maturation, the feeding reflexes come under the inhibitory control of suppressor cells in the medial hypothalamus and the limbic forebrain. The activity of these cells, in turn, is inhibited by the noradrenergic food reward system in the lateral hypothalamus. In the mature organism, appetizing foods disinhibit the feeding reflexes from forebrain suppression by activating the noradrenergic food reward system. Lateral hypothalamic damage stops feeding because the forebrain suppressor cells no longer may be inhibited in this way and, in fact, may be in rebound activation. Finally, during recovery from hypothalamic damage, the noradrenergic regulation of suppressor cell activity is gradually and at least partially restored.

However, opposite effects of amphetamine on self-stimulation and feeding apparently contradict the view that the noradrenergic feeding and reward systems may be identified. This norepinephrine-releasing drug lowers thresholds of self-stimulation but raises thresholds of elicited feeding from the same lateral hypothalamic probe (*12*). According to our theory of food reward, a drug that lowers reward thresholds by release of norepinephrine (*6*) should facilitate feeding by the same action.

Although amphetamine generally suppresses feeding and is used clinically as an anorexic agent, it also facilitates feeding under some conditions. Thus, amphetamine increases food intake if eating is induced by high intensity stimulation of the lateral hypothalamus (*13*), or if severely deprived rats are offered a wet and highly palatable food (*14*). These observations may be reconciled with the anorexic action of amphetamine if the drug exerts multiple and antagonistic effects on feeding. Under most circumstances, the anorexic effect of amphetamine will obscure its facilitating action; however, facilitation may predominate if the noradrenergic feeding system is activated by electrical stimulation or if it is sensitized by severe food deprivation.

As one test of this idea, we attempted to activate the noradrenergic feeding system of amphetamine-treated rats by intraventricular administration of norepinephrine. Figure 2 shows, in a responsive animal, that norepinephrine rapidly reverses the anorexia induced by amphetamine. This result fits our noradrenergic theory of food reward, and suggests, furthermore, that the anorexic action of amphetamine does not depend on the release of norepinephrine at α-noradrenergic synapses in periventricular regions of hypothalamus and forebrain (*15*).

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14. Twenty rats were reduced to 75 to 80 percent of normal body weight by limited feedings of dry food, and ten controls had free access to food. *d*-Amphetamine sulfate (2 mg/kg, intraperitoneally) or saline was administered in a balanced sequence 15 minutes before a 30-minute feeding test, in which the rats (food deprived for 24 hours) were offered a wet mash of Purina Lab Chow and water. Mean intakes in grams $\pm$ S.E.M. were: amphetamine, free access, 15.4 ± 3.8 compared to starved, 37.1 ± 2.2 ($P < .001$); saline, free access, 32.5 ± 1.3 compared to starved, 33.6 ± 0.9 ($P > .2$). For another example of amphetamine-induced feeding, also see S. F. Leibowitz, *Proc. 78th Annu. Mtg. Amer. Psychol. Ass.* **5**, 813 (1970).
15. Our findings apparently pose difficulties for a recent model [D. Margules, *Life Sci.* **8**, 693 (1969)] in which it is assumed that α-noradrenergic synapses in the hypothalamus mediate satiety rather than feeding. It is possible to reconcile this idea with the view of Miller (*4*), that α-receptors mediate feeding if one assumes that satiety depends on the accumulation of a fixed number of reward messages from the α-noradrenergic feeding system. If so, Margules' finding that the α-antagonist phentolamine increases the intake of a palatable food could be explained. In moderate doses, phentolamine would partially block the noradrenergic reward message, and thus require that a larger than normal number of messages (overeating) be received before the satiety cutoff criterion was reached. More complete blockade of the reward message by phentolamine (higher doses, intraventricular rather than intrahypothalamic administration) should of course reduce intake (see Table 2).
16. We thank H. Morris, W. J. Carmint, and A. Rothchild for technical assistance, and A. T. Shropshire for devising an improved intraventricular injection technique.

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Height and Antisocial Behavior in XY and XYY Boys

Abstract. *The observed association of the XYY genotype with both (i) large height in childhood and (ii) institutionalization for antisocial behavior suggested that large size per se in childhood might tend to establish personality patterns leading to eventual incarceration for delinquency. To investigate this question, the height distributions of four groups of XY boys in institutions for nonpsychotic, nonretarded juvenile offenders were compared with the published standards as well as the predicted gaussian distributions calculated from the mean and variance of the age-adjusted heights of each group. In none of these groups was there evidence for an increased number of large individuals. But three XYY individuals in the same institutions all had heights greater than the 90th percentile of XY boys of the same race.*

Almost all reports of XYY individuals with antisocial behavior have noted the presence of large height. Furthermore, while many descriptions are incomplete, the available published evidence indicates that tall stature has been present from childhood on. Thus a question of some interest is whether large size per se has been the factor accounting for the increased frequency of XYY individuals in institutions for antisocial behavior. There are at least four possible explanations as to why increased height might predispose to misbehavior. (i) Larger children would be more likely to be successful in fights with children of their own age and are more likely to find that threats or acts of aggression would succeed. (ii) Larger children, appearing older than their age, might elicit greater social and intellectual expectations from their elders than their ability would warrant. This might lead to greater opportunities to enter into mischief outside of school, as well as frustration resulting in disruption in school. (iii) Larger children may be more likely to be sin-

Table 1. Observed height distributions compared with published standards (XY boys only). Numbers in parentheses indicate number of subjects.

