

科技部人文社會科學研究中心  
補助科技部跨領域研究計畫之前置規劃案結案報告

從創傷後壓力症到酒精使用障礙症：社會相關因子的潛在影響效果

**The Path from Posttraumatic Stress Disorder to Alcohol Use**

**Disorder: The Potential Influential Effect of Social-related Factors**

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## 中文摘要

創傷事件具有極端、突然、不尋常的威力，容易引發個體的害怕、焦慮、退縮、和逃避，而經歷創傷事件可能促使個體發展出創傷後壓力症（PTSD）。依據自我藥療假說，因為受創個體傾向使用飲酒來因應創傷經驗，患有 PTSD 個體更容易發展出有問題的飲酒行為，像是酒精使用障礙症（AUD），導致 PTSD 和 AUD 具有高共病。但 PTSD 和 AUD 關聯性的可能機制目前尚不清楚，有鑑於情緒調節困難是 PTSD 和 AUD 的共同影響因子，加上低社會支持是發展出 PTSD 的一個重要預測因子。因此，本規劃案企圖從生理、認知、社會等三個角度，探討情緒調節和社會支持兩個因子在 PTSD 和 AUD 共同發生機制上的可能角色。在子計畫一中，研究假設創傷經驗會透過影響下視丘-腦下垂體-腎上腺（HPA）此壓力賀爾蒙系統中的皮質酮水平，而影響 PTSD 和 AUD 的關聯性，故在子計畫一中，將檢查 c-Fos IL-1 $\beta$ 、IL-6、TNF $\alpha$  等神經炎症細胞因子，作為上述途徑中的生物指標。在子計畫二中，將從社會認知因子來探討受創個體於不安全依戀關係中發展出的社會認知缺損，不安全依戀傾向可能會讓受創個體受到適應不良依戀相關基模的激發的影響，無法正確地處理他人的情緒訊息，產生情緒調節的困難，進一步也會損害受創個體與他人建立信任關係的能力，降低尋求社會支持傾向，進而有較高的風險發展出創傷後壓力症狀和飲酒行為。最後，在子計畫三中，考量負向社會反應可能是創傷後心理病理的重要風險之一，包括覺知社會支持、社會認可，對於災後心理健康通常具有直接或緩衝的作用，以及覺知汙名可能會透過降低社會支持進而影響 AUD 的嚴重度，故研究三將探討覺知社會反應對於 PTSD 與 AUD 的中介效果。綜上，本規劃案企圖從生理、認知、社會因子等三個角度，來完整地探討 PTSD 和 AUD 共病的可能機制。

關鍵詞：酒精成癮、生理、認知、社會、創傷

Traumatic events have extreme, sudden, and unusual features that can trigger fear, anxiety, withdrawal, and avoidance, which in turn, make traumatized individuals develop post-traumatic stress disorder (PTSD). According to the self-medication hypothesis, traumatized individuals tend to use alcohol to cope with their traumatic experiences. As a result, people with PTSD are more likely to develop problematic drinking behaviors, such as alcohol use disorder (AUD), which leads to high correlations between PTSD and AUD. However, the possible mechanism of the relationship between PTSD and AUD is still unclear. Because difficulty in emotion regulation is a common factor of PTSD and AUD, and low social support is an important predictor of the development of PTSD. Therefore, this proposal will explore the co-occurrence mechanism of PTSD and AUD from three perspectives of physiology, cognition, and society, and two factors of emotional regulation and social support. In the first project, trauma experience may have an impact on PTSD and AUD by affecting the level of corticosterone in the Hypothalamus - Pituitary gland - Adrenal gland stress hormone system. The first project aims to examine neuroinflammatory cytokines such as c-Fos, IL-1 $\beta$ , IL-6, TNF $\alpha$  as biological indicators in the above-mentioned pathway. In the second project, we will explore the role of social cognitive impairment that traumatized individuals develop in insecure attachment relationship. By the activation of maladaptive attachment-related schemas, the insecure attachment tendency may cause traumatized individuals to bias the emotion information processing and result in difficulty in emotional regulation. The difficulty will further damage the ability to establish trust relationships with others and reduce the tendency to seek social support, which causes a higher risk for developing post-traumatic symptoms among traumatized individuals. The second project thus aims to examine the potential comorbidity mechanisms of PTSD and AUD from social cognitive factors. Finally, negative social reactions, including awareness of social support and social acknowledgment, may be one of the important risk factors for post-traumatic psychopathology and usually have a direct or buffering effect on post-disaster mental health. In addition, stigma may affect the severity of AUD via social support. Therefore, the purpose of the third project aims to explore the mediating effect of perceived social responses on PTSD and AUD. In sum, this proposal attempts to explore the roles of emotional regulation and social support on the co-occurrence mechanism of PTSD and AUD from three perspectives: physiology, cognition, and social factors.

*Keywords: alcohol addiction, biology, cognition, sociology, trauma*