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## CORRIGENDUM

Tables 2, 4 and 5 published in J. Mol. Med. <u>73</u>:123–132 (1995) were composed in a manner allowing misinterpretation. Therefore, these tables are presented here in a structurally modified version.

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REVIEW

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## **Receptors and G proteins as primary components** of transmembrane signal transduction

## Part 2. G proteins: structure and function

**Table 2** Covalent modifica-<br/>tions of G protein  $\alpha$  subunits

<sup>1</sup> functional uncoupling of receptor-G-protein interaction <sup>2</sup> also modification of  $G\alpha_t$ by lauric acid and unsaturated fatty acids (C-14:2, C-14:1) <sup>3</sup> in platelets additionally acylation of  $G\alpha_i$ ,  $G\alpha_q$ ,  $G\alpha_z$ ,  $G\alpha_{13}$ by arachidonic acid <sup>4</sup> substoichiometric phosphorylation; unknown physiological

role <sup>5</sup> unknown physiological role <sup>6</sup> inhibition of pertussis toxin-

sensitive PI response

**Table 4** Posttranslational modifications of G protein γ subunits

<sup>1</sup> A = aliphatic amino acid,  $X \neq \Phi$ <sup>2</sup> abbreviations of amino acids: Ala = alanine, Cys = cysteine, Glu = glutamic acid, Leu = leucine, Met = methionine, Ser = serine <sup>3</sup> members of monomeric GTPases

ADP-ribosylation	
constitutively activated G protein	$\begin{array}{l} G\alpha_{s} \ (Arg^{186/201}), \ G\alpha_{t} \\ G\alpha_{i1-3}, \ G\alpha_{o1-2} \end{array}$
Pertussis toxin: inactivated $\alpha\beta\gamma$ heterotrimer <sup>1</sup> sequence motif: <i>C</i> GA $\Phi$ (C-terminus)	$G\alpha_{i1-3}, G\alpha_{o1-2}, G\alpha_t$
Acylation	
Myristoylation <sup>2</sup> (cotranslational, irreversible) sequence motif:MGXXXS/T? (N-terminus) enzyme: N-myristyl transferase	$G\alpha_{i13}, G\alpha_{o12}, G\alpha_t, G\alpha_z$
Palmitoylation <sup>3</sup> (posttranslational, reversible) sequence motif:MGC? (N-terminus)	$\begin{array}{l} G\alpha_{11-3}, G\alpha_z, G\alpha_{o1-2}, G\alpha_s, \\ G\alpha_{11-13}, \end{array}$
Phosphorylation	
cAMP-dependent protein kinase <sup>4</sup>	$G\alpha_i$ ?, $G\alpha_s$ ?
Protein kinase C <sup>5</sup>	$G\alpha_z$ , (Ser <sup>16/27</sup> ), $G\alpha_{i2}$
cGMP-dependent protein kinase <sup>6</sup>	$G\alpha_{o1-3}$

	(3) Cysteine carboxymethylation		<u> </u>
5	(2) Endoproteolytic cleavage of three C-terminal amino acids		
	CCXX; CXC	(c) geranyigeranyi mansterase m (c 20)	rab-family <sup>3</sup>
	CAAX: X = Leu	(c) geranylgeranyl transferase II (C-20)	$G\gamma_2, G\gamma_3, G\gamma_5$ ?, $G\gamma_7$ ?
ч :		(b) geranlygeranyl transferase I (C-20)	
Б	CAAX: $X = Ala^2$ , Cys. Glu. Met. Ser	(a) farmesyl transferase (C-13)	$G\gamma_1$ , ras-family <sup>3</sup>
	(1) Cysteine-polyisoprenylation	(x) for an equilation of each $(C, 15)$	
		sequence motif:CAAX <sup>1</sup> (C-terminus)	

Table 5 Effectors regulated by G protein  $\alpha$  subunits and  $\beta\gamma$  complexes

<sup>a</sup> recently it was shown that a PI-3 kinase (p110 $\gamma$ ) cloned and sequenced from a U 937 cDNA library is activated by both G protein  $\alpha$  and  $\beta\gamma$  subunits

*ref:* Stoyanov, B., Volinia, S., Hanck, T., Rubio, I., Loubtchenkov, M., Malek, D., Stoyanova, S., Vanhaesebroeck, B., Dhand, R., Nürnberg, B., Gierschik, P., Seedorf, K., Hsuan, J.J., Waterfield, M.D., Wetzker, R. (1995): Cloning and characterization of a G protein-activated human phosphatidylinositol-3 kinase, Science, in press

	α subunit	$\beta\gamma$ complex
cGMP phosphodiesterase βARK ras-regulating proteins PI-3 kinase-γ <sup>a</sup> Phospholipases C-β Adenylyl cyclases	↑ ↑ ↑,↓	↑ ↑ ↑,↓
Ca <sup>2+</sup> channels Cl <sup>-</sup> channels Na <sup>+</sup> channels K <sup>+</sup> ch. (inw. rect. ATP regul.)	↑,↓ ↑,↓ ↑,↓	↑