

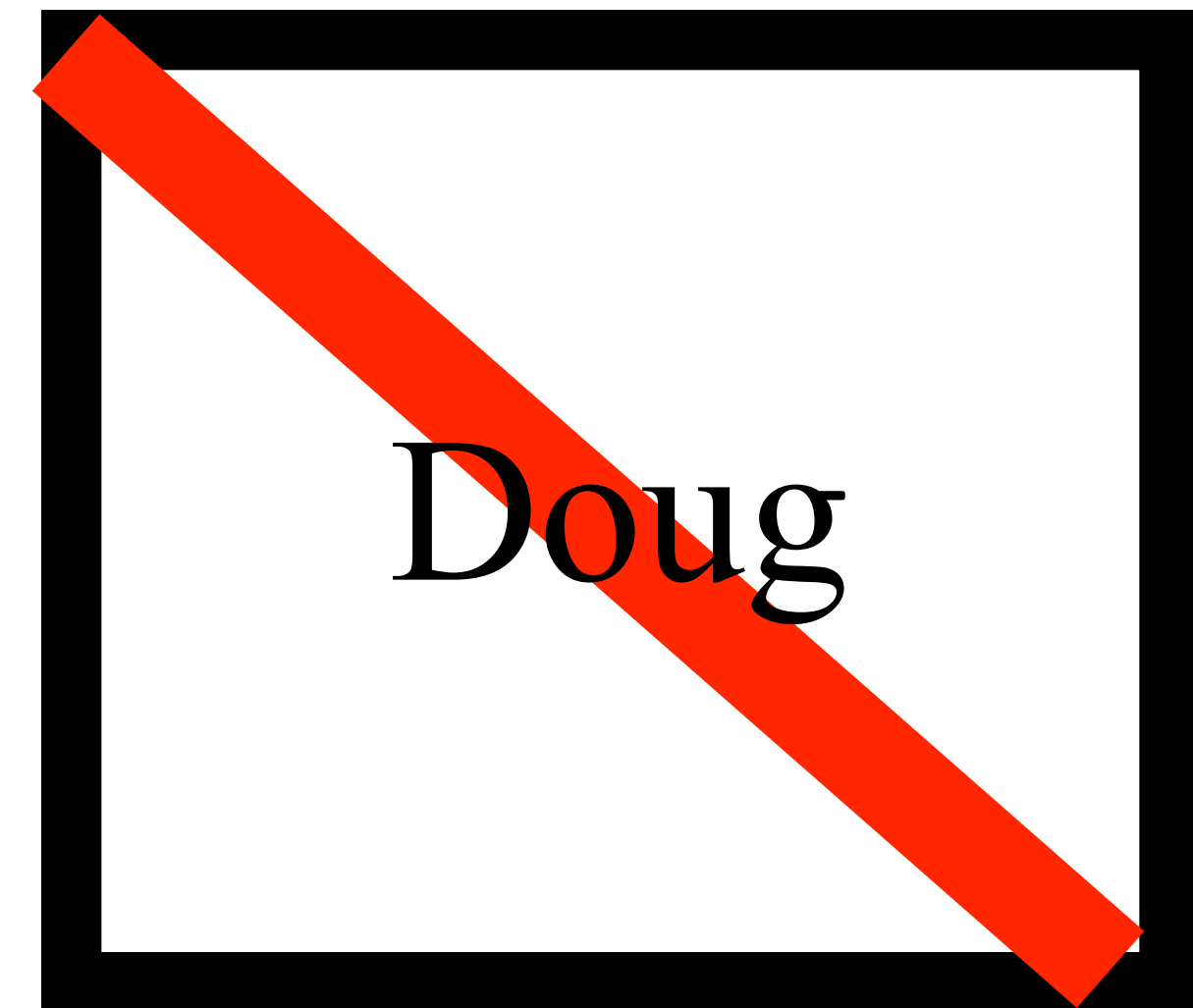
# 1. Clicker Attendance

- Launch your Top Hat app on your smart phone, or load the TopHat.com website, or text to the course phone number.

