sources at all-- is a sure sign that the examiner lacks relevant expertise in the subject matter. Some C&P examiners attempt to describe what sleep apnea is characterized by as if this is what it is ultimately caused by, however this confuses the signs and symptoms of the sleep apnea process for sleep apnea's cause; this is an error. Factors such as sleeping position, weight gain, dentition, and pharyngeal anatomy are symptoms of or may worsen the symptoms of the sleep apnea process but do not necessarily reflect the ultimate cause of sleep apnea. His PTSD has likely had a profound impact on the development and subsequent substantial aggravation of his sleep apnea. Pharyngeal anatomy explains only a minimal portion of the variability in measures like the Apnea-Hypopnea Index (AHI). We know it is not just an anatomical disease when surgery to alter anatomy is of minimal benefit to a large number of individuals with OSA. Individuals with OSA often likely have hyperarousal concerns— a low threshold for arousal— which is an underlying mechanism of obstructive sleep apnea. Individuals have difficulty activating the muscles and stabilizing breathing in a normal fashion. PTSD can lead to this hyperarousal. The supporting research is outlined below. It is at least as likely as not that the Veteran's sleep apnea is secondary to his PTSD.

Shah, et. al. (2024) conducted an interesting twin study related to PTSD and sleep apnea [see Shah AJ, Vaccarino V, Goldberg J, Huang M, Ko YA, Ma X, Levantsevych OM, Smith NL, Alagar N, Mousselli I, Johnson DA, Clifford GD, Bremner JD, Bliwise DL. *Posttraumatic Stress Disorder and Obstructive Sleep Apnea in Twins. JAMA Netw Open.* 2024 Jun 3;7(6):e2416352. doi: 10.1001/jamanetworkopen.2024.16352]. The authors found that PTSD was "independently" associated with sleep apnea. The authors noted "PTSD diagnosis and symptoms were associated independently with OSA, even after controlling for demographics, behavioral factors, cardiovascular risk factors, and familial factors." They noted that "each 15-point increase" in the PTSD checklist score (PCL) "was associated with a 4.6 (95% CI, 0.1-9.1) events-per-hour higher AHI. Current PTSD diagnosis was associated with an adjusted 10.5 (95% CI, 5.7-15.3) events-per-hour higher AHI per sleep-hour. Comparable standardized estimates of the association of PTSD symptoms and BMI with AHI per SD increase (1.9 events per hour; 95% CI, 0.5-3.3 events per hour) were found." The examiners concluded that they "found a strong dose-response association between PTSD and OSA. The twin design allowed close control of familial influences, and the sampling strategy using the Vietnam Era Twins Registry minimized bias. The results suggest that functional neurobiologic and stress pathways modulating respiratory

regulation and airway collapse are important in the etiology of OSA.” The authors note “our findings emphasize the need for more studies to examine mechanisms underlying endotypes of OSA that incorporate psychological stress pathways. Possible mechanisms include pharyngeal collapsibility and exaggerated loop gain, which describe the centrally mediated respiratory response to the mild carbon dioxide retention at the onset of sleep.⁴⁰ Posttraumatic stress disorder may cause a lower sleep arousal threshold and decreased autonomic and respiratory reflexes.⁴⁰ Nighttime PTSD symptoms, such as nightmares, may increase sleep fragmentation, which in turn may increase airway collapsibility.⁶ We speculate that specific brain pathways that may be altered in PTSD may also be involved.⁴¹ This speculation is supported by studies of OSA using functional brain imaging that have shown alterations in regions of the brain involved in stress regulation, such as the thalamus and anterior cingulate cortex.⁴² Neurologic substrates between the brain and the visceral organs that regulate respiration and pharyngeal patency may also be involved.” The authors noted that increased PTSD symptoms “were associated with statistically and clinically higher AHI. This analysis is, to our knowledge, the most rigorously controlled to date for examining the association between a psychiatric anxiety disorder such as PTSD and OSA.” This was a strong effect, as the authors noted “with our discordant twin models, we also found that the standardized effect size for PTSD symptoms was remarkably similar to BMI, which is one of the most established risk factors for OSA.” Examiners often note that obesity/BMI/excess weight is the “strongest risk factor” associated with obstructive sleep apnea, yet they should also consider these findings related to the BMI from Shah, et. al. (2024). The authors indicated that the severity of PTSD symptoms, as measured by the PTSD checklist, were “remarkably similar to BMI.” Examiners should consider the severity of PTSD as an independent risk factor given that the scientific research reflects that the severity of PTSD symptoms as a risk factor for OSA is “remarkably similar to BMI.”

