and in which commissurotomy was not performed, the abnormal behavior, even when tested soon after the operation, was either mild or did not occur.

The monkeys' behavior with the affected arm may represent the visual counterpart of the somatosensory guided grasping and groping behavior (*4*) that can occur in monkey and man with contralateral premotor lesions. The behavior in some respects resembles the defect in delayed alternation response in which animals show an overriding tendency to reach to where food had previously been obtained. It also resembles Stepién's (*5*) "magnet reaction," in which animals presented with a conditioned stimulus at one place and a reward at another tend to go and to remain at the place where the conditioned stimulus is delivered. However, these behavioral defects are the result of bilateral lesions located more rostrally than our premotor ablation (*5, 6*).

The behavior of our animals might be due to the fact that the ablation, which presumably interferes with the cortical steering of axial and proximal movements (*7*), also results in a disinhibition of a subcortical region that directs the contralateral arm and hand straight to a visual target, in the same way as the superior colliculus, for example, may direct the gaze (*8*).

This postulated subcortical region probably contributes fibers to the ventromedial group of descending brainstem pathways (*9*), since accurate reaching movements of the arm to a visible object can be readily elicited in bilaterally pyramidotomized animals (*9*), but are largely abolished when in these animals the ventromedial brainstem pathways (*9*) are also transected. The cells of origin of these brainstem pathways receive many cortical connections directly from the ablated premotor areas and the rostral part of the precentral gyrus (*10*), as well as indirectly by way of the striatum and the substantia nigra (*11*). Elimination of some of these connections may be responsible for the disinhibition of the postulated subcortical region steering the arm directly to a visual target.

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How Many Anticodons?

Abstract. *Much new information on codon composition is becoming available from the sequencing of molecules of DNA and RNA. The "wobble rules" for codon-anticodon pairing are applicable to this information. These rules provide for only 54 anticodons to pair with 61 codons, because the base A is not found in the first position of anticodons.*

Recent advances in molecular biology have enabled long sequences of nucleotides to be identified in DNA and RNA molecules (*1*). This new information enlarges the scope of molecular evolution, which formerly depended mainly on comparisons of the amino acid sequences of proteins. Such sequences reveal very little about the third bases of codons, but, with the new information on nucleotide sequences, these are now identified. As a result, Sanger *et al.* (*2*) found that there is a disproportionately high use of T (U) (*3*) in the third base position of the codons of ϕX174. Such information draws attention to the "wobble rules" of pairing (*4*) between the first bases of anticodons and the third bases of codons. Some amino acids have only pyrimidine-terminated codons. The only known anticodons for such amino acids start with G, for example, GAA for phenylalanine. The wobble rules do not exclude the possibility of AAA; the absence of A from the first position of all known anticodons is thought to result from the action of anticodon deaminase (*5*). This changes adenine to hypoxanthine, so that anticodons starting with I (inosine) occur in the case of amino acids with more than two codons, such as valine. The absence of an IAA anticodon for phenylalanine is explainable by concluding that this anticodon is eliminated by lethality, for it could "mis-pair" with UUA (leucine), and similarly pari passu for the other amino acids whose codons terminate only with a pyrimidine. It is therefore anticodon deaminase rather than [as stated elsewhere (*6*)] the wobble rules that explain "why there are not 61 transfer RNA's (tRNA's)." Amino acids with two purine-terminated codons can have two anticodons, such as lysine with anticodons UUU and CUU. However, in the case of, for example, valine, with four codons, there are three known anticodons (IAC, GAC, and UAC) and there is no reason why CAC may not exist. The wobble rules provide for redundancy in coding rather than for postulating a number of anticodons less than 61, but the redundancy does not extend to amino acids whose codons terminate only with a pyrimidine. The total number

Table 1. Amino acid anticodons, possible and identified (italic).

GAA	Phe	*IGC*	Ala
UAA	Leu	GGC	Ala
CAA	Leu	*UGC*	Ala
IAG	Leu	CGC	Ala
GAG	Leu	*GUA*	Tyr
UAG	Leu	*GUG*	His
CAG	Leu	*UUG*	Gln
IAU	Ile	*CUG*	Gln
GAU	Ile	*GUU*	Asn
UAU	Ile	*UUU*	Lys
CAU	Met	*CUU*	Lys
IAC	Val	*GUC*	Asp
GAC	Val	*UUC*	Glu
UAC	Val	CUC	Glu
CAC	Val	*GCA*	Cys
IGA	Ser	*CCA*	Trp
GGA	Ser	*ICG*	Arg
UGA	Ser	GCG	Arg
CGA	Ser	UCG	Arg
IGG	Pro	CCG	Arg
GGG	Pro	*GCU*	Ser
UGG	Pro	*UCU*	Arg
CGG	Pro	GCU	Arg
ICU	Thr	ICC	Gly
GGU	Thr	*GCC*	Gly
UGU	Thr	*UCC*	Gly
CGU	Thr	*CCC*	Gly

of anticodons, counting them as transcribed, before modification of bases, should therefore be 54: one apiece for Phe, Met, Tyr, His, Asn, Asp, Cys, and Trp; two apiece for Gln, Lys, and Glu; three for Ile; four apiece for Val, Pro, Thr, Ala, and Gly; five for Ser; and six apiece for Leu and Arg. Thirty-seven of these have so far been identified in tRNA molecules (*7*). Some of the anticodons in tRNA molecules contain modified first bases (other than hypoxanthine). Such modifications may either restrict (*8*) or extend (*9*) wobble pairing without, of course, engendering ambiguity in amino acid incorporation during peptide synthesis.

