Signaling pathway for resting cyst formation in the ciliated protozoan *Colpoda cucullus*

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SUMMARY

Resting cyst formation (encystment) of *Colpoda cucullus* is induced by an increase in external Ca^{2+} or overpopulation of *Colpoda* vegetative cells, and is suppressed by the addition of food to the external medium. The Ca^{2+}-mediated or overpopulation-mediated cyst formation was suppressed by calmodulin antagonists (W-7, trifluoperazine), 2'-deoxyadenosine (a P-site inhibitor of adenylate cyclase) and *R*p-cAMPS (a cAMP analog antagonist for cAMP-dependent proteins). In contrast, IBMX (a non-selective inhibitor of phosphodiesterase), cAMP or its membrane-permeable derivative (db-cAMP) had an encystment-inducing effect even in a Ca^{2+}-free medium. These results suggest that encystment induction in *Colpoda* may involve a rise in cAMP concentration through the activation of adenylate cyclase, and its activity is possibly regulated by Ca^{2+} /calmodulin (Ca^{2+}/CaM). *Colpoda* encystment was suppressed by the addition of D-glucose to the external medium. When D-glucose uptake was blocked by phlorizin, Ca^{2+}-mediated or overpopulation-mediated encystment was markedly suppressed. However, db-cAMP-mediated encystment was not suppressed by D-glucose. The results suggest that internalized D-glucose or its metabolic products have an effect upstream of cAMP production in the signaling pathway for induction of encystment.