The incidence of insomnia and obstructive sleep apnea (OSA) in service members is increasing. “Between 2005 and 2019, incidence rates of OSA and insomnia increased from 11 to 333 and 6 to 272 (per 10,000), respectively.” The incidence rates have “markedly increased” since 2005 [see Moore, et. al. (2021), *Incidence of Insomnia and Obstructive Sleep Apnea in Active Duty United States Military Service Members*. Sleep, doi: 10.1093/sleep/zsab024]. However, the evidence for service connection is not based on incidence rates alone. Significant associated factors that reflect both a causal relationship and substantial aggravation have been identified in the research literature and they are consistent with his mental health records. His mental health symptoms, which appear to be a response to traumatic stress from the military, have likely created a significant and substantial progression and increased severity of his sleep apnea above and beyond the natural progression of the disease. His sleep apnea aggravates his PTSD symptoms and his PTSD symptoms have substantially aggravated his sleep apnea. The course of the Veteran’s sleep apnea is not inconsistent with exacerbation tied to traumatic stress and other psychiatric conditions including depression. There is scientific evidence supporting a link between his psychiatric disorders and his sleep apnea. The research literature establishes that PTSD is commonly associated with sleep apnea and that there is an arousal-based mechanism initiated by PTSD that promotes the development of sleep apnea in trauma survivors. Sharafkhaneh, et. Al. (2005), in the article Association of Psychiatric Disorders and Sleep Apnea in a Large Cohort (*SLEEP*, Vol. 28, No. 11, 2005) found that sleep apnea is associated with a higher prevalence of psychiatric comorbid conditions in Veterans Health Administration beneficiaries, including PTSD. Compared with patients not diagnosed with sleep apnea, a significantly greater prevalence ($P < .0001$) was found for posttraumatic stress disorder in patients with sleep apnea. The research supports the presence of a significant connection between PTSD and sleep apnea, including an arousal-based mechanism initiated by PTSD that promotes the development of sleep apnea in trauma survivors: This is supported by Krakow, et. al. [see Krakow, B., Melendrez, D., Warner, T.D. et al. *To Breathe, Perchance to Sleep: Sleep-Disordered Breathing and Chronic Insomnia Among Trauma Survivors*. *Sleep Breath* 6, 189–202 (2002)] who note that emerging evidence invites a broader comorbidity perspective, based on recent findings that post-traumatic sleep disturbance frequently manifests with the combination of insomnia and a higher-than-expected

