results from a third group (six animals) in which AD's were elicited in the ventral hippocampus proper without direct activation of the ventral subicular cortex. Markedly increased activity was found bilaterally throughout the ventral hippocampus (Fig. 1E) and, to a lesser extent, the dorsal hippocampal formation (Fig. 1F). Variable increases found in the ventral subicular cortex were associated with increased DG uptake in the nucleus accumbens, entorhinal cortex, and basal, medial, and cortical amygdaloid nuclei, as well as the confluence zone. The pattern of increased metabolism seen in the lateral septum was similar to that found in the dorsal hippocampal group but showed increased activity extending more ventrally in the ipsilateral lateral septum (Fig. 1, G and H).

When the ventral hippocampus proper was stimulated below the threshold for eliciting an AD (two animals) (*11*), a more localized pattern of increased activity was found in the ipsilateral hippocampal formation, limited to parts of the ventral hippocampus proper and posterior subiculum. Outside the hippocampal formation, increased activity was seen only in the dorsal margin of the lateral septum. The more frequently stimulated animal (*11*) showed a similar although lighter pattern of increased activity contralaterally in homologous structures.

Control rats implanted with electrodes (eight animals) showed increased activity only immediately adjacent to the electrode shaft.

This study demonstrates that the site of AD initiation within the hippocampal formation determines the pattern of increased activity seen in the temporal lobe and basal diencephalon. The close correlation between the propagation of AD's initiated in the dorsal and ventral hippocampus, shown by the DG technique, and known hippocampal projections (*4*) supports the concept that hippocampal AD's spread along the same efferent pathways used by less intense physiological activity.

Of particular interest is the finding of a far more extensive ventral subicular influence on hypothalamic structures than would be expected from the findings of axonal transport studies which show ventral subicular projections via the fornix confined primarily to the ventromedial region and mammillary nuclei of the hypothalamus (*5*, *6*). This suggests that some of the increased activity seen in the hypothalamus might arise from other projections.

It has been shown in our laboratory that the ventral hippocampal formation influences the hypothalamus primarily via nonfornix pathways (*8*). One possibility suggested by our findings is that the ventral subicular cortex influences the hypothalamus via the amygdala. The amygdala has been found by Krettek and Price (*12*) to project to anterior, lateral, and ventral premammillary regions of the hypothalamus, structures all showing increased metabolic activity. In the primate we found a high percentage of units in the basomedial nucleus of the amygdala responding with short latencies to ventral hippocampal formation stimulation (*13*). In addition, a direct projection from the ventral subiculum to the basomedial nucleus of the amygdala was demonstrated by Rosene and Van Hoesen (*6*). Increased activity in the claustrum associated with ventral subicular AD's may point to an as yet undescribed projection, either via the amygdala or perhaps directly from the ventral subicular cortex.

The DG technique has been shown to be highly effective in mapping the differential spread of temporal lobe AD activity initiated in different parts of the hippocampal formation. This system could be used to determine new hippocampal projections, such as the nonfornix pathway or pathways to the hypothalamus (*8*). It could also be employed to study kindling as well as the actions of drugs used in the treatment of epilepsy. These models might provide additional insight into the nature and treatment of this disease.

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1. As used in this report, the term hippocampal formation includes the dentate gyrus, the hippocampus proper (fields CA1 to CA4 of Ammon's horn), and the subicular cortex (prosubiculum, subiculum, presubiculum, and parasubiculum). Hippocampus alone refers to both the hippocampus proper and the dentate gyrus.
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Brain Grafts Reduce Motor Abnormalities Produced by Destruction of Nigrostriatal Dopamine System

Abstract. *In order to determine if brain tissue grafts can provide functional input to recipient central nervous system tissue, fetal rat dopamine-containing neurons were implanted adjacent to the caudate nucleus of adult recipients whose endogenous dopaminergic input had been destroyed. The grafts showed good survival and axonal outgrowth. Motor abnormalities, which had been induced by the destruction of the endogenous dopaminergic input to the caudate, were significantly reduced after grafting of the fetal brain tissue. These data suggest that such implants may be potentially useful in reversing deficits after circumscribed destruction of brain tissue.*

Many neurological disorders are associated with degeneration of discrete populations of neuronal elements. Parkinson's disease, for example, manifested primarily by abnormalities of movement and posture (*1*), is characterized by neuronal loss and gliosis in the dopamine-containing zona compacta of the substantia nigra (SN) (*2*). The syndrome is also characterized by decreased concentrations of dopamine (DA) and its principal metabolite, homovanillic acid

