presented technique of plasma membrane sheet preparation from nonadherent cells may facilitate research in this field. It must be, however, mentioned that a plastic view of signal transduction across the plasma membrane can be achieved only by combination of various mutually complementary approaches.

Conclusions

Three techniques of isolation of plasma membrane sheets from nonadherent BMMC mast cells have been developed. One of them, based on adsorption of leukocytes to glass surface, turned out to be very promising and provided many scientific data (*article E*).

Activation of RBL mast cells by FcεRI receptor dimerization led to increase of Grb2 adaptor content in the plasma membrane. However, by contrast to the case of receptor multimerization, this Grb2 did not significantly colocalize with FcεRI, and, by immunolabeling of membrane sheets, distribution of FcεRI was not distinguishable from the distribution on nonactivated cells (*article A*). BMMC, in contrast to RBL cells, after multimerization of FcεRI did not form larger aggregates of this receptor than nonactivated cells did. FcεRI multimerization led to its internalization of comparable intensity and overall dynamics in BMMC and RBL cells, but local redistribution of FcεRI fundamentally differed between these two cell types (*article E*). Established model of large (approximately 200 nm) signaling domains of FcεRI receptor has been challenged.

NTAL adaptor protein has been clustered in the plasma membrane and distributed analogously to LAT adaptor, and also their topographic relations to FcεRI receptor and Thy-1 glycoprotein were analogous. In RBL cells, after FcεRI multimerization, both adaptors were occasionally localized at the periphery of FcεRI aggregates; despite it, however, LAT and NTAL were organized in distinct domains in the course of BMMC as well as RBL cell activation (*articles B, E*). Both adaptors strongly colocalized with aggregated Thy-1 glycoprotein, but even under these conditions they preserved their distribution in separate domains (*article C*). These results support the view of membrane rafts as a system constituted by interplay of lipidic and protein interactions.

Neither excessive, nor decreased expression of NTAL adaptor, which is a negative regulator of mast cell signaling, affected FcεRI distribution in the course of RBL cell activation. These changes in amount of NTAL molecules in the plasma membrane caused corresponding changes of NTAL domain size, but the character of NTAL distribution was not changed (*article D*).

Thy-1.1 and Thy-1.2 isoforms were localized to a great extent independently within the plasma membrane. Aggregation of Thy-1.1 lead to only weak coaggregation of Thy-1.2. By contrast, molecules of identical isoform showed much higher coherence (*article C*).