prevalence of sleep-disordered breathing (SDB). In this model of complex sleep disturbance, the underlying sleep pathophysiology interacts with PTSD and related psychiatric distress; and this relationship appears very important as demonstrated by improvement in insomnia, nightmares, and post-traumatic stress with successful SDB treatment, independent of psychiatric interventions. Continuous positive airway pressure treatment in PTSD patients with SDB reduced electroencephalographic arousals and sleep fragmentation, which are usually attributed to central nervous system or psychophysiological processes. Related findings and clinical experience suggest that other types of chronic insomnia may also be related to SDB. “We hypothesize that an arousal-based mechanism, perhaps initiated by post-traumatic stress and/or chronic insomnia, may promote the development of SDB in a trauma survivor and perhaps other patients with chronic insomnia. We discuss potential neurohormonal pathways and neuroanatomical sites that may be involved in this proposed interaction between insomnia and SDB” per the authors. These neurobiological processes are further reviewed by Kelly, et. al. (2016) [see *Understanding Recent Insights in Sleep and Posttraumatic Stress Disorder from a Research Domain Criteria (RDoC) Framework*; *Curr Sleep Medicine Rep*, 2016, 2:223–232]. They note that PTSD is associated with sleep disturbances, including insomnia, nightmares, REM abnormalities and “sleep-disordered breathing” such as in sleep apnea. They note that “recent studies have expanded our knowledge of the neurobiology of trauma and sleep. In addition, intervention research has provided valuable information about how sleep treatments affect PTSD symptoms and how PTSD treatments affect sleep symptoms.” Chronic activation of stress hormones (hypothalamo-pituitary adrenal axis activity) caused by PTSD is known to lead to a neural sensitization leading to upper airway dysfunction such as sleep apnea [see van Lier, et. al. (2013) *Sympathetic activity and hypothalamo-pituitary-adrenal axis activity during sleep in post-traumatic stress disorder: A study assessing polysomnography with simultaneous blood sampling*. *Psychoneuroendocrinology*, 38(1), 155-165; and Kritikou, et. al. (2016) *Sleep apnoea and the hypothalamic-pituitary-adrenal axis in men and women: effects of continuous positive airway pressure*. *European Respiratory Journal*, 47(2):531-540.] PTSD causes airway instability through this hyperarousal, leading to sleep apnea concerns. a low threshold for arousal during sleep in relation to airway narrowing is viewed as one specific phenotype of OSA [ex: see Osman AM, Carter SG, Carberry JC, Eckert DJ. *Obstructive sleep apnea: current perspectives*. *Nat Sci Sleep*. 2018 Jan 23;10:21-34 and Eckert DJ. *Phenotypic approaches to obstructive sleep apnoea - New pathways for targeted therapy*. *Sleep Med Rev*. 2018 Feb;37:45-59]. As noted by Eckert (2018) “people develop obstructive sleep apnoea (OSA) for different reasons. The ability to understand these reasons, easily identify them in individual patients, and develop therapies that target one or more of these reasons are the keys to unlocking new approaches for the treatment of OSA. In line with this approach, recent advances in OSA pathogenesis using upper airway and respiratory phenotyping techniques have identified four key causes of OSA. A narrow or collapsible upper airway ('impaired anatomy') is the primary cause. However, the anatomical contribution to OSA varies substantially. Indeed, impairment in pharyngeal anatomy can be modest and in many patients (~20%), pharyngeal collapsibility asleep is not different to people without OSA. Thus, non-anatomical factors or 'phenotypes' that modulate pharyngeal patency are crucial determinants of OSA for many people. These include impairment in pharyngeal dilator muscle control and function during sleep, increased propensity for awakening during airway narrowing (low respiratory arousal threshold) and respiratory control instability (high loop gain). Each phenotype is a potential therapeutic target.” Individuals with a low respiratory arousal threshold-- or hyperarousal concerns-- are particularly relevant when discussing sleep apnea secondary to PTSD.

Colvonen, et. al. (2015) [see *Obstructive Sleep Apnea and Posttraumatic Stress Disorder among OEF/OIF/OND Veterans*; *Journal of Clinical Sleep Medicine* 11(5):513-518] found that “PTSD symptom severity increased the risk of screening positive for OSA.” The authors concluded that “veterans with PTSD screen as high risk for OSA at much higher rates...” It is notable that the VA’s own website implies a “relationship” between sleep apnea and PTSD and cites this study for support (see the VA’s website at

