study stand. By 32 days after wildebeest reached the study site, substantial numbers of Thomson's gazelle were entering the area, and a transect was made of randomly located sites. Vegetation properties were measured, and the sites were scored according to the presence or absence of Thomson's gazelle. Sites where gazelle were absent could be stratified into low and high biomass stands, and the data indicated that gazelle entered the area of the vigorous regrowth and high forage density that followed wildebeest grazing (Table 2). The stands favored by gazelle had intermediate biomass, a short canopy, and high biomass concentration. Over the following 30 days, consumption by gazelle averaged 1.05 g m^{-2} day^{-1} in areas previously grazed by wildebeest, but only 0.27 g m^{-2} day^{-1} in areas not grazed by wildebeest (t = 6.916 for $P < .001$; d.f. = 9). In fact, consumption in areas not previously grazed by wildebeest could not be discriminated from zero (t = 1.825 for P not significant; d.f. = 9). The distinctive character of wildebeest-grazed stands, and the association of gazelle with them, was maintained throughout the dry season of 1974. A final transect through the area was done on 14 November 1974, just at the end of the dry season. Randomly chosen stands were scored as follows: wildebeest-grazed, gazelle present, 11; wildebeest-grazed, gazelle absent, 4; not grazed by wildebeest, gazelle present, 0; not grazed by wildebeest, gazelle absent, 8. This represents a significant (Fisher's exact P = .001) association between grazing by wildebeest during their migratory passage and dry season exploitation by gazelle.

Wildebeest and Thomson's gazelle are the two most abundant grazers in the Serengeti-Mara ecosystem. The mechanisms of coexistence of such numbers of large grazers in a small area have been studied for some time (*10*). The present data indicate that heavy grazing by the migratory wildebeest population as it leaves the Serengeti Plains prepares the plant community for subsequent dry season exploitation by gazelle. The association of gazelle with areas previously grazed by wildebeest suggests that coexistence of these two species is a consequence of coevolution that has partitioned the grassland exploitation patterns during the critical dry season. Rather than competition, there is facilitation of energy flow into the gazelle population by the wildebeest population through the impact of the latter on the plant community. In addition to converting a senescent plant community into a productive one, it seems likely that wildebeest grazing substantially improves forage quality. Nutrient content and digestibility are considerably greater in rapidly growing grasses than in mature tissues (*11*). It therefore seems likely that total nutrient and energy flow to the gazelle population is facilitated even more than is suggested by these data on net primary productivity and gazelle grazing.

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12. I thank the trustees and director of Tanzania National Parks for permission to reside and work in the Serengeti National Park; T. Mcharo, director of the Serengeti Research Institute (SRI), for inviting me to work there; and F. Banyikwa, F. Kurji (both of the SRI), and M. McNaughton for valuable assistance in collecting data. Supported by the NSF ecosystem studies program grant BMS74-02043 to Syracuse University, and by a grant of sabbatical leave from Syracuse University. This is SRI publication No. 207.

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Human Heart and Platelet Actins Are Products of Different Genes

Abstract. *The amino acid sequences of selected cyanogen bromide peptides from human blood platelet actin and human cardiac muscle actin were compared; it was found that, at position 129, platelet actin has threonine, and that cardiac muscle actin has valine. Thus human cytoplasmic and myofibrillar actins must be synthesized under the control of different genes.*

Actin has been identified in many kinds of cells including muscle, where it is the major constituent of the thin filament. Actin participates in several reversible intermolecular interactions, and one of these, the formation of "arrowhead" complexes (*1*) with a truncated form of myosin, heavy meromyosin, has been used to identify "actin" in both animal (*2*) and plant (*3*) cells. Actins have been isolated from various muscles as well as from nonmuscle sources, and comparisons of their properties reveals that the different actins are very similar with respect to molecular weight and amino acid composition, including the presence of one residue of the unusual amino acid N^{τ}-methylhistidine. The results suggest that the structure of actin has been conserved and recent studies have shown that, although not completely invariant, muscle actins from such diverse sources as rabbits (*4*) and fish (*5*) are extremely similar in amino acid sequence.

The presence of actin in different tissues within the same organism raises the question of whether these actins are the products of the same gene, and thus identical in sequence, or whether a given organism has multiple separate, independently controlled genes for actin. Previous studies that bear on this question have employed peptide pattern techniques, and have yield-

Human Platelet:	Phe-Glu-Thr-Phe-Asn-Thr-Pro-Ala-Hse
Human Cardiac Muscle:	Phe-Glu-Thr-Phe-Asn-Val-Pro-Ala-Hse
Rabbit Skeletal Muscle:	Phe-Glu-Thr-Phe-Asn-Val-Pro-Ala-Hse

Fig. 1. Comparison of the sequences of peptide CB-4 from three actins. These peptides correspond to residues 124-132 in rabbit skeletal muscle, and the Val → Thr substitution is at position 129.

ed conflicting results. Booyse *et al.* (*6*) compared the gel electrophoretic patterns of cyanogen bromide fragments of rabbit skeletal muscle and human platelet actins; at least one of the peptides migrates differently, suggesting that the proteins are not the same. Bray (*7*) compared tryptic peptide patterns of chick muscle and brain actins, found them to be indistinguishable, and concluded that in a given animal, the actins are probably identical—the products of a single gene. Gruenstein and Rich (*8*) compared the tryptic peptide patterns of chick muscle and brain actins and reported that brain actin shows additional peptides and thus must be different, and probably larger, than muscle actin. In an attempt to determine unequivocally the relation between muscle and nonmuscle actins in a single species, we have directly compared the amino acid sequences of specific segments of human blood platelet actin and human cardiac muscle actin.

