세미나 초록

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발표 주제	The role of intracellular Ca^{2+} and phosphoinositide coupling for Metabolic diseases
발표 내용	Intracellular calcium (Ca^{2+})andphosphoinositides(PIPs)arecrucialforregulatingcellularactivitiessuchasmetabolismandcellsurvival.CellsmaintainpreciseintracellularCa^{2+}andPIPlevelsviatheactionsofacomplexsystemofCa^{2+}channels,transporters,Ca2+ATPases,andsignalingeffectors,includingspecificlipidkinases,phosphatases,andphospholipases.RecentresearchhasshedlightonthecomplexinterplaybetweenCa^{2+}andPIPsignaling,suggestingthatelevatedintracellularCa^{2+}levelsnegativelyregulatePIPsignalingbyinhibitingthemembranelocalizationofPIP-bindingproteinscarryingspecificdomains,suchasthepleckstrinhomology(PH)andCa^{2+}independentC2domains.Thisdysregulationisoftenassociatedwithcancerandmetabolicdiseases.PIPsrecruitvariousproteinswithPHdomainstotheplasmamembraneinresponsetogrowthhormones,whichactivatesignalingpathwaysregulatingmetabolism,cellsurvival,andgrowth.However,abnormalPIPsignalingincancercellstriggersconsistentmembranelocalizationandactivationofPIP-bindingproteins.Inthecontextofobesity,anexcessiveintracellularCa2+levelpreventsthemembranelocalizationofthePIP-bindingproteinsAKT,IRS1,andPLCδviaCa^{2+}-PIPs,contributingtoinsulinresistanceandothermetabolicdiseases.Furthermore,anexcessiveintracellularCa^{2+}levelcancausefunctionaldefectsinsubcellularorganellessuchastheendoplasmicreticulum(ER),lysosomes,andmitochondria,causingmetabolicdiseases.ThisreviewexploreshowintracellularCa^{2+}overloadnegativelyregulatesthemembranelocalizationofPIP-bindingproteins.