<https://www.research.va.gov/topics/respiratory.cfm>). The VA's own website notes "sleep apnea (pauses in breathing that occur at night) can cause excessive daytime sleepiness, trouble concentrating, high blood pressure, cardiac and pulmonary disease, and motor vehicle accidents. Relationship with PTSD—In 2016, researchers at the VA San Diego Healthcare System and the University of California found that the risk of obstructive sleep apnea among Iraq and Afghanistan Veterans increased with the severity of their PTSD symptoms. The investigators looked at 195 Iraq and Afghanistan Veterans—more than 93% were men—who had visited a VA outpatient PTSD clinic for evaluation of their symptoms. Using clinical questionnaires to evaluate both levels of PTSD and sleep apnea risk, researchers found that nearly 70% of Veterans in the study were at high risk for developing sleep apnea, and that the risk increased with the severity of their PTSD symptoms. This was despite the fact that many of them did not have a high body mass index or high blood pressure, considered risk factors for sleep apnea." Zhang, et. al. (2017) [see *Prevalence of obstructive sleep apnea in patients with posttraumatic stress disorder and its impact on adherence to continuous positive airway pressure therapy: a meta analysis; Sleep Medicine, 36:125-132*] performed a meta-analysis and concluded the OSA is commonly seen in patients with PTSD. They noted that "patients with PTSD and OSA demonstrated significantly lower adherence to CPAP therapy." There is ample evidence that treating OSA improves PTSD symptoms. In Orr, et. al. (2017) [see *Treatment of OSA with CPAP is Associated with Improvement in PTSD Symptoms Among Veterans; J Clin Sleep Med, 13(1):57-63*] the authors describe "a growing body of research has suggested a link between obstructive sleep apnea and PTSD" and "prevalence estimations for OSA in PTSD patients range from 52% to 69%, with some as high as 95%." They note "treatment of OSA with PAP therapy is associated with improvement in PTSD symptoms." They note one factor is that "treatment of OSA with CPAP appears to reduce the frequency of nightmares in PTSD patients." The authors concluded that sleep apnea treatment "should be considered an important component of PTSD treatment for those with concurrent OSA." Psychiatric concerns in general have been noted to be associated with sleep apnea. Rezaeitalab, et. al. (2014) [see *The correlation of anxiety and depression with obstructive sleep apnea syndrome, Journal of Research in Medical Sciences, Mar; 19(3): 205–210*] studied individuals with obstructive sleep apnea. They found that "53.9% of the individuals had some degree of anxiety, while 46.1% demonstrated depressive symptoms. In terms of OSAS severity, this study showed that OSAS severity was associated with the frequency of anxiety, choking, and sleepiness (P : 0.001). According to polysomnographic results, we found that the majority of patients suffering from anxiety and choking (66.7% and 71.4%, respectively) had severe OSAS." Jehan, et. al. (2017) [see *Depression, Obstructive Sleep Apnea and Psychosocial Health, Sleep Med Disord, 1(3): 12*] note that "there is a co-linear relationship between" obstructive sleep apnea and depression." Patients with OSA have impaired health and their psychosocial health and daily performance also decrease. They note "because disturbed sleep can cause poor concentration, mood problems, anxiety, and MDD, these factors are also the part of poor daytime performance." Some postulate that weight gain may be a primary cause of sleep apnea, however it is interesting to note that treating sleep apnea with CPAP often actually leads to more weight gain, not less weight gain [see de Milo and Genta (2016), *Continuous Positive Airway Pressure and Weight Gain: Do We Know the Mechanisms? American Journal of Respiratory and Critical Care, 194(7): 915*]. The research evidence, and the course of the Veteran's disease, support a link between his psychiatric difficulties and sleep apnea.

Mr. Doe's report of his 1/23/23 sleep study from Acme Sleep Medicine included a BMI in the obese range; this is consistent with the obesity diagnosis in his VA treatment records. A classic but outdated view of OSA is that it is only structural, caused by obstruction of the upper airways and is predominantly found in obese individuals. As we've seen from the research this is an oversimplification of the evidence. The Veteran has a history of possible weight issues. However, it would be an error to suggest that any weight gain occurred in a vacuum. Factors associated with his psychiatric disturbance likely significantly contribute to weight gain.

