Corrigendum

Corrigendum to “Somatostatin Negatively Regulates Parasite Burden and Granulomatous Responses in Cysticercosis”

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In the paper titled “Somatostatin Negatively Regulates Parasite Burden and Granulomatous Responses in Cysticercosis” there was an error in the Discussion and Bibliography section.

The changes made to the references section are as follows. Reference 26 has been added: H. Wu, X. Chen, Y. Deng, X. Huang, and Z. Zhang, “Effect of somatostatin on modulation of IL-10 and TGF-beta I during acute pancreatitis,” Journal of Sichuan University (Medical Science Edition), vol. 34, pp. 315-316, 373, 2003.


Somatostatin is known to be an inhibitory neuropeptide [3, 10, 11, 26]. The inhibitory role of somatostatin is depicted in studies wherein it has been shown to downmodulate production of cytokines such as IFN-γ and IL-10. For example, studies using human peripheral blood mononuclear cells demonstrated that somatostatin inhibited the secretion of IFN-γ [11]. Similarly, other studies have shown that somatostatin decreased the production of IFN-γ by splenocytes and T lymphocytes isolated from murine schistosome granulomas in vivo and in vitro [3]. Furthermore, validation of the inhibitory role of somatostatin is seen in studies wherein somatostatin has been shown to depress the increase of IL-10 that is noted in response to acute pancreatitis [26] or systemic lupus erythematos [10]. Therefore our studies demonstrating more IFN-γ and IL-10 in the absence of somatostatin coincide with the above studies, wherein somatostatin has been shown to have an inhibitory role in IFN-γ and IL-10 production.

The finding of reduced IL-1β in the absence of somatostatin is not completely unexpected. Earlier studies examining the ability of somatostatin to modulate production of IL-1β, along with TNF-α and/or IL-6, are conflicting. Some studies showed that somatostatin stimulates the production of IL-1β, TNF-α, and/or IL-6 by human blood cells [27, 28], as well as the expression of IL-1β in articular tissues of rats with ongoing adjuvant-induced arthritis [29]. However, other studies showed that somatostatin decreased secretion of one or more of these cytokines [7, 9, 29–32]. Thus our results demonstrating lesser IL-1β levels in the absence of somatostatin coincide with the first set of studies, wherein somatostatin has been shown to have a stimulatory role in IL-1β production.

There are various studies performed using knockout mice by Dr. Terrazas’ group [33–36]. The protective role of Th1 responses has been evident in these knockout studies. For example, T. crassiceps-infected STAT6−/− mice have been shown to mount a strong Th1 response in the absence of Th2 development and to control the infection [35]. Similarly, other studies wherein knockout mice with decreased Th1 responses such as IL-12 p35−/− mice have been shown to demonstrate increased susceptibility to the larval stage of T. crassiceps [34].

The above studies using various knockout animals and other studies wherein IFN-γ and anti-IFN-γ antibody administration, respectively, led to lower and higher parasite levels [21] indicated that a Th1-type response is essential for resistance against experimental cysticercosis, which coincides with our observation that Th1 cytokine IFN-γ plays an important role in limiting infection in the somatostatin knockout mice.

In contrast to Th1 cytokines, Th2 cytokines are implicated with susceptibility with this parasitic infection. For example, studies done by Dr. Terrazas’ group have shown that IL-10 increases parasite load [21]. Similarly, studies using toll-like receptor 2 knockout mice demonstrated a reduction in the production of proinflammatory cytokines, resulting in a Th2 bias and significantly impaired resistance to T. crassiceps infection [33]. Furthermore, another study showed that STAT4−/− mice that mount a strong Th2 response were highly susceptible to infection and displayed large parasite loads [36]. However our studies showing lower parasite levels in the somatostatin knockout mice that have higher IL-10 levels implicate that IL-10 does not have a role to play in the parasite levels in our somatostatin knockout mice or additionally and/or alternatively it may implicate that other cytokines such as IFN-γ and IL-1β may be the predominant players in inducing parasite reduction and may far outweigh the parasite-stimulating effects of IL-10.

Our findings have potential implications for treatment and prevention of the detrimental effects of granulomatous inflammation induced as a result of anthelmint treatment in patients with neurocysticercosis and viable cysts. Current options for management of patients with viable cysts include anthelmint treatment along with corticosteroid administration aiming at reducing granulomatous inflammation. However, corticosteroids can have severe side effects and cannot be used in patients with concurrent latent tuberculosis, strongyloidiasis, and optical cysticercosis. Our finding that somatostatin downmodulates granuloma growth suggests the possibility of using somatostatin analogues, instead of corticosteroids, to downmodulate granulomatous inflammation in the brain of neurocysticercosis patients.
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