

by hibernaculum spinning. Between the 2 development patterns i.e. no interruption and prolonged development-diapause one can produce, with proper manipulation of temperature and photoperiod, a situation of delayed metamorphosis characterized by an immaculate morph, stationary molting but no spinning. For such an intermediate development situation to occur in the whole population, the insect must spend at least 32 days at 12:12, 21°C and then experience conditions that do

not induce arrested development (14:10, 27°C). This also indicates that the fifth instar is quite sensitive to photoperiod/temperature conditions. In conclusion then *D. saccharalis* is capable of 1) averting diapause; 2) entering a period of delayed metamorphosis, and 3) entering diapause but the latter cannot be considered to start with the appearance of the immaculate morph according to the generally accepted definition of diapause. It is really initiated at hibernaculum spinning.

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Oxygen consumption in rat skeletal muscle at various rates of oxygen delivery

F. Kolář and L. Janský

Department of Comparative Physiology, Faculty of Sciences, Charles University, CS-12844 Prague (CSSR), 19 April 1983

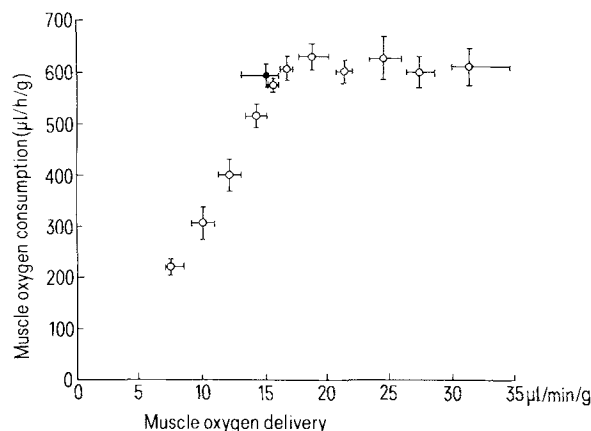
Summary. Resting oxygen consumption of the vascularly isolated gracilis anticus muscle of the rat perfused by natural circulation via the femoral artery with diluted or undiluted blood depends on oxygen delivery (the product of flow rate and arterial oxygen concentration - $\dot{V}O_2$) only when $\dot{V}O_2$ falls below 16 $\mu\text{l}/\text{min}/\text{g}$.

The role of blood flow (\dot{Q}) as a determinant of the resting muscle metabolic rate ($\dot{V}O_2$) has been studied on many experimental objects, using different techniques. The values of \dot{Q} in rat skeletal muscles at which $\dot{V}O_2$ was measured have varied between 2.0 and 432.5 $\mu\text{l}/\text{min}/\text{g}$ ^{1,2}. The dependence of $\dot{V}O_2$ on muscle perfusion rate has been clearly demonstrated^{1,3,4}, but the mechanism responsible for this effect has not yet been clarified. The aim of the present work was to evaluate the role of \dot{Q} and $\dot{V}O_2$ as determinants of the resting $\dot{V}O_2$ and to find out the range in which this control takes place. In order to induce different \dot{Q} and $\dot{V}O_2$ values of the gracilis muscle preparation an isovolemic hemodilution was applied and $\dot{V}O_2$ was measured under stationary conditions.

Materials and methods. Male Wistar SPF rats, 355-430 g b.wt, anesthetized (Thiopental Spofa, 50 mg/kg, i.p.) and heparinized (Heparin Spofa, 2500 IU/kg, i.v.) were used for the experiments. Before the experiments, rats were kept at $6 \pm 1^\circ\text{C}$ for at least 21 days in order to obtain cold-adapted individuals. These animals are generally used to study the adrenergic control of muscle nonshivering thermogenesis in our laboratory. The left gracilis anticus muscle was completely isolated vascularly, with the exception of the femoral artery through which the muscle was perfused with blood from natural circulation. Venous blood leaving the muscle was returned by a cannula from the left femoral vein into the right jugular vein. The muscle surface was kept moist with a Krebs-Henseleit solution at $36-37^\circ\text{C}$. The diluting solution (6% dextran Spofa) was injected into the left jugular vein at the rate of 1 ml/min and the corresponding volume of arterial blood was taken out simultaneously from the right femoral artery. \dot{Q} through the muscle was measured by timed collection of femoral vein effluent in calibrated capillary tubes. $\dot{V}O_2$ was calculated from \dot{Q} and the arteriovenous difference in O_2 concentration according to the Fick principle. Samples of