The scientific literature links psychiatric disturbance to decreased activity and emotional eating which can lead to weight gain. While weight gain potentially contributing to sleep apnea could also likely be due to psychiatric disturbance, it would be an error to rely on opinions that sleep apnea was simply due to being overweight. This is not consistent with the current state of the scientific literature. In fact, neither being overweight nor the presence of structural abnormalities fully explains the presence of sleep apnea in a substantial number of cases. Being overweight is not sufficient to explain the existence of sleep apnea in many individuals. Simply suggesting that OSA was due to being overweight would reflect an overly simplistic and mechanistic view of sleep and obstructive sleep apnea which does not reflect the current state of the science. It would overlook the sleep regulating functions of the brain and the impact of psychiatric disturbance on the sleep regulating functions of the brain, including arousal-based mechanisms associated with sleep apnea which have been identified as a likely causally-based phenotype of sleep apnea. In fact, the scientific literature tells us that “a substantial portion” of patients with obstructive sleep apnea are “not obese” (Gray et. al. 2017). The authors of a 2017 study note that “a substantial proportion of OSA patients are not obese. Non-obese patients with OSA are a challenging group to treat with existing therapies... Our data also indicate that a key nonanatomical contributor to OSA pathogenesis, a low threshold for arousal, is likely to be particularly important in the pathogenesis of OSA in non-obese patients with OSA. A greater propensity for awakening in nonobese patients with OSA may also be a physiological factor contributing, at least in part, to poor CPAP tolerance in these patients. These findings have important implications for the treatment of OSA in nonobese individuals.” (Gray, et. al., 2017). In addition, “arousal” plays a significant role in sleep apnea [see Gray, et. al. 2017 *Obstructive Sleep Apnea without Obesity Is Common and Difficult to Treat: Evidence for a Distinct Pathophysiological Phenotype; Journal of Clinical Sleep Medicine*, 13(1): 81-88]. PTSD is also potentially linked to sleep apnea through weight gain, which can lead to sleep apnea. PTSD negatively impacts motivation (ex: reducing activity) and can lead to overeating. PTSD increases the risk for weight gain and obesity, which in turn further increases the risk for obstructive sleep apnea. The Veteran’s weight gain occurred in the context of PTSD. Subthreshold and threshold post-traumatic stress disorder (PTSD) are associated with binge eating symptoms in both men and women [see Braun J, El-Gabalawy R, Sommer JL, Pietrzak RH, Mitchell K, Mota N. *Trauma exposure, DSM-5 posttraumatic stress, and binge eating symptoms: results from a nationally representative sample. The Journal of Clinical Psychiatry*. 2019;80(6):19m12813]. Hoerster, et. al. (2015) noted in “PTSD and depression symptoms are associated with binge eating among US Iraq and Afghanistan veterans” [see *Eating Behaviors; Volume 17, April 2015, Pages 115-118*] that “PTSD and depression are common conditions among Iraq/Afghanistan Veterans. In the present study, PTSD and depression symptoms were associated with meeting binge eating screening criteria, identifying a possible pathway by which psychiatric conditions lead to disproportionate burden of overweight and obesity in this Veteran cohort.” Dorflinger & Masheb (2018), in the research article “PTSD is associated with emotional eating among veterans seeking treatment for overweight/obesity” [see *Eating Behaviors, Volume 31, December 2018, Pages 8-11*] presented findings that “suggest that emotional eating is common among veterans reporting PTSD symptoms, and that any degree of PTSD symptom severity is associated with more frequent emotional eating.” Mental health difficulties can have a significant impact on activity levels and eating behaviors, influencing weight gain. For example, it is notable that major depressive disorder in the DSM-5-TR (pg. 183) lists diagnostic criteria such as “markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day...” and “significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day...” and “psychomotor agitation or retardation,” and “fatigue or loss of energy nearly every day” and others. PTSD (pg. 302) includes criteria like “markedly diminished interest or participation in significant activities). Overeating and inactivity are symptoms of mental health difficulties which lead to weight gain and obesity. In addition, we know that psychotropic medications can lead to weight gain. “Psychotropic drug-related weight gain (PDWG) is a common occurrence” and “intersects with the elevated risk for obesity and associated morbidity that has been amply

reported in the psychiatric population” [see McIntyre RS, Kwan ATH, Rosenblat JD, Teopiz KM, Mansur RB. *Psychotropic Drug-Related Weight Gain and Its Treatment*. *Am J Psychiatry*. 2024 Jan 1;181(1):26-38]. The Veteran’s psychiatric difficulties increased the likelihood of weight gain. This is still a direct and proximate cause. It is at least as likely as not that his mental health concerns caused his weight gain. Weight gain is not simply a comorbidity; weight gain is a direct, physical, anatomical effect of PTSD.

Mr. Doe’s service connected PTSD is associated alcohol use, which is also service connected (self-medicating PTSD symptoms with alcohol). The medical literature also demonstrates that alcohol use can make sleep apnea worse. For example, a recent meta-analysis on the topic reviewed 1,266 studies and concluded “alcohol consumption is associated with worsening severity of snoring, altered sleep architecture, AHI, as well as lowest oxygen saturation among patients susceptible to snoring and obstructive sleep apnea” [see Burgos-Sanchez C, Jones NN, Avillion M, Gibson SJ, Patel JA, Neighbors J, Zaghi S, Camacho M. *Impact of Alcohol Consumption on Snoring and Sleep Apnea: A Systematic Review and Meta-analysis*. *Otolaryngol Head Neck Surg*. 2020 Dec;163(6):1078-1086]. The American Academy of Sleep Medicine (an organization I am a member of), has practice guidelines related to sleep apnea. The American Academy of Sleep Medicine’s practice guidelines related to obstructive sleep apnea include, as a standard of care in the field, that there be “patient education” on the impact of “alcohol avoidance” on sleep apnea. The “avoidance of alcohol” is also included as one of the “behavioral strategies” in managing sleep apnea [for the practice guidelines see *Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine (2009)*, Epstein LJ; Kristo D; Strollo PJ; Friedman N; Malhotra A; Patil SP; Ramar K; Rogers R; Schwab RJ; Weaver EM; Weinstein MD. *Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults*. *J Clin Sleep Med* 2009;5(3):263–276]. This reflects a recognition that the alcohol use which is tied to the Veteran’s mental health difficulties can substantially aggravate the Veteran’s sleep apnea directly (in addition to also leading to a history of excessive caloric intake in the form of alcohol).