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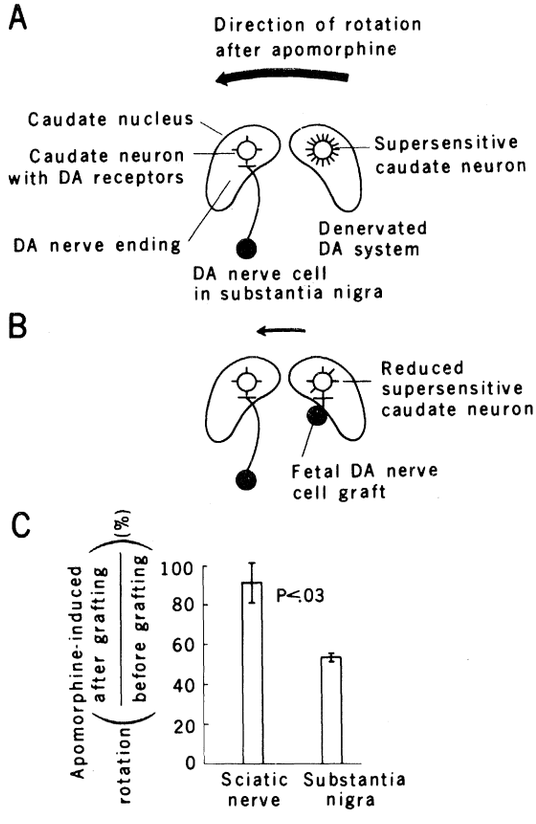

Fig. 1. (A) Schematic drawing of the nigrostriatal system with DA-containing cell bodies in the SN innervating neurons in the caudate nucleus. The right SN neurons were destroyed by the 6-OHDA injection. This caused the right caudate neurons to become supersensitive relative to normal left caudate neurons. Thus, systemic administration of the DA agonist apomorphine acts on the right caudate neurons to a greater extent than on the left caudate neurons. This causes the animal to walk or rotate in a counterclockwise direction. (B) When a DA-containing nerve cell graft is placed on the previously denervated caudate nucleus, the right caudate nucleus manifests less supersensitivity, as indicated by a reduction in the amount of counterclockwise rotation. (C) Graph of the ratio (expressed percentage) of apomorphine-induced turning after grafting to that before grafting (mean ± standard error of mean).

(HVA), in the neo- and paleostriatum (caudate, putamen, globus pallidus) and substantia nigra (*3*).

Current therapeutic approaches to Parkinson's disease involve peripheral administration of L-dopa, a precursor of DA, and DA-like agents. In practice, despite some dramatic improvements, such therapeutic regimens are frequently not completely effective (*4*), or are associated with severe untoward side effects (*5*). Many of these difficulties may result from (i) use of a precursor rather than the transmitter substance itself; (ii) generalized distribution of the drugs in brain rather than focal release from terminals in discrete areas; and (iii) absence of the physiological mechanisms which normally regulate transmitter release from dopaminergic terminals. We have attempted to circumvent these problems by using central nervous system grafts as a source of dopamine in an animal model of SN degeneration. We now report that SN neurons from fetal rat can be transplanted in juxtaposition to dopamine-denervated host adult caudate. The grafts proliferate extensively in situ and establish a behaviorally appropriate functional input to the denervated caudate.

As an experimental model, we chose the rat in which the DA system in the SN has been unilaterally destroyed by 6-hydroxydopamine (6-OHDA) (*6*). Within 1 week after a unilateral injection of 6-OHDA into the SN, DA-containing cells in that site, and their axonal terminals in the corpus striatum, degenerate; DA concentrations in the corpus striatum are greatly reduced (*7*). To compensate for the loss of DA, it is postulated that striatal neurons develop increased sensitivity to DA. This supersensitivity is manifested by increased numbers of DA receptor sites (*8*) and increased activity of DA-stimulated adenylate cyclase (*9*), and a decrease in the threshold of caudate neurons to the inhibitory effects of iontophoretically applied DA agonists (*10*). Behaviorally, animals with these lesions show pronounced rotation contralateral to the lesion after systemic administration of the DA receptor agonist apomorphine (Fig. 1A) (*11*). If a brain graft developed a DA input to the denervated caudate nucleus, we postulated that the supersensitivity, manifested as apomorphine-induced rotation, would be reduced.