arterial blood were taken from the right femoral artery. Blood O_2 concentration was analyzed using a Clark O_2 electrode (Radiometer Copenhagen) in the 0.2% ferricyanide solution saturated with nitrogen. Muscle $\dot{V}O_2$ was calculated as the product of \dot{Q} and arterial O_2 concentration. The O_2 extraction coefficient was taken as the ratio of arteriovenous difference in O_2 concentration to arterial O_2 concentration.

Results. Mean resting \dot{Q} in the muscle perfused with undiluted arterial blood containing 23.4 ± 1.1 ml $\text{O}_2/100$ ml was 64.0 ± 3.6 $\mu\text{l}/\text{min}/\text{g}$. Both parameters were kept un-



The relationship between oxygen delivery and oxygen consumption in the vascularly isolated muscle after isovolemic hemodilution. Closed circle represents the mean resting oxygen consumption of the muscle perfused with undiluted blood. Vertical and horizontal bars indicate SEM and variation of the argument, respectively. $n=8-12$.

changed during an experiment lasting at least 1 h. Under these conditions muscle $\dot{V}O_2$ was 14.9 ± 0.5 $\mu\text{l}/\text{min}/\text{g}$ and the resting $\dot{V}O_2$ was 594.5 ± 21.0 $\mu\text{l}/\text{h}/\text{g}$. The hematocrit value for undiluted blood was $60.4 \pm 2.1\%$. Various dilutions obtained by substitution of the dextran solution for blood resulted in hematocrit values between 44.4 and 18.0%. Under these conditions the perfusion flow rate spontaneously increased up to a maximum of 208.3 $\mu\text{l}/\text{min}/\text{g}$. Depending on the extent of hemodilution, $\dot{V}O_2$ either increased (moderate hemodilution) or decreased (severe hemodilution) with respect to its initial value, within the range 7.0–35.4 $\mu\text{l}/\text{min}/\text{g}$. \dot{Q} and arterial O_2 concentration were respectively 202.3 $\mu\text{l}/\text{min}/\text{g}$ and 17.5 ml $O_2/100$ ml at maximum $\dot{V}O_2$, and 120.7 $\mu\text{l}/\text{min}/\text{g}$ and 5.8 ml $O_2/100$ ml at minimum $\dot{V}O_2$.

A distinct relationship was found between $\dot{V}O_2$ and $\dot{V}O_2$ (fig.). $\dot{V}O_2$ increased proportionally to $\dot{V}O_2$ in the range of 7.0 to about 16 $\mu\text{l}/\text{min}/\text{g}$. The O_2 extraction coefficient remained submaximum in this $\dot{V}O_2$ range (which is in accordance with the ability of noradrenaline to increase it while stimulating $\dot{V}O_2$ ^{1,3}). The rise of $\dot{V}O_2$ above the level of 16 $\mu\text{l}/\text{min}/\text{g}$ did not, in itself, increase muscle respiration. Thus, at $\dot{V}O_2$ values beyond 16 $\mu\text{l}/\text{min}/\text{g}$ and up to 35.4 $\mu\text{l}/\text{min}/\text{g}$ the O_2 extraction coefficient decreased from 0.617 to 0.314.