The scientific literature supports a direct connection between sleep apnea and arousal associated with psychiatric disturbance, including PTSD. In addition, past VBA decisions have supported this interpretation. The scientific evidence and the Veteran’s medical records support a finding that the evidence for sleep apnea being secondary to his PTSD is at least in equipoise. What is established as quite likely is a bidirectional relationship between his psychiatric disturbance and his sleep apnea where each substantially aggravates the other, and that his sleep apnea was initially directly and proximately caused by his psychiatric difficulties. There is research evidence supporting a direct cause for sleep apnea associated with the arousal related to psychiatric disturbance. In this regard, based on the mechanism of hyper-arousal associated with both his psychiatric disturbance and sleep apnea, it is at least as likely as not that his sleep apnea is secondary to his service-connected psychiatric disturbance. Countless VBA decisions have been decided favorably based on this scientific literature, including, but not limited to: *Citation Nr: 19133904 (Docket NO. 18-05 0350 5/1/2019; Citation Nr: 19164013 (Docket NO. 15-12 470) 8/19/19; Citation Nr: 19136285 (Docket NO. 18-01 222) 5/10/19; Citation Nr: 20011070 (Docket NO. 19-05 577) 2/10/2020; Citation Nr: 19155477 (Docket NO. 18-28 822) 7/18/19; Citation Nr: 19107744 (Docket NO. 16-51 926) 2/1/19; Citation Nr: 19103608 (Docket NO. 17-05 487) 1/15/19; Citation Nr: 19144930 (Docket NO. 17-49 916) 6/11/19; Citation Nr: 19141163 (DOCKET NO. 18-18 750A) 5/29/19; Citation Nr: 19110858 (Docket NO 16-53 449), 2/12/19*. This is not an exhaustive list. This interpretation of the scientific literature should be utilized in favor of the Veteran as it would be arbitrary and capricious for the VA to treat the science differently for one Veteran over another and say the science says one thing for one Veteran but says the opposite for another Veteran.

Sleep apnea conclusions: It is at least as likely as not that the Veteran’s sleep apnea is the result of and would not have occurred but for the impact of his mental health difficulties. There is a link between his sleep apnea and the mental health difficulties from his military service. It is at least as likely as not (a 50

percent chance or greater) that the Veteran's sleep apnea is due to the psychiatric difficulties from his military service. His mental health concerns have created hyperarousal and other physiological issues including a risk for weight gain which in turn directly and proximately cause his sleep apnea while also subsequently substantially aggravating and worsening the course and severity of the disease. The Veteran's sleep apnea is at least as likely as not (a 50 percent chance or greater) substantially aggravated by his service-connected PTSD. His PTSD creates chronic sleep impairments which substantially impact the progression and treatment of his sleep apnea along with a risk for weight difficulties and treatment adherence concerns with PAP therapy which do the same. The issue is medically complex, as it requires knowledge of the interaction between multiple systems in the body. I am a psychologist with extensive training related to posttraumatic stress disorder and other psychiatric conditions; I am also trained in Behavioral Sleep Medicine and am a member of the American Academy of Sleep Medicine and the Society of Behavioral Sleep Medicine. The research supports the presence of a significant connection between psychiatric difficulties and sleep apnea, including an arousal-based mechanism initiated by PTSD that promotes the development of sleep apnea in trauma survivors. Also, weight gain is not simply a comorbidity; weight gain is a direct, physical, anatomical effect of PTSD. His mental health difficulties at least as likely as not impacted his eating behaviors and activity levels. As a result, his mental health difficulties at least as likely as not caused the direct effect on his anatomy of weight gain and obesity. Obesity is a significant risk factor and it along with PTSD itself are the most likely explanation for the presence of his sleep apnea. There is no support for the idea that he would have had sleep apnea in the absence of his PTSD and obesity. Sleep apnea does not cause itself. It is at least as likely as not that his PTSD caused his obesity which caused his sleep apnea. His sleep apnea would likely not have otherwise been impairing nor would he have otherwise developed sleep apnea at all.

