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In order to identify novel components of the circadian system in *Arabidopsis thaliana*, we carried out a large-scale forward genetic screen. Several mutants, displaying altered rhythmic expression pattern of the *CAB2:LUC* reporter gene in continuous red light conditions, were isolated. The mutant ct12 (circadian time 12) showed about 2 h period shortening under the screening conditions and was selected for further analysis. The mutation affected the expression of several clock-controlled genes in the same manner and the short period phenotype was independent of the light conditions. These findings indicated that the function of the core oscillator was altered in the mutant. In fact, expression of the core clock genes showed the expected short period phenotype, but the level of their expression was not affected significantly. This suggests that CT12 does not affect transcription of clock components directly. Consistent with the basic circadian dysfunction, ct12 showed early flowering phenotype in short day conditions. We have provided experimental evidences that the flowering phenotype of the mutant is caused by the altered circadian period/phase. Moreover, ct12 mutants produce long hypocotyls in red but not in blue light, suggesting a positive role for CT12 in light responses mediated by the red/far-red light absorbing phytochrome photoreceptors.

Genetic mapping followed by transgenic complementation showed that the *CT12* gene encodes a putative acyl-transferase. Although the exact biochemical function of this protein and the way it affects the function of the clock remains to be elucidated, we hypothesize that CT12 could represent a link between the clock and certain metabolic processes.

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The effect of recreational physical exercise on inflammatory markers in a rat model of colitis

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The sedentary lifestyle can lead to health problems such as metabolic syndrome including obesity with hypertension, insulin resistance and high blood lipid levels. Metabolic syndrome is associated with a chronic low-grade inflammatory state and oxidative stress. Many studies reported that physical activity is an effective way of controlling body weight, but the influence of long term low intensity exercise on inflammation and activity of anti- and proinflammatoric enzymes is not well known. Heme oxygenase-1 (HO-1), which is the inducible isoform of heme oxygenase enzyme (HO), is thought to play an important role in the protection of tissues from oxidative injuries. Another enzyme involved in oxidative stress and inflammation is nitric monoxide synthase enzyme (NOS) with 3 isoforms: the inducible (iNOS) and the two constitutively expressed (cNOS) isoforms namely neuronal NOS (nNOS) and endothelial NOS (eNOS). Nitric monoxide (NO) produced in different amount by the three NOS isoforms can be both harmful and beneficial. We used a rat model, trinitrobenzene-sulphonic acid (TNBS) induced colitis, to investigate the changes of inflammation and activity of HO and NOS enzymes in the colon after running.

We investigated the effects of long-term leisure-type physical exercise on the activity of HO, NOS and myeloperoxidase (MPO, an inflammatory marker) enzymes in the trinitrobenzene-sulphonic acid (TNBS) induced colitis in rats in dependence on time.

After 3, and 6 weeks self-administered physical activity (running wheel) male Wistar rats were treated with TNBS (10 mg). After 72 h TNBS challenge we measured colonic inflammatory parameters and HO, iNOS, cNOS, MPO activity.

While after 3-week running we found no difference in the severity and extent of colonic inflammation in the sedentary and running TNBS treated group, the 6-week freewheel running significantly increased the activity of HO (from $1,3\pm0,2$ to $2,8\pm0,3$ nmol bilirubin/h/mg protein), constitutive NOS isoforms (from $321,1\pm35,2$ to $438,0\pm30,1$ pmol/min/mg protein). The TNBS challenge after 6 weeks running significantly decreased the level of inflammatory markers including extent of lesions (from $54,6\pm2,6\%$ to $42,9\pm3,2\%$), severity of mucosal damage (from $7,6\pm0,3$ to $6,6\pm0,3$) and the level of MPO activity (from $880,6\pm79,3$ to $568,4\pm59,9$ mU/mg protein), increased the activity of cNOS (from $108,9\pm25,6$ to $333,9\pm32,3$ pmol/min/mg protein) decreased the iNOS activity (from $217,6\pm26,4$ to $128,9\pm15,8$ pmol/min/mg protein), but did not changed the activity of HO compared to the sedentary TNBS-treated group.

Long lasting recreational physical activity, at least 6 weeks by rats, improves body's defence mechanisms. Physical activity-induced increasing activation of HO and cNOS systems, decreased activation of iNOS system may play role of these mechanisms including colonic inflammation.

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