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Influence of melatonin and catecholamine(s) on cellular immune function: An *in vitro* study in human

Seema Rai[1], Cosentino Marco[2] and Chandana Haldar[3]
[1]Guru Ghasidas Vishwavidyalaya, India
[2]University of Insubria, Italy
[3]Banaras Hindu University, India

$CD4^+CD25^+$ regulatory T cells constitute 5-10% of the peripheral CD4+ T cells while 80-85% population belongs to effector T cells (Teffs). These specialized T cells play very crucial role in maintenance of immune homeostasis and also contributing to the immunologic self tolerance being critically involved in immunological disorder. The working hypothesis for the present study is that, effector T cells (Teffs) may release melatonin where as Treg cells are reported to release catecholamines. Cetacholamine in turn, might influence melatonin production which may have some impact on the cytokine production like interleukin-2, interleukin-10 (IL-2) including TGF b and Fox P3 in Teff cells. Melatonin (n-acetyl-5-methoxy tryptamine), the first methoxy indoleamine identified in mammalian tissue is a measure chronobiotic agent which synchronizes physiological process such as reproduction, metabolism, seasonality thermoregulation and immunity. The present study proposes a conceptual model for a bidirectional regulatory loop of arm of T regulatory and effector T cells. We found that $CD4^+CD25^-$ T effecter lymphocyte contains high endogenous melatonin where as $CD4^+CD25^+$ T regulatory lymphocyte contains high catecholamine. Catecholamine treatment (1 μM) to $CD4^+CD25^-$ T effecter lymphocyte completely diminished the level of melatonin *in vitro*. Presence of Mel1a and Mel1b receptor was noted in both $CD4^+CD25^-$ T effecter as well as in $CD4^+CD25^+$ T regulatory lymphocytes. Melatonin treatment (10-7) was able to activate $CD4^+CD25^-$ lymphocyte by increasing the production of IL-2 *in vitro*. Melatonin receptor antagonist luzindole treatment decreased IL-2 production and IL-2 expression by T effector lymphocytes thus confirming the role of melatonin in cytokine production. Melatonin treatment also increased Fox P3, TGF β expression significantly. Increase in Fox P3, TGF β, TH and IL-10 following melatonin treatment in T regulatory cells represented some internal regulation between T effecter and T regulatory cells. Therefore, we may suggest a tonic control of immune system by those two subsets of lymphocytes mediated via specific membrane/nuclear receptors. Based on the obtained data, we suggest that Tregs release catecholamines & expresses TH, leading to impaired suppressive activity of Tregs towards the Teff proliferation through reduced production of IL-10 and transforming growth factor-beta (TGF-β) which are the main immunosuppressive cytokines mainly produced by Tregs. Whereas IL-2 represented an important control point for manipulating the balance between Tregs and Teff cell function *in vivo*. Such a control may be explained in terms of a trade-off mechanism/regulation between the endogenous melatonin of T effecter cells and catecholamine of the T regulatory cells in maintaining the homeostasis in the immune system depending upon the physiological status or required status of the cells.

raiseema_pk@rediffmail.com