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3. The abbreviations used in this report are: A, adenine; T, thymine; U, uracil; G, guanine; C, cytosine; I, inosine; Ala, alanine; Arg, arginine; Asn, asparagine; Asp, aspartic acid; Cys, cysteine; Gln, glutamine; Glu, glutamic acid; Gly, glycine; His, histidine; Ile, isoleucine; Leu, leucine; Lys, lysine; Met, methionine; Phe, phenylalanine; Pro, proline; Ser, serine; Thr, threonine; Trp, tryptophan; Tyr, tyrosine; and Val, valine.
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Wolf-Pack Buffer Zones as Prey Reservoirs

Abstract. *In a declining herd, surviving deer inhabited overlapping edges of wolf-pack territories. There, wolves hunted little until desperate, in order to avoid fatal encounters with neighbors. Such encounters reduce wolf numbers and predation pressure and apparently allow surviving deer along territory edges to repopulate the area through dispersal of their prime, less vulnerable offspring into territory cores.*

Predator-prey systems tend to survive for long periods despite the negative effect of the predators. However, the mechanics of the survival process have been little discussed. The process must involve evolutionary strategies of predator, prey, or both, that (i) benefit one or both, (ii) allow the predator to obtain sufficient prey, and (iii) allow enough prey to survive.

One such evolutionary strategy is the tendency of wolves (*Canis lupus*) to prey disproportionately on older animals. Although those predators attempt to catch any prey they can, their physical abilities restrict them to capturing primarily, if not exclusively, disadvantaged or debilitated prey (*1*). Thus wolves can eat, yet their prey populations can themselves survive and produce a crop that wolves can continue to harvest. This strategy requires a precise adjustment of the abilities of both predator and prey.

A second strategy, which relies on the spatial organizations of both predator and prey, is the subject of this report. I recently discovered this strategy in studying drastically declining populations of white-tailed deer (*Odocoileus virginianus*) and wolves in northeastern Minnesota. Deer surviving the decline were distributed almost exclusively along the edges of wolf-pack territories. Although the published evidence is only suggestive (*2, 3*), enough supporting data are accumulating (*4, 5*) to warrant the exposition of a theory of the role of wolf-pack territory edges in the survival of deer populations.

Wolf packs in northeastern Minnesota inhabit a mosaic of adjoining territories of 125 to 310 km^2 each (*6*). Around each territory lies a strip about 2 km wide, the "buffer zone," in which the pack on either side can be found, but in which neither probably spends much time (*7*). Deer usually live throughout wolf territories. Individual deer inhabit areas of 0.48 to 4.10 km^2 in summer and tend during winter to congregate in "yards" as far as 38 km away from summer ranges (*3*).

The precise size and nature of the wolf-pack buffer zone, as well as the behavior of adjacent wolf packs when within it, are unknown. However, evidence indicates that wolves may feel insecure in this peripheral strip and thus may minimize the time they spend there. Wolves will try to kill members of neighboring packs when they meet (*8, 9*), and the maximum chance of an encounter is in the buffer zone. The rate of scent-marking by each pack in the buffer zone is about twice that in the territory center (*7*), which suggests higher anxiety near the territory edge.

One pack of wolves (Harris Lake pack) studied intensively for seven winters killed few deer in its buffer zone when the deer population was adequate to sustain wolf numbers (*2*). However, the deer herd declined rapidly during the next few years as a result of a combination of adverse factors including a high wolf population (*10*). Wolf pups then starved (*9, 11*), wolf productivity dropped (*9*), and wolves became desperate for food (*2*). Only then did packs begin trespassing widely into neighboring territories, and only then did the Harris Lake pack begin killing deer in its buffer zone (*2*). Other packs did likewise.

Meanwhile, the few remaining deer-wintering areas lay in wolf-pack buffer zones (*3, 12*). Deer migrated from them through one or two pack territories and summered in the buffer zones of other packs (*3*). Furthermore, those deer were generally older and had survived longer than deer that had lived throughout the area when the population was higher (*3*). There is little evidence that the deer sought out the buffer zones. Rather, it appears that these animals just happened to live there and that they survived longer because such areas were less used by wolves. Although such interactions became apparent only when the deer to wolf ratio decreased drastically, similar but less extreme interactions probably take place when deer to wolf ratios are more usual.

I propose that these relationships are important in helping to perpetuate the prey population, thereby also helping to perpetuate the predator and, thus, the entire predator-prey system. My theory is that because wolf packs tend to avoid intensive use of buffer zones, deer inhabiting those areas tend to survive longer and form a reservoir for maintaining and recovering deer populations in the wolf territory cores.

This theory implies that when deer populations are high, summer deer densities may be higher in buffer zones than in territory cores, but the disparity will be less than when populations are low, when it may not even be measurable. If deer numbers decline, they will become lowest in wolf-pack territory cores first, and dispersing deer from the buffer zone reservoirs can help replenish the cores. If the decline is too great and wolves are forced increasingly into the buffer zones, the probability of mortal strife (*9*) among wolf packs increases. This tends to discourage pack use of buffer zones and reduces the wolf population (*9*), thus minimizing predation in that area. Because buffer zones would constitute 25 to 40 percent of a region, enough deer would survive there to help repopulate the rest of the area.

Because deer in the buffer zones