Unilateral destruction of DA cell bodies in male Sprague-Dawley rats (150 to 160 g) was accomplished by injection of 6-OHDA into the right SN and ascending DA axon bundle (*12*). After 2 to 4 months, the rats were placed in a rotometer (*13*) and given a subcutaneous injection of apomorphine hydrochloride (0.1 or 0.25 mg per kilogram of body weight) (*14*). The number of clockwise and counterclockwise turns was recorded separately for each animal at 5-minute intervals for 40 minutes (Fig. 1A). Testing was repeated twice a week for 3 to 4 weeks.

Rats that had stable patterns of rotation (*14*) were subjected to grafting operations. Pieces of fetal ventral mesencephalon containing the SN or adult sciatic nerve were dissected out and injected into the lateral cerebral ventricle ipsilateral to the 6-OHDA lesion (*15*). Baseline turning rates were not statistically different for the animals in SN and sciatic nerve implant groups before transplantation ($P > .50$, F test).

Four weeks after grafting, animals were again tested for rotation in response to the same dose of apomorphine (twice a week for 3 to 4 weeks). By and large, most animals did not rotate as much after grafting as before (Fig. 1, B and C), but the reduction in turning was significantly greater for animals grafted with SN tissue than for animals with sciatic nerve implants. Data were analyzed statistically in terms of the ratios of the mean turning rate prior to transplantation, for each of the eight 5-minute segments of the 40-minute testing session. A two-way analysis of variance (groups by measures) revealed a significant main effect (Fig. 1C) for the type of graft ($F = 5.36$, d.f. $= 1$, $P < .03$). The main effect of measures (comparing 5-minute segments) was not significant (*16*).

The caudate nucleus and periventricular tissue of 12 of the 29 animals studied were examined by Falck-Hillarp fluorescence histochemistry and a double-blind protocol (*17*). Five of these animals had SN grafts and had at least a 70 percent reduction in their rotatory response to apomorphine (R animals). Four of 12 had SN grafts but less than 10 percent reduction in rotatory response to apomorphine (NR animals). The remaining three had sciatic nerve grafts (controls).

Although the sciatic nerve fibers degenerated after grafting, the histochemical studies revealed excellent survival and growth of the ventral mesencephalic SN grafts (*18*). Of 30 monoamine-containing implants examined in nine animals, all but one showed large numbers of catecholamine- and serotonin-containing neurons with a morphology typical of adult SN and raphe, respectively (Fig. 2, a and b). The catecholamine- and serotonin-containing cell bodies formed one or more dense clusters in the graft which were clearly separate. Monoamine-con-

taining fibers proliferated extensively within the grafts (Fig. 2, c and d) and invaded the adjacent caudate in all nine animals with SN implants (Fig. 2). The catecholamine fibers that grew into the denervated caudate formed a network only in the part of the caudate adjacent to the graft. A similar ingrowth was seen into the lateral septum. However, no monoamine-containing fibers invaded the corpus callosum, even at points of attachment of the graft. Monoamine-containing fibers also formed dense sheets of fibers within the ependyma of the lateral ventricle (Fig. 2d). For the most part, anteriorly placed grafts formed well-demarcated structures in the lateral ventricle opposed to or fused with the medial surface of the caudate and lateral surface of the septum (Fig. 2e). Some deeper grafts lay interstitially, fused with the host brain tissue. Transplants that were placed more posteriorly in the lateral ventricle were usually found adjacent to or fused with the fornix and dorsal alveus of the hippocampus (Fig. 2f). There was little or no evidence of any scarring or pathological disturbances of the host striatum.

We also compared the histochemical properties of grafts in animals that showed reduced turning after grafting with those that did not. Both groups had approximately equal mean numbers of catecholamine-containing cell bodies per animal and proliferating fibers in the graft (*17, 19*). Moreover, the ingrowth of fibers into the adjacent caudate was approximately equal in both groups. The major difference in the two groups was in the degree of caudate denervation produced by the 6-OHDA injection. The degree of initial 6-OHDA denervation could be estimated by histochemical examination of the host caudate, remote from the graft, and by counting the remaining DA-containing cells in the host SN. In all R animals these areas were virtually totally denervated. Every NR animal had some residual endogenous DA-containing cell bodies in the SN and weak to very weak diffuse endogenous DA fluorescence in large areas of the caudate. A second difference was in the location of the graft. The NR animals tended to have a more posterior graft localization within the ventricle, while R animals had a more anterior placement.

