Within eukaryotic cells a significant subset of proteins and lipids undergoes glycosylation, *i.e* enzyme-mediated attachment of glycans. Sugar parts of glycoproteins and glycolipids fulfill numerous important functions including various biological recognition events, therefore alterations in their biosynthesis causes severe metabolic disorders. Enzymes catalyzing glycan attachment, *i.e.* glycosyltransferases, act within the highly specialized, membrane-bound organelles, namely Golgi apparatus and endoplasmic reticulum (ER). Glycosylation enzymes utilize nucleotide-activated forms of monosaccharides, *i.e.* nucleotide sugars, which are formed within the cytosol. Catalytic centers of these enzymes face the interior of the Golgi apparatus and ER. However, membranes of these organelles *per se* are impermeable to nucleotide sugars. Despite this limitation they are able to access the above mentioned compartments due to existence of highly specific multitransmembrane transporter proteins, namely nucleotide sugar transporters. UDP-galactose is a substrate for galactosylation reactions. In mammalian cells SLC35A2 is the main protein responsible for delivery of UDP-galactose to respective enzymes and no other functions have been attributed to this transporter so far. However, we strongly believe that this protein plays many other important roles. Our preliminary data suggest that SLC35A2 is required for proper localization of certain glycosylation enzymes. We also found that this protein influences the arrangement of vimentin intermediate filaments, which in turn are important for proper distribution of some organelles including endosomes, lysosomes and mitochondria, all of which are perturbed in SLC35A2-deficient cells. Our findings clearly demonstrate that a nucleotide sugar transporter may affect processes occurring within the cytosol - a phenomenon which has not been suggested before. We also believe that cells are able to obtain a subset of monosaccharides from lysosomal degradation of glycoconjugates that are not useful anymore and to subsequently utilize them for incorporation into newly synthesized glycans. If these two processes were spatiotemporally coupled, galactose released from the interior of lysosomes could be readily activated in the cytosol and immediately sequestered by SLC35A2 to be transported across Golgi and ER membranes. Our preliminary results suggest that SLC35A2 plays an important role in this phenomenon. The overall aim of our proposal is to demonstrate that SLC35A2 facilitates galactosylation of macromolecules in many different ways by transporting UDP-galactose across Golgi and ER membranes, supporting proper Golgi localization of galactosyltransferases and increasing cytosolic content of galactose.