As cited in the narrative above, examiners often note that obesity/BMI/excess weight is the "strongest risk factor" associated with obstructive sleep apnea, yet they should also consider the findings related to the BMI from Shah, et. al. (2024). The authors indicated that the severity of PTSD symptoms, as measured by the PTSD checklist, were "remarkably similar to BMI" as it relates to being a risk factor for sleep apnea. Examiners should also consider the severity of PTSD as an independent risk factor given that the scientific research reflects that the severity of PTSD symptoms as a risk factor for OSA is "remarkably similar to BMI." The relationship between PTSD and sleep apnea is therefore likely not just a vague association or correlation (and the Veteran's burden of proof is not 100% certainty of causation anyway), but a multiplicative interaction where PTSD works as both an independent risk factor leading to sleep apnea and a factor worsening weight gain which also contributes to the development and worsening of sleep apnea.

Simply stating some evidence relates to an "association" or a correlation is not a sufficient rationale. The Veteran's burden of proof is not 100% certainty of causation (or the 95% or higher confidence often used in research studies), it is at least as likely as not. Rationales must relate to an analysis in this context in order to be sufficient, not a requirement of closer to 100% certainty of causation. I have evaluated the medical evidence and scientific evidence related to the Veteran's case and his burden of proof has been met. I disagree with the 6/6/26 decision to deny the Veteran's service connection for sleep apnea secondary to PTSD; the rating decision erred in finding the contract examiner's opinion sufficient and persuasive. The contract opinion should not be considered sufficient for rating purposes and relying on it was a clear and unmistakable error. The contract opinion is addressed extensively in the narrative above.

As you are likely aware, per the VA's own OIG "some of the exams produced by vendors have not met contractual accuracy requirements. As a result, claims processors may have used inaccurate or insufficient medical evidence to decide veterans' claims." The OIG found that the contractors QTC, VES and LHI "failed to consistently provide VBA with the accurate exams required by the contracts." The OIG notes that "ALL THREE VENDORS HAVE BEEN BELOW THE CONTRACT'S 92% ACCURACY REQUIREMENT SINCE AT LEAST

2017.” Most errors— including a significant number that “had the potential to affect claims decisions—” aren’t corrected before the claims processors decided the claims per the OIG [See: <https://www.va.gov/oig/publications/report-summary.asp?id=5152>]. The VA’s own researchers have also identified contract exams as being plagued with errors. The VA researchers noted “there are several possible explanations for the observed deficiencies in contract exams, including lack of supervision, more limited access to VA treatment records, and inadequate training and experience. An additional explanation is that, unlike exams by salaried VA staff, contractors are paid a flat fee for each exam which is a small fraction of the typical fees paid for forensic psychological evaluation in the community. Thus, there is a financial incentive to complete exams quickly, which would preclude careful record review, psychological testing, and detailed report preparation.” The authors go on to suggest that “anecdotally, it is not uncommon for veterans seen by contractors needing to be reexamined, at times with requests for second and even third opinions to resolve “conflicting medical evidence” after a contract examiner rendered an opinion that contradicted those in the veteran’s records or in previous C&P exams. Inefficiencies resulting from poor exams increase the workload for both examiners and VBA personnel and increase the costs for VA C&P operations overall” [see Meisler, A. W., & Gianoli, M. O. (2022). *The Department of Veterans Affairs disability examination program for PTSD: Critical analysis and strategies for remediation. Psychology, Public Policy, and Law*].

His mental health difficulties at least as likely as not caused his weight gain and obesity by impacting his food intake behaviors and activity levels. His obesity was a substantial factor in causing his obstructive sleep apnea. It is at least as likely as not that the Veteran’s sleep apnea is the result of and would not have occurred but for the mental health difficulties and obesity associated with his mental health difficulties.

He requires the use of a breathing assistance device such as a CPAP based on his treatment records.