Platelet actin was prepared as described (*9*) from washed frozen human platelets obtained from thawed platelet-rich plasma. Cardiac muscle actin was isolated (*10*) from an acetone powder of hearts obtained after autopsy of patients dying of noncardiac related diseases.

The purified proteins were reduced and alkylated, digested with cyanogen bromide, and passed over a Sephadex G-50 column. The peaks corresponding to CB-3, CB-4, and CB-5 in rabbit skeletal muscle were isolated (*11*), and the peptides were purified by chromatography on SP-Sephadex C-25 (*12*). The amino acid compositions of CB-3 and CB-4 are listed in Table 1.

Table 1. The amino acid compositions of cyanogen bromide peptides CB-3 and CB-4 from different actins. Numbers indicate moles of amino acid per mole of peptide. Italicized values are those that differ in the two human actins. The numbers in parentheses indicate assumed residues per mole.

Amino acid (*18*)	CB-3			CB-4		
	Rabbit skeletal muscle	Human blood platelet	Human cardiac muscle	Rabbit skeletal muscle	Human blood platelet	Human cardiac muscle
Arg	1	1.05 (1)	1.00 (1)			
Asx	1*	1.07 (1)	1.11 (1)	1†	1.04 (1)	1.11 (1)
Thr				1	*1.93* (2)	*0.97* (1)
Glx		0.25		1‡	1.10	1.20 (1)
Pro	1	1.21 (1)	1.05 (1)	1	1.10	1.03 (1)
Gly	1	1.13 (1)	1.02 (1)		0.16	0.16
Ala	1	0.96 (1)	1.09 (1)	1	1.05 (1)	1.02 (1)
Val				1	*0.04* (0)	*0.92* (1)
Ile	1	0.82 (1)	0.90 (1)			0.19
Tyr	1	0.60 (1)	0.80 (1)			
Phe				2	1.79 (2)	1.82 (2)
Hse	1	0.52 (1)	0.71 (1)	1	0.81 (1)	0.80 (1)

*Aspartic acid. †Asparagine. ‡Glutamic acid.

The composition of CB-3 from both platelet and cardiac actins appeared to coincide with that of rabbit skeletal muscle CB-3, and its sequence is thus probably identical. In contrast, CB-4 from platelet actin appeared to have two threonine residues and no valine, while both of the muscle actins, rabbit skeletal and human cardiac, have one of each; this suggests a substitution of threonine (Thr) for valine (Val) in platelet CB-4. The two human actin peptides were then sequenced by means of a solid phase sequencer (*13*), and threonine was found at step 6 in platelet actin, while valine occurred at step 6 in cardiac muscle actin. The rest of the sequences were identical, and the peptides are compared in Fig. 1. Peaks CB-5 from both human actins were subjected to sequence analysis and found to be identical with rabbit skeletal muscle CB-5.

On the basis of the identity of 28 of the 29 residues in these three peptides, it is clear that the human actins are very similar to each other and to rabbit skeletal muscle actin; yet the Val → Thr difference unequivocally demonstrates that human platelet and cardiac muscle actins are the products of different genes.

The significance of multiple forms of actin is not obvious, especially since muscle and nonmuscle actins from different cells are so similar in behavior. There may, of course, be subtle but functionally important differences in properties among various actins that are difficult to detect; suggestions that this may be the case come from the work of Abramowitz *et al.* (*14*), who found that there may be a form of platelet actin that has polymerization properties different from muscle actin, and the finding by Stossel and Hartwig (*15*) of an actin-binding protein that is involved in the aggregation of macrophage actin.

An additional explanation is that multiple actin genes, being expressed even within a given cell, are independently responsible for the synthesis of actins that participate in different nonsynchronous functions. For example, a developing muscle cell goes through a number of phases during which actin may be involved in a variety of cellular activities. For example, (i) before differentiation into a muscle cell actin is present in cytoplasmic filaments (*2*), (ii) during cell division it is part of the mitotic spindle apparatus (*16*), and (iii) after conversion of the presumptive myoblast into a muscle cell synthesis of myofibrillar actin begins, while synthesis of "cytoplasmic actin" continues (*17*) and synthesis of mitotic spindle actin presumably ceases. The existence of separate genes for each of the actins would permit the cell to exercise independent control over their synthesis, and in this way regulate the amount of actin available for each function; thus complex modulation of the activity of a single gene to provide the appropriate actins would not be required. The Val → Thr replacement that we describe here could be one of a limited number of substitutions that have occurred during parallel evolution of platelet and cardiac muscle actins.

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