

Environmental temperature and the growth of *Taenia crassiceps* cysticerci in mice

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Summary. Mice kept at low ($5 \pm 1^\circ\text{C}$) and high ($35 \pm 1^\circ\text{C}$) temperature harboured significantly less *Taenia crassiceps* cysticerci than controls kept at $21 \pm 1^\circ\text{C}$. This effect was more pronounced in heat-stressed than in cold-stressed animals and more in males than in females.

Webster mice of both sexes, 4-month-old, were divided into 3 groups of 15 mice each and acclimated to their respective environmental temperatures 7 days prior to infection. One group was acclimated to $5 \pm 1^\circ\text{C}$, and the other to $35 \pm 1^\circ\text{C}$. The control group was kept at $21 \pm 1^\circ\text{C}$. With the aid of electric timers, each group was placed on the same photoperiod of 15 h daylight and 9 h darkness. All mice were infected with 20 nonbudding cysticerci each and killed 20 days later. At autopsy the number of nonbudding individuals, budding individuals, and the total numbers of larvae were counted.

2 experiments were performed; in both the cold-stressed and heat-stressed mice of both sexes harboured significantly less larvae than the controls. Low temperature decreased the total number of larvae from 42.06 ± 4.24 to 20.93 ± 1.26 and 32.40 ± 2.45 to 15.60 ± 0.66 in females, and from 28.60 ± 4.53 to 19.86 ± 1.49 and 26.71 ± 2.04 to 16.36 ± 1.53 in males. High temperature depressed the number of larvae from 42.06 ± 4.24 to 24.13 ± 1.78 and 32.40 ± 2.45 to 16.00 ± 0.92 in females and from 28.60 ± 4.53 to 14.40 ± 1.04 and 26.71 ± 2.04 to 8.14 ± 1.04 in males. The mortality of larvae (with subsequent disintegration) was highest in heat-stressed males. In both experiments heat-stressed males had significantly less larvae than cold-stressed males. But there was no significant difference in the total number of larvae recovered from respective female groups.

All temperature-stressed mice had significantly lower numbers of nonbudding larvae than controls. In cold-stressed mice their number decreased from 28.93 ± 3.14 to 9.33 ± 0.81 and from 12.93 ± 2.24 to 2.20 ± 0.57 in females and from 23.07 ± 3.85 to 7.60 ± 0.88 and from 14.86 ± 1.86 to 2.00 ± 0.58 in males. In heat-stressed mice the mean number of non-

budding cysticerci decreased from 28.93 ± 3.14 to 11.73 ± 1.21 and from 12.93 ± 2.24 to 3.13 ± 0.41 in females and from 23.07 ± 3.85 to 11.07 ± 0.90 and from 14.86 ± 1.86 to 4.36 ± 0.50 in males. However the cold-stressed males harboured in both experiments significantly less nonbudding larvae than heat-stressed males.

In cold-stressed males of the 1st experiment the number of budders increased significantly from 5.53 ± 0.82 in controls to 12.33 ± 1.47 , but remained more or less constant in the 2nd experiment (11.86 ± 0.78 and 14.36 ± 1.18 respectively). There was no significant difference between the number of budders in cold-stressed and control females in the 1st experiment (13.13 ± 1.42 and 11.53 ± 1.46 respectively), but there was a significant decrease in this number in the 2nd experiment (19.47 ± 1.23 and 13.40 ± 0.65 respectively). High temperature decreased significantly the number of budders in males in both experiments, from 5.53 ± 0.82 to 3.33 ± 0.48 and from 11.86 ± 0.78 to 3.79 ± 1.06 respectively. Also in both experiments heat-stressed males had significantly less budding larvae than cold-stressed males. The number of budders in heat-stressed females was close to that in cold-stressed females. It remained almost constant in the 1st experiment (13.13 ± 1.42 in controls and 12.40 ± 0.83 in heat-stressed mice) but was significantly lower in the 2nd experiment 19.47 ± 1.23 and 12.87 ± 0.74 respectively. The present study, which seems to be the first dealing with the effect of environmental temperature on proliferating cestode larvae in homeothermic hosts, showed that this temperature affects the growth of *T. crassiceps* cysticerci in mice. Similar studies with other proliferating cestode larvae are currently in progress.

Demonstration of the presence of M-creatine kinase in mammalian myogenic cell lines

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Summary. Unequivocal identification of M-CK in cell extracts from fused cells of myogenic cell lines is difficult due to almost identical behaviour of the M-CK and a contaminating enzyme activity in electrophoresis. If CK dimers present in cell extracts were subjected to dissociation and reassociation in the presence of exogenous B-CK subunits, the formation of easily identifiable MB-CK was demonstrated, indicating the presence of M-CK in the myogenic rat cell lines.

The transition of B-creatine kinase (B-CK) to muscle-specific M-creatine kinase (M-CK) is a well documented event during terminal differentiation of myogenic cells in the embryo^{2,3} or in culture^{3,4}. Terminally differentiated myotubes, derived from presumptive skeletal muscle contain, among other typical muscle proteins, also M-CK, which has proved to be a useful marker to evaluate the differentiated state of myogenic cells⁴⁻⁶. In mammalian myogenic cell lines, the determination of the creatine kinase isoenzymes has been hampered by the fact that creatine kinase activity and some contaminating enzyme,

probably myokinase, giving rise to high background staining, cannot be separated easily from each other by electrophoresis.

So far no B-CK has been found in these cell lines, and thus even if the M-CK gene were active one could not hope to find any hybrid enzyme MB-CK, an activity that clearly can be separated from the unspecific staining by electrophoresis. The presence of MB-CK would indicate both the M-CK and B-CK genes to be active within the same cytoplasm and give some further information on the state of differentiation of such cells. As there is no BB-CK