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# SCHILDDRÜSENHORMONWIRKUNG IM RIBOFLAVINMANGEL

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Seit den Arbeiten von *Lee* und *Lardy* wird der m-Glycerophosphat-Dehydrogenase (m-GDH), einem Flavoenzym des Glycerin-1P-Cyclus, bei der schilddrüsenhormoninduzierten Erhöhung der basalen Stoffwechselrate eine besondere Bedeutung beigemessen. Im Gegensatz zu diesen Befunden an der Ratte fand sich jedoch weder beim Menschen noch beim Meerschweinchen ein $T_3$-induzierbarer Anstieg der m-GDH. (1). In der vorliegenden Arbeit soll versucht werden festzustellen, inwieweit eine $T_3$-induzierte Erhöhung der basalen Stoffwechselrate bei der Ratte vom Anstieg der m-GDH abhängig ist.

120 g schwere Ratten wurden 82 Tage riboflavin-mangel-ernährt und anschließend mit und ohne Riboflavin (rf)-Substitution hyperthyreot gemacht. In den verschiedenen Versuchsstadien wurde der Grundumsatz bestimmt. In Leber, Herz, m. rectus, m. soleus und Gehirn wurden Flavoenzyme wie m-GDH, Flavinstoffwechselenzyme (Flavokinase) und Enzyme der Glykolyse, Citratcyclus und Fettsäureoxydation gemessen.

Die basale Stoffwechselrate sowie alle Flavoenzyme fielen im rf-Mangel ab, z. T. sehr organunterschiedlich. Nach 4tägiger rf-Substitution erreichten Aktivitäten der Flavoenzyme die der Kontrollen. Nach $T_3$-Gabe stiegen die basalen Stoffwechselraten der rf-Mangeltiere auf die der hyperthyreoten Kontrolltiere an, während die Aktivitäten der m-GDH nur $^1/_3$—$^1/_5$ der hyperthyreoten Kontrollen erreichte. Auch hier fanden sich deutliche Organunterschiede. Es wird angenommen, daß auch bei der Ratte der extra-intramitochondriale $H_2$-Transfer durch den Glycerin-1P-Cyclus nicht die alleinige Voraussetzung für den erhöhten basalen Stoffwechsel darstellt, sondern daß auch andere $H_2$-Transfer-Cyclen, wie der Malat-Shuttle, hierfür in Frage kommen. Auf Veränderungen der $T_3$-$T_4$-Serumspiegel im rf-Mangel wird eingegangen.

1. *Nolte, J., Pette, D., Bachmaier, B., Kiefhaber, P., Schneider, H.* und *Scriba, P. C.:* Europ. J. Clin. Invest. Nr. 3, 1972. Enzyme response to thyrotoxicosis and hypothyroidism in human liver and muscle: Comparative aspects.