From the above studies, we conclude the following: (i) The grafts of fetal SN to the lateral ventricle adjacent to the caudate nucleus of the rats survived and proliferated. All but one of the grafts examined survived without rejection for at least 2 months. (ii) Grafting of the fetal

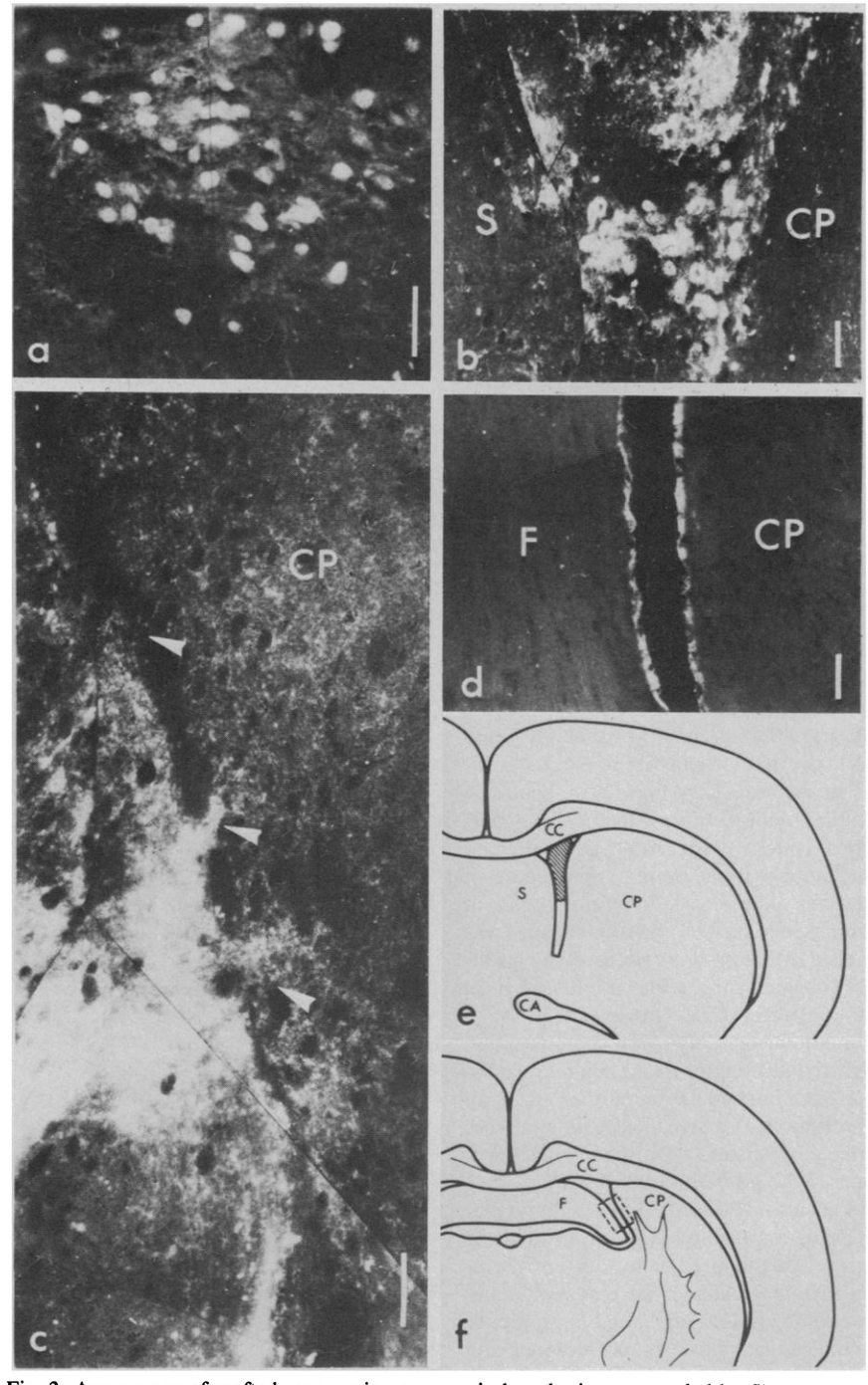

Fig. 2. Appearance of grafted monoamine neurons in host brain, as revealed by fluorescence histochemistry, 8 weeks after grafting. Before grafting hosts were treated with nialamide, a monoamine oxidase inhibitor, to increase intraneuronal monoamine levels. (a) Green-fluorescent, catecholamine-containing nerve cell bodies of the type found in all animals bearing SN grafts. Most cell bodies are small or medium-sized, similar in appearance to the DA neurons of the normal SN. (b) Yellow-fluorescent, serotonin-containing nerve cell bodies found in the lower part of another graft. This particular graft fills the lateral ventricle and is attached to the septum medially and to the caudate laterally. (c) SN graft fused to host caudate. The graft (lower left triangle) contains dense aggregates of catecholamine fibers. The ependymal lining of the caudate is traversed at several places (arrows) by tissue bridges carrying catecholamine fibers from the graft to the parenchyma of the caudate, in which the ingrowing fibers form thin varicose terminals. (d) Thick catecholamine fiber plexus from a SN graft covering the ventricular surfaces of the fornix and the caudate. The area photographed is indicated in (f). (e) Schematic coronal section illustrating typical graft location and size in the lateral ventricle. (f) The grafts often reach as far posteriorally as the level of the fornix, where they contacted the hippocampus; *CA*, commissura anterior; *CC*, corpus callosum; *CP*, nucleus caudatus putamen; *F*, fornix, and *S*, septum; calibration bars, 50 μm.

SN reversed, to some degree, the behavioral changes associated with destruction of the host nigrostriatal system. (iii) This recovery was associated with the growth of DA-containing cells and fibers in the grafts and into host brain tissue. However, from the data reported here, the functional recovery cannot be ascribed strictly to reinnervation of the caudate; release of DA and subsequent diffusion from the grafts into the caudate is another