Prelimary Notes

PN 10022

Occurrence of transamidinase in decidua and its repression by dietary creatine

Model systems for the study of negative-feedback control of enzyme levels in intact higher animals are limited in number. From the experimental, if not conceptual, standpoint, the simplest known system appears to be the lowering of glycine transamidinase (L-arginine: glycine amidinotransferase, EC 2.6.2.1) activity by dietary creatine. The creatine effect, first reported for rat-kidney transamidinase[1], has been operationally termed end-product repression, by analogy with bacterial systems, until its exact mechanism can be elucidated. Study of the creatine-transamidinase model system has subsequently been extended to embryonic tissues of the chick[2], in an effort to relate this process to the establishment and maintenance of tissue-specific enzyme patterns. Recently SHELESNYAK[3] has suggested that the process of decidualization can serve as a model system for study of cell growth and differentiation in mammals, under experimentally controllable conditions. The purpose of this investigation is to explore the possibility of merging these two model systems.

It was found that decidual tissues from pregnant rats have transamidinase activity higher than that of any other rat tissue, equivalent to 27 μmoles of hydroxyguanidine formed from arginine and NH_2OH per h per g tissue (wet wt.). This can be compared with activities of 14 for rat pancreas, 6.7 for female-rat kidney, and 11 for male-rat kidney[1,2]. Little or no transamidinase was found in the spongy tissue or labyrinth of fetal placenta, which is lost at birth, or in portions of uteri not having decidual tissue. Residual decidual tissue walled off after birth still contains significant transamidinase activity for a number of days *post partum*. Decidual transamidinase activity is not dependent upon the presence of a fetus, since deciduomas induced in pseudopregnant virgin rats[3] also contain the enzyme. Other experiments have shown that rat-decidual tissue, like kidney but unlike pancreas, lacks guani-

TABLE I

REPRESSION OF TRANSAMIDINASE IN VARIOUS RAT TISSUES BY DIETARY CREATINE

Pregnant rats were divided into 2 groups. 1 group (7 rats) was fed laboratory chow, while the other group (9 rats) was fed chow containing 5% creatine, from the 6th–16th day of pregnancy. Tissues were harvested on the 16th day and assayed for transamidinase[1].

Diet	*Transamidinase activity*[*]		
	Decidua	*Pancreas*	*Kidney*
Control	26.5 ± 3.5[**]	14.1 ± 1.8	6.7 ± 1.2
5% creatine	16.7 ± 2.7	4.2 ± 0.9	1.86 ± 0.24

[*] μmoles of hydroxyguanidine formed from arginine and NH_2OH per h per g tissue (wet wt.).
[**] Standard deviation.

dinoacetate methyltransferase (EC 2.1.1.2) activity. These enzymes, which have a restricted distribution, may well serve as markers for future studies on the problem of the origin of decidual tissue[3]. As for related tissues, we have observed transamidinase activity for the first time in commercial beef-placenta preparations and chick-egg yolk sac, but not in human term placenta.

The presence of significant levels of transamidinase in each of three tissues of a single animal offers the possibility of comparing the relative repressibility of transamidinase in these tissues. Table I shows that dietary creatine represses transamidinase in rat kidney and pancreas to the same relative extent, whereas decidual transamidinase is repressed to a significant, but lesser, extent. Whether this difference in repressibility reflects a difference in permeability to creatine, or other factors, is not known.

It would appear that the creatine-transamidinase and deciduoma model systems can indeed be combined, affording an opportunity to study enzyme repression, hormone effects[3,5], and differentiation[3,6] in the same non-embryonic mammalian tissue.

This work was supported by research grants from the Robert A. Welch Foundation, Houston, Texas, and the U.S. Public Health Service.

Department of Biochemistry,
Baylor University College of Medicine,
Houston, Texas (U.S.A.)

JAMES B. WALKER*
WILLIAM T. GIPSON

1 J. B. WALKER, *Biochim. Biophys. Acta*, 36 (1959) 571.
2 M. S. WALKER AND J. B. WALKER, *J. Biol. Chem.*, 237 (1962) 473.
3 M. C. SHELESNYAK, *Perspectives Biol. Med.*, 5 (1962) 503.
4 J. B. WALKER, *Federation Proc.*, 19 (1960) 52.
5 W. E. KNOX, in D. RUDNICK, *Synthesis of Molecular and Cellular Structure*, Ronald Press, New York, 1961, p. 13.
6 J. MONOD AND F. JACOB, *Cold Spring Harbor Symp. Quant. Biol.*, 26 (1961) 389.

Received October 26th, 1962

* Research Career Development Awardee of U.S. Public Health Service.

PN 10024

A soluble c-type cytochrome from anaerobically grown *Escherichia coli* and various Enterobacteriaceae

It is generally accepted that *Escherichia coli* and related facultative anaerobes lack cytochromes *c* and *b*, but contain substantial amounts of cytochrome b_1[1]. It is the purpose of this note to show that a soluble cytochrome *c* consistently is formed when bacteria of this group are cultivated anaerobically on a synthetic medium containing glucose as the principal carbon source. This finding is an outgrowth of comprehensive studies being carried out in this laboratory on the enzymic and chemical constitution of *E. coli* cells grown on complex and synthetic media under both aerobic and anaerobic conditions. An important feature of these studies is the characterization of enzymes as either membrane bound or soluble. In order to make