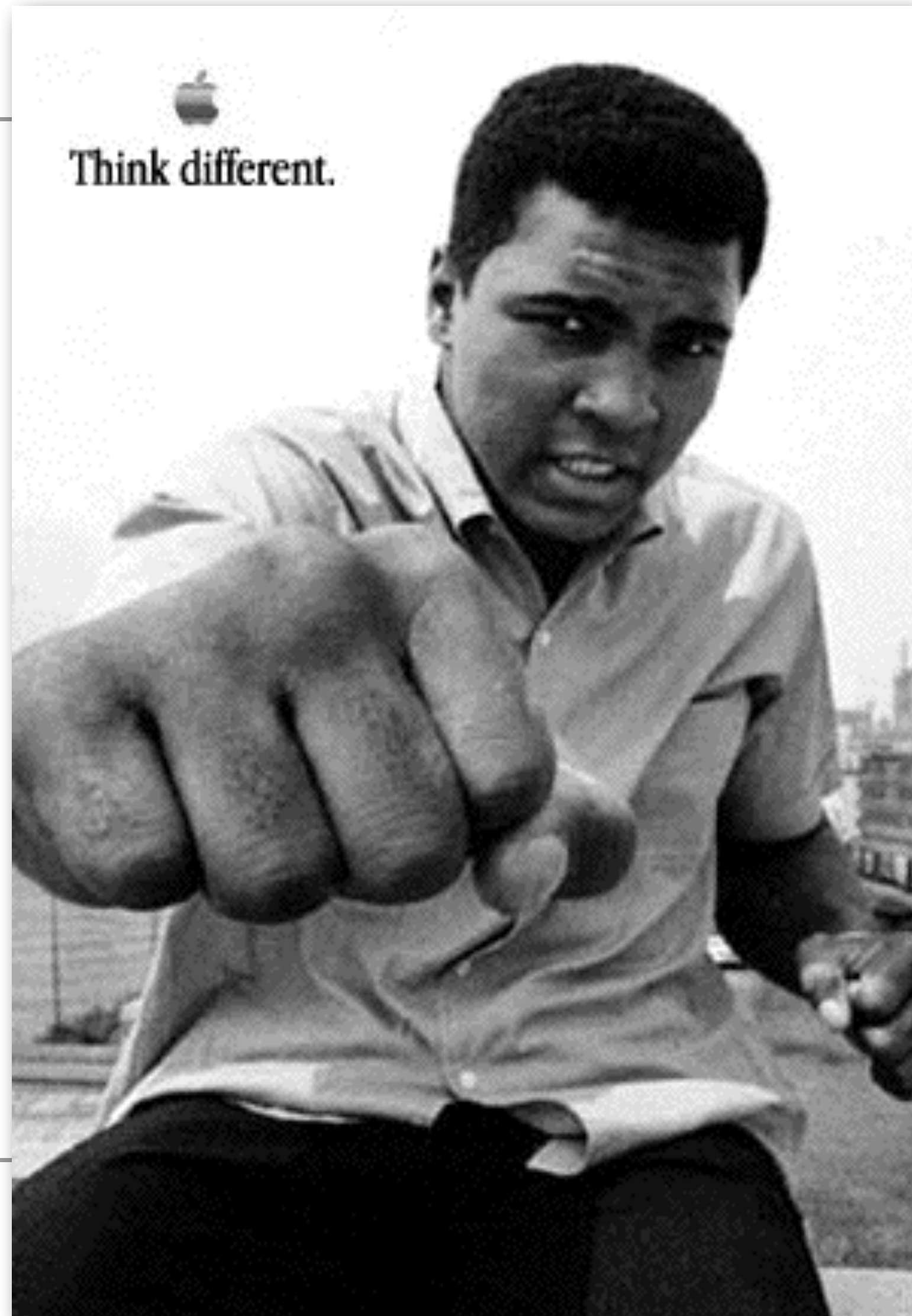
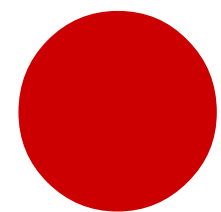
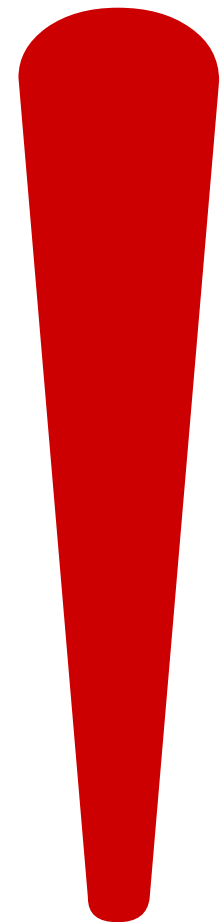
# 2. Sit with your group in lecture & lab