possibility. (iv) Incomplete destruction of the endogenous DA input, although sufficient to induce "behavioral supersensitivity," may antagonize reversal of the supersensitivity by a subsequent SN graft. (v) The small reduction in turning seen in control animals may result from a disturbance in caudate function secondary to grafting procedure, although histological examination did not reveal evidence of caudate injury.

Although numerous studies have demonstrated (i) that mammalian nervous tissue regenerates to some extent following injury (*20*), (ii) that various parts of the mammalian central nervous system can be grafted to peripheral tissue (*21*), and (iii) that nonnervous tissue can be grafted to the brain (*22*), there have been few studies demonstrating that central nervous system tissue could be grafted to and survive in the brain or spinal cord of another animal (*23*). In some studies in which the grafted nervous tissue was placed into host brain areas, fiber proliferation and synapse formation have been demonstrated (*23*). However, this is, to our knowledge, the first demonstration that the grafting of mammalian brain tissue from one animal to another elicits an alteration in the behavior of the recipient animal consistent with the normal function of the grafted tissue (*24*).

Parkinson's disease has a wide variety of signs and symptoms; not all of these are reliably improved by current therapy. Our data show that catecholamine-containing tissue, implanted in proximity to the caudate survives, proliferates, and can produce improvements in lesion-induced motor abnormalities. While clearly much additional research is required to establish the safety and long-term stability of grafts, there is a possibility that such a local catecholamine source could offer the patient with Parkinson's disease a more physiological treatment than is presently available. In this context, it is important to explore autografts or homografts (or both) of peripheral catecholamine-containing tissues, such as adrenal medulla (*25*). If further studies can establish the efficacy and safety of this approach for therapy, it may eventually be possible to employ techniques similar to those that we have used here in the treatment of other neurological disorders where the loss of nerve cells is reasonably well circumscribed.

