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24. Several observations strongly suggest that the behavioral changes seen after SN grafts are not secondary to tissue degeneration or some breakdown product. First, in a recent histochemical study of transplants in situ for 8 to 9 months, we have found little change in number or appearance of DA-containing cells or fiber proliferation from the 2- to 3-month-old grafts studied here. Second, we have seen no evidence of fibrous astrocytosis, abnormal lipofuscin accumulation, or reactive macrophages in the transplanted tissue or host caudate in either graft age group. Third, when SN is grafted into the anterior chamber of the eye, there is long-term growth, survival, and fiber proliferation with little evidence of toxicity (*21*). Finally, although there is considerably more tissue breakdown in the sciatic nerve grafts, no significant behavioral changes are seen. Taken together, these observations suggest tissue breakdown or toxic products in the SN grafts are not significant factors in the behavioral change. Proof that this change is due to a dopaminergic input, however, requires further experimentation.
25. The adrenal medulla of adult rats survives transplantation and the catecholamine-containing cells elaborate neuronal-like processes which invade the host tissue [L. Olson, *Histochemie* **22**, 1 (1970)].
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Human Serum Fucosyltransferase and Tumor Therapy

Bauer *et al.* have described an elevation in plasma fucosyltransferase activity in plasma of patients with neoplastic disease (*1*). Three assays were performed: transfer of fucose onto endogenous plasma acceptors, transfer of fucose onto the 2′-position of the terminal galactose of desialated fetuin (α_2-fucosyltransferase), and transfer of fucose onto the 3′-position of the terminal *N*-acetylglucosamine of desialodegalactofetuin (α_3-fucosyltransferase). While the data cannot be disputed, it appears that the enzymatic activities they actually measured are different from those that they described.

An acceptor with terminal galactose and subterminal *N*-acetylglucosamine such as desialated fetuin can accept fucose in two positions: the 2′-site on the terminal galactose and the 3′-unsubstituted position on the subterminal *N*-acetylglucosamine (*2*). Only with a different acceptor such as phenyl-β-galactoside (*3*) can α_2-fucosyltransferase be measured unambiguously. The use of an acceptor with a terminal *N*-acetylglucosamine appears to measure transfer of fucose onto an internal asparagine-linked *N*-acetylglucosamine (*4*), not onto the 3′-position of the terminal *N*-acetylglucosamine. Watkins does mention transfer onto the 3′-position in the summary of (*2*), but in the text and in (*5*), it is clear that the author means an *N*-acetylglucosamine residue subterminal to galactose. What Bauer *et al.* call α_2-fucosyltransferase is therefore a mixture of α_2- and α_3-fucosyltransferases, while the so-called α_3-fucosyltransferase is something else.

Since plasma may contain endogenous acceptors for several fucosyltransferases, it seems unlikely that the activity of any individual enzyme can be deduced by subtraction of an "endogenous" level of activity from results obtained by addition of an exogenous acceptor. Furthermore, we have shown (*6*) utilizing *N*-acetylglucosamine terminal acceptors that the plasma level of fucosyltransferase rises markedly during bone marrow hyperplasia after chemotherapy. A specific inhibitor of endogenous activity of other plasma fucosyltransferases was used in the latter study. In order to delineate fucosyltransferase elevation related to neoplasia from an elevation related to regeneration of a normal marrow population after drug therapy, it is therefore necessary to know your acceptor.

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Kessel's comment (*1*) on our report (*2*) was surprising since he and his co-workers use, in principle, the same assay system as we (*3*).

The hydrogen ion–dependent fucosyltransferase adds fucose primarily to the terminal galactose residues of both glycoproteins (*4*) and glycolipids (*5*) by forming (1 → 2) linkages. Only when using a low molecular weight acceptor such as *N*-acetyllactosamine are considerable amounts also transferred to *N*-acetylglucosamine (*6*). Though *N*-ethylmaleimide (NEM) preferentially, not exclusively, inhibits α_2-fucosyltransferase, α_3-fucosyltransferase can be affected as well. It has been recently reported (*7*) that in a patient with leukemia, the activity of α_3-fucosyltransferase was inhibited by 55 percent in the presence of 3.3 m*M* NEM. The determinations are further complicated by our observations that (i) NEM can be less effective when inhibiting serum α_2-fucosyltransferase of patients with recurrent malignancy, and (ii) a fucosyltransferase with different characteristics probably occurs in the serum of tumor patients (*8*). Thus, that addition of NEM is suitable when differentiating unequivocally between the two major human fucosyltransferases is probably restricted to normal subjects and certain cases of neoplasia.

We determined the acceptor for α_3-fucosyltransferase from the literature (*5*, *6*). Our report (*2*) states that α_3-fucosyltransferase adds L-fucose at the C-3 atom of *N*-acetylglucosamine. Since a terminal *N*-acetylglucosamine on the acceptor is essential for enzyme activity (*9*), and since this prerequisite is fulfilled by desialodegalactofetuin, desialofetuin (as used as the acceptor for α_2-fucosyltransferase) is a very poor acceptor for α_3-fucosyltransferase.

The observation of elevated plasma fucosyltransferase activity during bone marrow hyperplasia does not contradict our findings. We had previously demonstrated (*10*) that a substantial increase of serum glycosyltransferases (apart from deterioration of cell function) is due to proliferative and secretory processes of neoplastic or even normal cells.

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