Interaction caused the lipid content in jurkat t cells to increase.

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Abstract

Plasma membrane domains that form at T-cell antigen receptor (TCR) signalling foci transmit activating stimuli to T lymphocytes. In this study, the molecular lipid makeup of immunoisolated TCR activation domains was determined. In comparison to control plasma membrane fragments, we found that they accumulate cholesterol, sphingomyelin, and saturated phosphatidylcholine species. This presents the first direct evidence that the molecular lipid composition of TCR activation domains is distinct and resembles that of liquid-ordered raft phases in model membranes. Interestingly, plasmenyl phosphatidylethanolamine and phosphatidylserine were also enriched in TCR activation domains. Plasma membrane condensation at TCR signalling was compromised when polyunsaturated fatty acids were used to modify the T-cell lipidome. Foci and caused a disruption in the molecular lipid composition. These findings relate early TCR activation responses and specific plasma membrane condensation at sites of TCR activation to the accumulation of particular molecular lipid species.

Keywords: TCR signalling, Quantitative shotgun lipidomics, Lipid rafts, Plasma membrane domains.

Introduction

When T lymphocytes couple with the appropriate antigen-presenting cells, they become activated. The outcome of this encounter is determined by numerous receptor-ligand interactions at the immunological synapse, the point of contact between these cells. When the T-cell antigen receptor (TCR) binds to a cognate peptide-major histocompatibility complex expressed on the surface of the antigen-presenting cell-it sends out the crucial activation signal. Signaling protein complexes that assemble in the T-cell plasma membrane transmit the TCR signals to the T-cell interior [1].

Cholesterol, sphingolipids, and saturated glycerophospholipids are functionally segregated in lipid rafts that are found in plasma membrane domains involved in TCR signal transduction. Lipid rafts are suggested to adopt a condensed liquid-ordered (Lo) state that coexists with a non-raft liquid-disordered bilayer on the basis of model membrane studies. Planar cholesterol ring systems align with the saturated fatty acid moieties of sphingomyelin (SM) and phosphatidylcholine (PC) during the Lo phase (SM). The idea of raft domains at TCR signalling sites has received experimental support from this biophysical perspective: The T-cell plasma membrane domains involved in TCR activation are more compressed than other plasma membrane regions, according to research using Laurdan 2-photon fluorescence microscopy [2-3].

In this study, we directly tested the hypothesis that the plasma membrane's TCR activation domains have a distinctive molecular lipid composition. In order to accomplish this, we used mass spectrometry to conduct a comprehensive analysis of the molecular lipid composition of immunoisolated native TCR activation domains and compared it to control plasma membrane fragments that were enriched in transferrin receptors (TfRs). We demonstrate for the first time that distinct glycerophospholipids species, SM, and cholesterol are functionally segregated at TCR activation domains, highlighting the significance of lipid composition in defining these plasma membrane domains [4].

The immunoisolation process resulted in the production of two distinct plasma membrane fragments, each of which had a different protein composition and was similarly depleted of lipids and proteins from intracellular compartments. It should be noted that TFR was present in some CD3 immunoisolates, indicating that some of the plasma membrane was not signalling. The molecular lipid makeup of the TCR activation domains in the T-cell plasma membrane was examined globally in this study. For the first time, this analysis offers concrete proof of the lateral segregation of various lipid species into membrane domains during TCR activation [5].

Conclusion

The immunosuppressive activity of this steroid may be caused by the CPHS-induced disorder in the lipid organisation of the plasma membrane (9, 10). The presence of a negatively charged phosphoserine group on the exterior of lymphocytes may influence the activity of receptors mediating their contact with other cells because the immune response depends on cell-cell communication. Additionally, the progression of signals

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that originate from the cell surface inside the cells may suffer as a result of the CPHS interaction with the sphingolipid-cholesterol-based domains of plasma membrane. This will be made clearer by looking at how CPHS affects T cells that have been exposed to soluble and cellular stimuli.

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