Sterols are lipid components of plant membranes necessary for membrane sorting and trafficking processes (1). In mammalian cells and yeast, alteration of the amount of free sterols leads to defects in the endocytic pathway. The internalization of various plasma membrane (PM) proteins in animal cells engages clathrin-coated vesicles. Yet, comparatively little is known about clathrin-dependent endocytosis and its role during plant cytokinesis. The isolation of the sterol biosynthesis mutant \textit{cyclopropylsterol isomerase 1-1} (\textit{cpi1-1}) in \textit{Arabidopsis}, has provided a valuable tool for further understanding the role of plant sterols during endocytosis [2-4]. The \textit{cpi1-1} mutant displays a strong conversion of the sterol profile and almost exclusively accumulates cyclopropyl sterols on expense of the wild-type membrane sterols [2]. In this mutant, a mediator of cytokinetic vesicle fusion in the cell division plane, the syntaxin KNOLLE, is no longer constricted to the division plane but mis-localized to the lateral membrane upon fusion of the cell plate with the PM in late cytokinetic cells [3]. Treatment of wild type and \textit{cpi1-1} mutant roots with the endocytic recycling inhibitor brefeldin A revealed that KNOLLE internalization into endosomal membrane agglomerations compared to its level at the cell division plane and plasma membrane is affected in the \textit{cpi1-1} mutant. These findings suggested that KNOLLE is constrained to the cell division plane at the end of cytokinesis through sterol-dependent endocytosis. Consistent with this view, pharmacological interference with internalization mediated through clathrin-coated vesicles induced KNOLLE mis-localization at lateral membranes in late cytokinetic cells. Similarly, in \textit{dynamin-related protein1a} (\textit{drp1a}) mutants defective in a component associated with the clathrin machinery and affected in endocytosis, KNOLLE displayed lateral mis-localization. Therefore, KNOLLE appears to be constrained to the cell division plane at the end of cytokinesis through a sterol-dependent endocytosis mediated by clathrin and DRP1A [4]. Here, we present further biochemical evidence supporting that KNOLLE is affected in the \textit{cpi1-1} mutant and also that components of the clathrin mediated endocytosis are preferentially found in sterol-enriched membrane fractions. For this purpose, we carry out analysis of detergent-resistant membrane (DRMs) fractions. In addition we have performed immunolocalization experiments of DRP1A in roots of both wild type and the \textit{cpi1-1} mutant and initiated clonal loss-of-function mosaic analyses of the \textit{CPI1} gene.