Neurochemical approaches to antidepressant effects and depressive disorder are also focusing on G-protein coupled receptors (GPCR) and subsequent signalling. Furthermore it is evident that immune system participates in antidepressant mode of action by neurotransmitter GPCR.

We studied the effect of acute administration of fluoxetine (specific serotonin reuptake inhibitor, SSRI) or non-selective adenosine agonist 5'-N-ethylcarboxamidoadenosine agonist NECA of adenosine receptor (AR) on C6 glioma cells or natural killer (NK) cell line, effectors of innate immunity. Acute fluoxetine or NECA agonist influence on decreased G alpha q/11 level of C6 glioma cells was observed. In contrast, no significant changes of G alpha s and G alpha i1,2 subunit levels were observed. Lowered G alpha q/11 signalling was in accordance with decreased 2nd messenger 1,4,5 IP3 formation by phospholipase C. Acute effect of fluoxetine or NECA agonist on NK cell line resulted also in significantly reduced G alpha q/11 levels without changes in G alpha s and G alpha i1,2. Furthermore, we determined that NECA agonist was able to abolish fluoxetine-evoked changes of G alpha q/11 levels of NK cell line. Thus similar inhibiton of G alpha q/11 by NECA agonist in both C6 glioma cells and NK cell line was determined. Furthermore dose-dependent NECA induced attenuation of fluoxetine evoked Galpha q/11 decrease can indicate parallel interference between GPCR and postreceptor signalling in functionally different cell types. Study was supported by grant of Academy of Sciences CR No. IAA 601680801.