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12. The rats were anesthetized with chloral hydrate (4 percent) and placed in a Kopf stereotaxic instrument with the incisor bar 2.4 mm below the interaural line. Injection of 6-OHDA (Sigma) (8 μg of free base in 4 μl of saline containing ascorbic acid, 0.2 mg/ml) was administered through a 27-gauge needle by a micrometer-driven 10-μl Hamilton syringe at a rate of 1 μl/min, after which the needle was left in place for an additional 2 minutes. The stereotaxic coordinates were: 4.2 mm posterior to bregma, 1.1 mm lateral to the midsagittal suture, and 7.5 mm below the dura. After removal of the needle, the skull defect was covered with bone wax, the skin incision closed with wound clips, and the animal given 30,000 units of penicillin subcutaneously.
13. The testing procedure was similar to that of Ungerstedt and associates (*11*). The animals were placed in polycarbonate cylinders of 12-inch diameter containing a layer of sawdust. A rubber band harness was fastened over the chest of each animal just behind the forelegs and attached to an overhead swivel by a length of movie projector spring 1/8 inch in diameter. After a 15-minute period of adaptation to the cyclinder, apomorphine was administered subcutaneously. Subsequent full 360° turns made by the animals were sensed by photocell-activated logic devices and recorded on printing counters. Back and forth movements without full turns were not counted by the logic circuitry.
14. Animals with lesions that did not turn after treatment with apomorphine (0.25 mg/kg, subcutaneously) were not considered well denervated (*7*), and were not used for further study. All animals were screened for circling behavior prior to the lesion. Circling movement was selected as the behavioral parameter since it is highly correlated with the degree of destruction of the SN and denervation of the caudate (*7*) and shows little compensation as a function of time after the lesion [W. Schultz and U. Ungerstedt, *Neuropharmacology* **17**, 349 (1978)]. The dose range of apomorphine was selected to elicit maximal responses in animals with adequate lesions (*11*), and minimal change in animals with insufficient lesions.
15. The monoamine neuron-containing brain grafts were obtained from Sprague-Dawley rat embryos of 17 to 18 days gestation (17 to 23 mm crown-rump length). They were dissected out of the ventral mesencephalon as described by Olson and Seiger (*21*). Three pairs of fetal brain areas, or an equivalent volume of adult rat sciatic nerve, were injected with 30 to 40 μl of Ringer solution (over 2 minutes) into the right lateral ventricle of the 6-OHDA lesioned rats by a modified lumbar puncture needle. The recipient rats were anesthetized with chloral hydrate and placed in a stereotaxic instrument with the incisor bar 5.0 mm above the interaural line. The stereotaxic coordinates of the injection site were 0.0 to 0.5 mm anterior to bregma, 1.5 to 1.6 mm lateral to the midsagittal suture, and 3.5 to 4.0 mm below the dura. After removal of the needle, the skull defect was covered by gelatin (Gelfoam), the scalp incision closed with wound clips, and the animal given 60,000 units of penicillin subcutaneously.
16. The measures by groups interaction was also significant ($F = 2.68$, d.f. = 7, $P < .01$). Individual comparisons between treatment groups for each 5-minute segment (Tukey B test) revealed statistically significant differences between treatment groups ($P < .01$) for three of the eight 5-minute segments; these were the segments from 0 to 5 minutes, 20 to 25 minutes, and 30 to 35 minutes following apomorphine injection. Since the analysis of variance did not reveal a significant main effect of testing interval, these intervals were grouped together to facilitate graphic presentation of this data. The fact that the largest differences were seen in the first and last intervals suggests that denervation-induced rotational changes are best differentiated using the lowest doses of apomorphine. Similar conclusions were reached by Marshall and Ungerstedt (*11*).
17. After treatment with nialamide (500 mg/kg intraperitoneally over 4 hours), brains were freeze-dried [L. Olson and U. Ungerstedt, *Histochemie* **22**, 8 (1970)] and further processed for Falck-Hillarp fluorescence histochemistry [B. Falck, N.-A. Hillarp, G. Thieme, A. Torp, *J. Histochem. Cytochem.* **10**, 348 (1962); H. Corrodi, G. Jonsson, *ibid.* **15**, 65 (1967)]. Cell bodies were counted on every tenth section (8 μm thick) and the number of cells derived from the formula $N = nt(t + d)^{-1}$ [M. Abercrombie, *Anat. Rec.* **94**, 239 (1946)], where n is the number of cell bodies counted times the sectioning period, t is the thickness of the sections, and d is the average diameter of the counted cell bodies. The mean numbers of neurons in the SN grafts were for catecholamines, 1974 ± 411 (mean ± standard error of mean) per animal, and for serotonin, 1369 ± 374. The number of DA neurons in the normal substantia nigra is approximately 3500 per side [N.-E. Anden, K. Fuxe, B. Hamberger, T. Hökfelt, *Acta Physiol. Scand.* **67**, 306 (1966)].
18. Some long-term survival of supporting cells in control grafts was seen, even though the sciatic nerve fibers had degenerated.
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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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