[Dr. Finnerty’s Signature appears here]

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Dr. Todd Finnerty is a psychologist in private practice in Columbus, Ohio. He has significant training related to PTSD, including VA-specific training through the contractors VES and QTC. Dr. Finnerty has had the same amount of training or more training than the third-party contractors used by the VA. In the past, Dr. Finnerty has performed hundreds of examinations on veterans for VA third-party contractors. Dr. Finnerty also helped make decisions on Social Security disability claims for the state of Ohio for approximately 19 years and has substantial experience in evaluating impairment. In this role Dr. Finnerty was named the 2012 “Disability Review Physician of the Year” by the National Association of Disability Examiners, Great Lakes Region and the 2010 “Consultant of the Year” by the Ohio Association of Disability Examiners. He has training in behavioral sleep medicine and is a member of the American Academy of Sleep Medicine and the Society of Behavioral Sleep Medicine. Dr. Finnerty is a forensic specialist and adheres to the American Psychological Association’s Ethical Principles of Psychologists and Code of Conduct as well as the APA’s Specialty Guidelines for Forensic Psychology (<https://www.apa.org/practice/guidelines/forensic-psychology>). These guidelines include the responsibilities of integrity, impartiality and fairness and note: “When offering

expert opinions to be relied upon by a decision maker, providing forensic therapeutic services, or teaching or conducting research, forensic practitioners strive for accuracy, impartiality, fairness, and independence. Forensic practitioners recognize the adversarial nature of the legal system and strive to treat all participants and weigh all data, opinions, and rival hypotheses impartially. When conducting forensic examinations, forensic practitioners strive to be unbiased and impartial, and avoid partisan presentation of unrepresentative, incomplete, or inaccurate evidence that might mislead finders of fact. This guideline does not preclude forceful presentation of the data and reasoning upon which a conclusion or professional product is based.” While it would be convenient if Veterans in need of first or second opinions on mental health related claims could seek them from treatment providers at the VA, VA policy outlined in VHA Directive 1134(2) Provision of Medical Statements and Completion of Forms by VA Health Care Providers recommends that VA mental health treatment providers not complete forms such as “mental health DBQ’s” in order to “maintain the integrity of the patient-provider relationship.” Therefore, both the VA and Veterans often must seek forensic specialists outside of a treatment relationship to provide independent opinions related to their case.

Notice to the VA from Dr. Finnerty: I am happy to cooperate with any needs or requests the VA may have related to the Veteran’s claim. For example, should anyone at the VA consider any forms or opinions I provide not authentic, insufficient, inadequate or otherwise incomplete, I will happily address those concerns free of charge to the Veteran and the VA (with a release of information/the permission of the Veteran of course). I respectfully request that the VA notify me of any disagreement with my opinions or concerns related to a potential insufficiency or inconsistency, as I will happily address those concerns at no charge and help the VA resolve any concerns related to form sufficiency or concerns related to potential inconsistencies in the evidence in a timely manner. If the VA has concerns related to the authenticity of any opinion from me, I will happily help resolve those concerns as well. This could help the VA avoid the expense of unnecessary contract exams, for example, and also help the VA arrive at the correct decision quickly, instead of requiring both the VA and myself to continually provide new opinions and evidence when the concerns likely could have been addressed by me in an expeditious manner in the first place.

I will also review any C&P examiner’s inconsistent medical opinions that the VA wants to provide to me to review. I will do this at no charge and address anything the VA views as inconsistent or other questions the VA may have for me; this is particularly relevant given the significant delays and difficulties Veterans sometimes experience attempting to get copies of their records related to their claims (such as contract C&P opinions). I of course stand by my opinions, so if the VA does not provide me with the timely opportunity to review any such sufficiency or inconsistency concerns, that will likely lead to the unfortunate instance of delays for the Veteran obtaining their evidence and then subsequent delays related to issuing additional opinions addressing that evidence. I of course will continue to provide these additional opinions addressing any erroneous denials and inconsistent examiner opinions at no cost to the Veteran.

I may be contacted here:

Office: (614)924-8427

My assistant’s direct line: (614)696-6706

My cell phone #: (330)495-8809

Fax # where records requests can be sent: (614)573-0505

A guaranteed free phone appointment time to speak with me directly can be scheduled here:

<https://toddfinnerty.com/schedule.html>