$\dot{V}O_2$ was not found to be a simple function of \dot{Q} , since at the same \dot{Q} , different $\dot{V}O_2$ were achieved when the muscle was supplied with different amounts of O_2 . Conversely, to any given $\dot{V}O_2$ value corresponded different \dot{Q} values: for example, at the $\dot{V}O_2$ corresponding to $\dot{V}O_2 = 15$ $\mu\text{l}/\text{min}/\text{g}$, \dot{Q} varied from about 65 to about 160 $\mu\text{l}/\text{min}/\text{g}$, the last value being obtained at 25% hematocrit level.

Discussion. Previous studies indicated that $\dot{V}O_2$ of the rat skeletal muscle may change according to changes in \dot{Q} . Our results show, however, that rather than the \dot{Q} , the product of \dot{Q} and arterial O_2 concentration (i.e. O_2 delivery – $\dot{V}O_2$) is the decisive factor determining $\dot{V}O_2$ in resting skeletal muscles, because the same $\dot{V}O_2$ may be obtained at different \dot{Q} , depending on arterial O_2 concentration. This is true, however, only when $\dot{V}O_2$ to the muscle is less than 16 $\mu\text{l}/\text{min}/\text{g}$. Above this value the muscle $\dot{V}O_2$ becomes independent on $\dot{V}O_2$.

This finding is in contrast with the data reported by Honig et al.⁵, who observed that after denervation the rat muscle $\dot{V}O_2$ increased with the increase of $\dot{V}O_2$ even above 16 $\mu\text{l}/\text{min}/\text{g}$. These results have been interpreted as a physiological limitation of resting muscle $\dot{V}O_2$ by O_2 transport, due to the inhomogeneity of capillary function and uneven distribution of O_2 within the organ. At present it cannot be

decided whether or not such a limitation really takes place in the normal resting muscle, because methods for measuring the heterogeneity of O_2 distribution to muscle cells are still inadequate⁶. In any case, our data show that the mean $\dot{V}O_2$ to the innervated, vascularly isolated muscle, perfused physiologically by undiluted blood achieves the value above which the $\dot{V}O_2$ ceases to further control the muscle $\dot{V}O_2$ (fig.).

The objection still exists that the vascularly isolated muscle may have more open capillaries than the intact resting muscle and thus it may have a larger $\dot{V}O_2$, because it is known that \dot{Q} and functional capillary density are controlled independently⁷. This does not seem to be the case in our experiments, however; our unpublished data demonstrate that the vascular isolation used in this work does not change functional capillary density and capillary spatial distribution in the gracilis muscle (271.1 ± 25.0 and 265.7 ± 17.8 capillaries/ mm^2 in intact and isolated muscle, respectively).

In conclusion, we assume that the metabolic rate of the resting rat skeletal muscle is not limited by O_2 transport in the range corresponding to physiological values, like in the dog^{5,8,9} and the cat¹⁰. Adams et al.¹¹ came to the same conclusion. They observed no change of whole body $\dot{V}O_2$ of the rat when the total O_2 transport was increased above the normal level.

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Sensitivity of *Botrytis cinerea* Pers. to elemental sulfur in the presence of surfactants

R. Pezet and V. Pont¹

Swiss Federal Agricultural Research Station of Changins, CH-1260 Nyon (Switzerland), 2 May 1983

Summary. The sensitivity of *Botrytis cinerea* Pers. to elemental sulfur is enhanced in surfactant solutions (Triton X-100 or SDS). The role of surfactants in the change of permeability to sulfur is discussed.

Botrytis cinerea Pers. is a major grape pathogen in Swiss vineyards. Growers know that it is insensitive to elemental sulfur, and this fungicide has never been used to control the Grey Mould of grapes.

Investigations on the metabolism of endogenous and ex-

ogenous elemental sulfur in *Phomopsis viticola* Sacc.^{2,3} and *Monilia fructicola*⁴ showed that this product interferes with some vital biochemical pathways, such as cell respiration and the resulting oxidative phosphorylation. Thus we can postulate that the sensitivity to elemental sulfur is correlat-