# 3. To Opt-OUT of being called upon

- Name Card with red stripe means you Opt-OUT (can Opt-OUT 3 times)



# LB144-Pandemic *2022 edition*





# **Remind me**

- 10 minutes left remind me to talk about Exam

## Section 16.1: What causes individual variation?

### **Biology Learning Objectives**

- Evaluate the processes by which variation is generated in organisms and how this affects information at the population level and natural selection.
- Differentiate between independent assortment and crossing over.



**Budgeting homework time (70 min):** Read the start of Chapter 16 and the first half of section 16.1. This is 2559 words with 6 figures; and 4 are data figures that require thinking and notetaking. Just reading the text will take 12 minutes. Yet the data figures are important. Of course, when done properly, when you pause to decipher each figure, try Integrating Questions, and take notes, this assignment will take you more like 70 minutes. **Special Allowance:** Your group can divide up the Trifectas for this lecture.

1. \_\_\_\_\_ **For In-person lecture, first** read the first cover page of Chapter 16. Look at the Chapter location in the textbook and the Learning Objectives. No notes are necessary here.
2. \_\_\_\_\_ **Then, slowly** read the first half of section 16.1 on genes & blood pressure that asks the question: What causes individual variation? You can stop reading when you get to the blue box with the title "Variation caused by the environment". Please carefully take written notes on this reading in your lecture notebook.
3. \_\_\_\_\_ **Try to answer at least one Integrating Question in each set.** As you read the ICB textbook always attempt to test yourself a little, answer at least one IQ in each set.
4. \_\_\_\_\_ (Trifecta): **Prepare to explain (aloud) Figures 16.2, 16.3, 16.4 and 16.5 in class.**  
\*Special Allowance today\*: If you wish your group can designate who will be responsible for each figure and thus split up the responsibility and reduce the load (Purpose, Methods, Findings).
5. \_\_\_\_\_ **Advanced:** Try to make sense of Table 16.1.



Chapter 16 (section 16.1) <sup>the Rise of genetics + genes soon Meiosis</sup> Evolution-themed  
What causes individual variation? → <sup>consider</sup> why = evolution?  
→ caused by mechanisms, which...

L.O.s  
→ Evaluate processes by which variation is generated in org  
→ Differentiate between independent assortment + crossing over

Intro  
This chapter focuses on individuals but <sup>this</sup> populations evolve. (w/ variation)  
Individuals have genes (genotypes) which result in traits (phenotype)  
Variation among individuals: eye color, height are phenotypes that can

• Heritable variation, mutations and independent assortment

Study #1

Fig 16.1 - Sir Francis Galton - 1889 UK

Purpose - Influence of parent height on offspring (heredity?)

