CONCLUSIONS

Our measurements of agonist-stimulated high-affinity GTPase activity and GTPγS binding in the developing rat brain cortex have revealed a markedly higher functional activity of GTP-binding proteins in adult (90-day-old) than in immaturate (12-day-old) rats. This study further demonstrates that RGS1, by contrast to RGS16, might function as strong regulator of high-affinity GTPase activity in this tissue.

AC activity is regulated similarly in brain cortex from immaturate and adult rats, but the enzyme activity is much lower in adult than in immaturate animals. As argued previously, the difference between AC activity in these two age groups is not explicable on the basis of the developmental expression profiles of either AC1, AC2, AC4 and AC6 or different G proteins (Ihnatovych et al., 2002a; Ihnatovych et al., 2002b). It might be speculated, however, that the complement of some other types of AC could be changed in adulthood. This supposition is supported by our recent finding of altered characteristics of [^{3}H]forskolin binding in cerebrocortical membranes from adult compared to immaturate rats (Stöhr et al., 2005b). Nevertheless, a possibility can not be ruled out that AC activity in adult rat brain cortex might be perhaps affected by some yet not known negative regulatory factor/mechanism, which is switched on shortly after maturation.

That high-affinity as well as super-high-affinity binding sites for [^{3}H]forskolin can be detected in rat brain cortex. In addition, we have found that parameters (K_D and B_{max}) of specific [^{3}H]forskolin binding differ quite significantly in cerebrocortical membranes from immaturate and adult rats. It might be assumed that the markedly different affinity of [^{3}H]forskolin binding sites in these two age groups, which certainly reflects altered ability of forskolin to interact with AC, is above all given by qualitative change(s) in the enzyme. There are some indications that forskolin need not bind to and activate all AC isoforms with the same rank of potency (Sutkowski et al., 1994). Our present finding of lower affinity of

binding sites for [^{3}H]forskolin and presumably lesser coupling efficiency of G_s protein to AC in adulthood may thus imply that the complement of AC isoforms in immaturate and adult rat brain cortex might be different. This speculation remains to be proved or disproved in future studies.