

Figure 1. Effect of GABA esters 3-3HCl and fluphenazine and their respective equimolar doses of GABA esters 3-3HCl and 4-3HCl on plasma prolactin levels. Rats (four/group) were treated ip with perphenazine (5 mg/kg), fluphenazine (7.5 mg/kg), or the equimolar doses of their respective esters 3-3HCl and 4-3HCl. Blood from animals, under light ether anesthesia, was drawn by puncturing their orbital vein prior to treatment and 1 and 2 h post-treatment. Prolactin levels were determined using a double-antibody Rat Prolactin RIA kit (Biocode Belgium). Compared to the control and the level of prolactin at time 0, the tested treatments significantly increased prolactin level (**t* test $p < 0.05$).

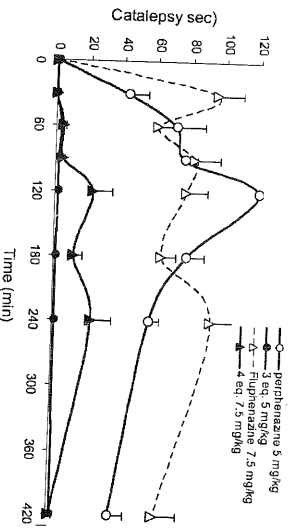


Figure 2. GABA esters 3-3HCl and 4-3HCl when administered ip to rats abolish or attenuate catalepsy. Wall descending test: Rats (six/group) were treated ip with perphenazine (5 mg/kg), fluphenazine (7.5 mg/kg) or equimolar doses of their respective esters 3-3HCl and 4-3HCl. Using the wall descending assay, the rats were monitored for catalepsy every 30 min and up to 420 min post treatment.

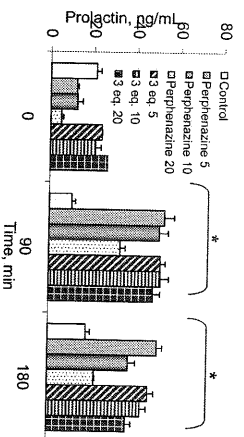


Figure 3. Plasma prolactin levels in rats treated po with perphenazine or 3-3HCl. Rats divided into 5 groups (4/group) were treated po with 1% lactic acid (vehicle control), perphenazine 5 10 and 20 mg/kg, and the respective equimolar doses 7 and 14 and 28 mg/kg of 3-3HCl. The prolactin level was determined prior-administration (time 0) and 90 and 180 min post-administration of the treatment. The prolactin determination was performed as described in Figure 1. Compared to the control and the level of prolactin at time 0, the tested treatments significantly (by *t* test) increased prolactin level (**p* < 0.05).

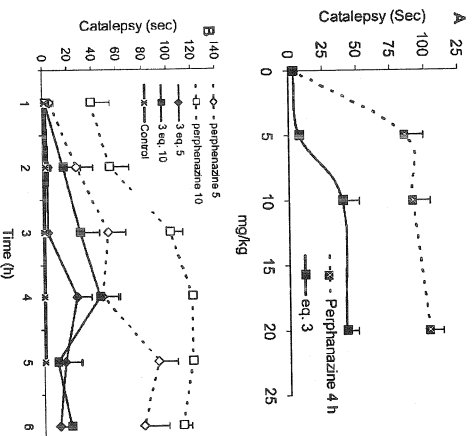


Figure 4. Catalepsy induced by orally administered 3·3HCl. Bar test: Rats were treated po with perphenazine (5, 10, and 20 mg/kg) and equimolar doses of 3·3HCl and catalepsy was determined 4 h after the treatment (A). The time course of catalepsy was determined in animals treated with 5 and 10 mg/kg perphenazine compared to the respective equimolar doses of 3·3HCl (B).

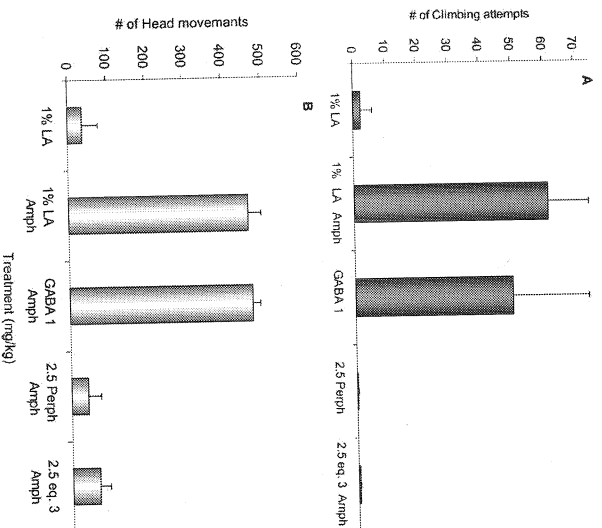


Figure 5. Perphenazine and 3·3HCl abrogate hyperactivity induced by D-amphetamine in rats. Male Wistar rats divided into five groups (six/group) were treated po respectively with: vehicle (1% lactic acid, two groups); perphenazine (2.5 mg/kg); an equimolar dose of 3·3HCl; and GABA (1 mg/kg). With the exception of one of the vehicle treated groups, which served as negative control, after 90 min all other animals received D-amphetamine (2.0 mg/kg, ip). The rats were then placed individually in barrels, and the number of head movements and climbing attempts on the barrel walls were recorded double-blindly.