Subjects	Height bracket by ½ standard deviations											Goodness of fit χ^2 (9 d.f.)	Mean and S.D. of height units
	< −2.50	−2.50 to −2.01	−2.00 to −1.51	−1.50 to −1.01	−1.00 to −0.51	−0.50 to −0.01	0.00 to 0.49	0.50 to 0.99	9.2% 1.00 to 1.49	1.50 to 1.99	2.00 to 2.49	≥ 2.50	
	Expected												
	0.5%	1.7%	4.4%	9.2%	14.9%	19.2%	19.2%	14.9%	4.4%	1.7%	0.5%		
						Institution A							
White (86)													
Observed	3	6	8	15	11	19	14	7	1	0	1	1	49
Expected	2.0		3.8	7.9	12.8	16.5	16.5	12.8	7.9	3.8	2.0		$P \sim .01$ −.65±1.13
Black (117)													
Observed	0	4	7	12	15	15	25	21	9	6	2	1	5.9
Expected	2.6		5.2	10.8	17.4	22.5	22.5	17.4	10.8	5.2	2.6		$P \sim .75$ +.04±1.11
						Institution B							
White (44)													
Observed	0	1	1	4	6	11	15	3	3	0	0	0	11.5
Expected	1.0		1.9	4.0	6.6	8.4	8.4	6.6	4.0	1.9	1.0		$P \sim .25$ −.17± .75
Black (44)													
Observed	0	1	1	3	2	12	6	9	7	3	1	0	10.7
Expected	1.0		1.9	4.0	6.6	8.4	8.4	6.6	4.0	1.9	1.0		$P \sim .30$ −.15± .91

gled out of an unruly or antisocial group for censure. (iv) Larger children would be more likely to associate with older children of the same size and be led into antisocial activity by more experienced individuals. We recognize that these are rather simplistic explanations, and they may be operative in only a fraction of large children. However, they do suggest mechanisms whereby large height in childhood might indirectly lead to an increase in aggressiveness, increase in conflict with authority, and an expectation of immediate satisfaction in interaction with peers and consequent frustration when this is not achieved. (These tendencies might be particularly enhanced in individuals who were below average in intelligence, another frequent observation in those with XYY syndrome.)

If large size contributed significantly to misbehavior in XYY individuals during childhood, then one might expect that large children would also be proportionately overrepresented in populations of XY boys who have committed antisocial acts. While a great number of analyses of the physical dimensions of adult criminals have been published, we are unaware of any reports of stature in delinquent children. However, such observations would be particularly relevant if there is any causal relationship of size to antisocial behavior, since personality patterns are likely to be established well before final stature is attained.

To investigate this question we determined the heights of all inmates of two institutions for nonpsychotic offenders under the age of 16 who had been committed by the courts because of antisocial behavior. All boys had IQ's above 69. Cytogenetic studies were also performed, and the findings have been reported (*1*).

Institution A receives all nonpsychotic, nonretarded boys, in the state investigated, under age 13 who are referred by the courts because of antisocial behavior and for whom no private placement is deemed available or advisable. It is the only public facility for such delinquent boys under age 13 in the state. Almost all of the boys are from low socioeconomic backgrounds and are removed from the home, frequently on petition of a parent, because of misbehavior and inadequacies in family environment. Children of wealthier parents who commit infractions usually initially enter a private or semiprivate setting, but institution A will eventually receive them if they are expelled by private institutions because of behavioral difficulties. Boys unmanageable in institution A will be transferred to institution B. In addition, institution B receives all boys ages 13 or 14 who are convicted of murder, arson, or rape, and all boys 13, 14, or 15 who are referred from other public institutions for nonretarded, nonpsychotic offenders because of unmanageable behavior. Institution B is thus the placement of last resort for all such offenders under 15 in the state. The population is more unmanageable, has a greater criminal history, and is from a higher socioeconomic background than that of institution A.

Our expectation was that the average height would be lower than the norms for age. We anticipated that poverty, neglect, and malnutrition would be present in the background of a significant number of the children studied, and that these factors in general would diminish size. In addition, if the hypothesis regarding large height were correct, we also expected an overrepresentation of children much taller than this suboptimum mean.

The heights of all boys were taken without shoes, by standard methods, in midmorning. Their ages were calculated to the nearest day. Since height in children is age-dependent, comparisons can only be made by the use of age-adjusted standards and comparison of the deviations of each child's measurement from the expected normal mean for his age. This was done by making growth curves from the data of Reed and Stuart (healthy white children of upper-middle-class Boston) and Verghese *et al.* (healthy black children of low-income families in Washington, D.C.) (*2*). (There were about 10 percent Puerto Rican children in the institutions, but the data on them were not analyzed because there are no published standards for these children, and they were a small proportion of the population.) For each age the values of the published means and the standard deviations from the means were plotted, and the appropriate points were connected. The height data were then plotted on the graph.

