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Biology
The protein secreted by HAT medium and does not grow in HAT medium on the same mouse strain is expressed by cells that are selected for their expression of HAT medium. The expression of HAT medium in the absence of HAT medium is not detected in the secreted proteins. The expression of HAT medium in the absence of HAT medium is not detected in the secreted proteins.

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malian organs might argue against the possibility that thymus muscle clones are derived from pre-existing committed precursor cells.

Thus, we are left with the third possibility, namely, differentiation of thymus muscle clones from pluripotent stem cells. Several arguments seem to favour this possibility. It has been shown that, apart from muscle, tissues as different as osteocytes and chondrocytes (D. Yaffe, personal communication) can differentiate from thymus cells in particular conditions in vitro. In our cultures, the relatively long latency period of 8 d preceding muscle cell fusion could be due to differentiation processes of originally pluripotent stem cells. This is well in line with the strikingly similar behaviour of mouse teratoma cells. Undifferentiated OTT 6050 embryoid body cells differentiate into muscle tubes when they are cultured in very similar conditions to our thymus cells.

The relationship of thymic cells and neoplastic pluripotent stem cells may be more than coincidental. It should be noted that the anterior mediastinal space shows the highest rate of teratomas, next to the gonads, and that these teratomas are thought to originate from thymic tissue. It is tempting to speculate that mediastinal teratomas arise from neoplastic transformation of pluripotent stem cells in the thymus. Such stem cells, accumulated in this organ, may have a physiological role in the regeneration of the body's tissues. Experimental studies of congenital aplasias, for example in nude mice, should yield further information.

A final, very obvious implication could concern the correlation of thymic lesions and muscle autoimmune diseases like myasthenia gravis in particular. As we demonstrated before, thymocytes can be in vitro sensitised against autochthonous thymus reticulum cells. It seems possible, therefore, that, following a defect of control, thymus lymphocytes could be autosensitised against a thymus component bearing muscle antigens, and that such autosensitised lymphocytes cause the pathological lesions leading to myasthenia. Whether or not this is the case, our culture system provides the means to approach this problem experimentally.

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Fig. 1 Thymus reticulum-derived myotubes at varying magnifications. a. Low magnification (× 32) of a fixed muscle clone containing spindle-shaped myoblasts as well as fused myotubes (day 12, May-Grünwald-Giemsa stain). b. Phase contrast microscopy of a living thymus muscle culture (day 10, magnification × 130). c. Electron microscopy of a myotube (MT) containing myofibrils (F), triad formation (→), Z- and M-bands (day 10, magnification × 8795, stained with uranyl acetate and lead citrate).