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# Role of sICAM-1 and sVCAM-1 as biomarkers in early and late stages of schizophrenia

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ABSTRACT

Schizophrenia (SZ) is a neuroprogressive disorder presenting with biochemical, functional, and structural changes, which differ from early to late stages of the illness. We explored the differences in serum levels of soluble intercellular cell adhesion molecule-1 (sICAM-1) and soluble vascular cell adhesion molecule-1 (sVCAM-1) between early and late stages of SZ, in regard to clinical characteristics and treatment application.

Serum levels of sICAM-1 and sVCAM-1 were measured in 80 patients with SZ (40 early stage; 40 late stage), and compared with 80 healthy controls, matched by age, gender, body mass index, and smoking habits with each SZ group. Serum levels of sICAM-1 and sVCAM-1 were measured using ELISA. The severity of psychopathology was assessed using the Clinical Global Impression Scale and five-factor Positive and Negative Symptoms in Schizophrenia Scale.

After adjustment for confounders, we noticed normal levels of sICAM-1 in the early stage, and elevated levels of sICAM-1 in the late stage of SZ. sVCAM-1 levels were decreased in both stages of SZ. Higher sICAM-1 levels have been related to more pronounced cognitive deficit and excitement symptoms in the early stage of SZ and to favorable characteristics of treatment application in both stages.

SZ is associated with changes in the levels of adhesion molecules that vary from early to late stages of the illness. This implies that the concept of biochemical staging is applicable in SZ, at least for markers of cellular adhesion.



## 1. Introduction

Schizophrenia (SZ) is a neuroprogressive disorder that presents with biochemical, functional, and structural changes, which differ from the early to late stages of the illness (Agius et al., 2010; Davis et al., 2014; Meyer, 2013; Schennach et al., 2012). Differentiating the evolution of SZ into stages aims to distinguish the earlier and milder clinical symptoms from those that mark illness progression. Such a staging model of SZ is based on the longitudinal development of the disorder, ranging from the asymptomatic at-risk stage to the emergence of prodromal symptoms, first episode of illness, and recurrence and eventually to a chronic and treatment-resistant forms (Agius et al., 2010). In addition, well-defined clinical presentations are associated with each individual stage. Prospective studies and retrospective research indicate that behavioral deviations can be dated to early infancy, progressing to subtle changes in cognition and effect as well as sub-threshold psychotic symptoms and finally to full-blown psychosis in late adolescence. On the other hand, chronic forms are most often characterized by the progressive deterioration of function and more prominent changes in the neurobiological basis of the disorder (Agius et al., 2010; Davis et al., 2014; Meyer, 2013; Parnas, 1999).

According to the recent literature, inflammatory processes are strongly involved in the impairment of several physiological

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