For each child's height the number of standard deviations from the mean of his exact age was determined graphically to the nearest tenth. To avoid confusion in the discussion below we will refer to these deviations as height units. A child "0" units tall

Table 2. Height distributions compared with distribution predicted from mean and S.D. of height units calculated in Table 1. Numbers in parentheses indicate number of subjects.

Subjects	Height bracket by ½ standard deviations											Goodness of fit χ^2 (7 d.f.)
	< −2.50	−2.50 to −2.01	−2.00 to −1.51	−1.50 to −1.01	−1.00 to −0.51	−0.50 to −0.01	0.00 to 0.49	0.50 to 0.99	1.00 to 1.49	1.50 to 1.99	2.00 to 2.49	≥2.50	
Institution A													
White (86)													
Observed	0	3	2	5	16	14	19	17	7	1	1	1	7.5
Expected	——— 2.0 ———		3.8	7.9	12.8	16.5	16.5	12.8	7.9	3.8	——— 2.0 ———		$P \sim .35$
Black (117)													
Observed	0	2	6	12	17	16	29	17	10	6	1	1	4.6
Expected	——— 2.6 ———		5.2	10.8	17.4	22.5	22.2	17.4	10.8	2.5	——— 2.6 ———		$P \sim .7$
Institution B													
White (44)													
Observed	1	0	3	4	4	8	8	11	2	2	1	0	5.7
Expected	——— 1.0 ———		1.9	4.0	6.6	8.4	8.4	6.6	4.0	1.9	——— 1.0 ———		$P \sim .55$
Black (44)													
Observed	0	1	3	1	9	9	7	6	6	2	0	0	6.1
Expected	——— 1.0 ———		1.9	4.0	6.6	8.4	8.4	6.6	4.0	1.9	——— 1.0 ———		$P \sim .50$

is the appropriate height for his age, a child "− 1.5" units is one and a half standard deviations below the mean appropriate height for his age, and so forth. The distributions of these data for the XY boys were compared with the normal distribution of the standards (Table 1). The means of these calculated units and their standard deviations from the means were determined and are also noted. The observed distributions were then compared with that predicted by normal distributions with the same means and variances (Table 2).

The two groups of black children and the white children in Institution B have height distributions that are not significantly different from those predicted by the published standards, and the mean heights are not significantly different from 0 units. (It is possible that the standards for the black children would have been higher if data from upper-middle-class black families were available.) The distribution is markedly different for the white children in institution A. Children here are on the average −0.65 height units and are significantly smaller than the standards.

However, there was not a significant enrichment of tall boys judged either by the published standards (Table 1) or by the calculated distributions of the populations themselves (Table 2). For instance, in the total of the four groups there are 15 boys greater than 1.5 height units in custody (Table 1) and 16 boys with heights greater than 1.5 standard deviations for the mean of their group (Table 2). But the expected number of such individuals is 19.6 in both cases. For 2 height units or 2 standard deviations the corresponding figures are 6 and 5 observed, compared to 6.4 expected.

If large height were the sole explanation for the higher incidence of the XYY genotype among delinquents, then one would expect the height of XYY children to have the same distribution as that of delinquent XY boys in these institutions.

In the four groups there were three XYY children all in Institution A. Their socioeconomic backgrounds were similar to those of the other boys. Their IQ's ranged from 1 to 15 points lower than the average of boys of the same race and age in their institution. One white XYY child was 0.52 height units and in the upper 90 percent of his group for size, and two black children were 2.13 and 2.91 height units and in the upper 97 percent and 99 percent of their group for size, respectively. The probability of finding randomly three children with these or taller relative heights is extremely small ($P < .001$).

We interpret the data as indicating that large height per se during late childhood is not exclusively responsible for the frequency of delinquency among XYY individuals and is unlikely to be a strong contributory cause.

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3. Note added in proof: While this report was in press we became aware that S. Glueck and E. Glueck found no significant difference in height between delinquent and nondelinquent boys between 11 and 18 years of age [*Unraveling Juvenile Delinquency* (Commonwealth Fund, New York, 1950), pp. 184–85].

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Unilateral Ablation of the Auditory Cortex in the Cat Impairs Complex Sound Localization

Abstract. *Unilateral ablation of the auditory cortex in the cat results in a profound deficit in attending to stimuli on the side contralateral to the lesion. The deficit is also manifested in an abnormal perception of left-right pulse pairs when the pulse which leads by a few milliseconds is contralateral to the damaged hemisphere.*

We planned these experiments to test the idea that an important function of the auditory cortex may be the identification of a particular locus when identical sounds emanate from different loci at closely spaced temporal intervals (*1, 2*). In the course of this inquiry a profound deficit in auditory discrimination was uncovered after the unilateral ablation of the auditory