Methods - Measure height of parents + children, plot data, best

Findings - Correlation but reduction <sup>lots variation</sup> - not perfect. Offspring of  
parent were generally shorter <sup>in extreme</sup> in population but taller than p

Phenotypic traits in humans height, eye color, skin color, have some her  
one or more genes impact phenotype. Also environment plays role, height  
changes w/ malnutrition or exposure to toxins.

Darwin + Galton were contemporaries + cousins. Galton's path led  
to infancy as early founder of Eugenics movement in Europe.

Chapter 16 (cont.) section 16.1

Study #2 Giuseppe Bianchi et al 1994 - Milan

Much variation is caused by variation in DNA sequences.

Fig 16.2 - like in humans some rats have high BP (→  
systolic + diastolic levels)

Purpose - develop colonies of similar animals to determine  
"inbreeding" <sup>Q! who is this reminding</sup>

Methods - slowly over time breed specimens with low BP  
Perform breeding over 85 generations (how low?)  
Test 20 rats from each colony for genes / DNA

Findings → high BP colony had similar phenotypes across  
and homozygous for adducin genes  $\alpha^Y$  +  
"Milan hypertensive strain" (MHS) Low BP colony had similar phenotypes (i.e.  
MNS normative only homozygous for  $\alpha^F$  adducin gene (two point mutations in membrane skeleton)

Popcorn Reading of Abstract from Bianchi paper

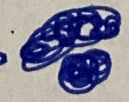
Adducin = proteins form heteroduplex <sup>Q!</sup> = means  
on different chromosomes ( $\alpha\beta$ ) →  two different proteins

Figure 16.3 - DNA sequences of  $\alpha$  adducin +  $\beta$   
point mutations that lead to  $\Delta$  BP.  
 $A = B$  <sup>sub optimal</sup>  $\leftarrow$  oops  
 $B = \alpha$

Purpose + Methods + Finding derived from those

found  $\alpha$  adducin w/  $Y = \alpha^Y$  w/  $F = \alpha^F$ ;  $\therefore \beta^R, \beta$

low BP colony had genotypes of:  $\alpha^F: \beta$  <sup>homozygous R</sup>  
<sub>heterozygous</sub>  
<sub>homozygous</sub>

Figure 16.4 - test BP + compare from rats w/ low BP  
Find: all lower than high BP but Q/R mix low

Chapter 16 (cont.) section 16.1

Study #3 Bianchi 1994

Figure 16.5 - Cross breeding genotype leads to BP?

Purpose - does BP phenotype align and follow genes/genotype?  
<sup>P = parents</sup>

Methods - cross breed HBP + Low BP rats - check BP of  $(F_1)$   
 $\alpha^Y \beta^R \times \alpha^F \beta^Q \Rightarrow$  all same genes <sup>now offspring</sup>  $\alpha^Y \alpha^F \beta^R \beta^Q$   
then cross-breed children → check BP grandchildren  $(F_2)$

Findings - second generation rats never as high BP as parents  
nor as low as low BP parents <sup>Q! sound familiar?</sup>  
Much variation (because other genes involved in BP,  
and outside colony environment too)

Independent assortment <sup>non-homologous</sup>  $\alpha$  vs  $\beta$  free to shuffle not linked  
(or recombination) since on separate chromosomes in meiosis

Crossing over <sup>homologous</sup> when  $\alpha + \alpha$  alleles slam into each other  
+ exchange DNA pieces during meiosis.  
other genes + environment → variation great (aka Galton)

What's this look like in a cell? (which cells express adducin?)



# Chapter 16: Variation and Population Genetics

Look around your classroom – how many people in the room look just like you? What is the makeup of the class in terms of hair, eye or skin color? What about height? You will find that there is some variation in all of these traits. Much of that variation has a genetic component and all of the variation relates to information. In this chapter you will consider information at the level of the individual, first by investigating the causes of variation among individuals, then by examining how genetic information within individuals plays out at the population level. You will learn how genetic and environmental changes have led to variation within species.

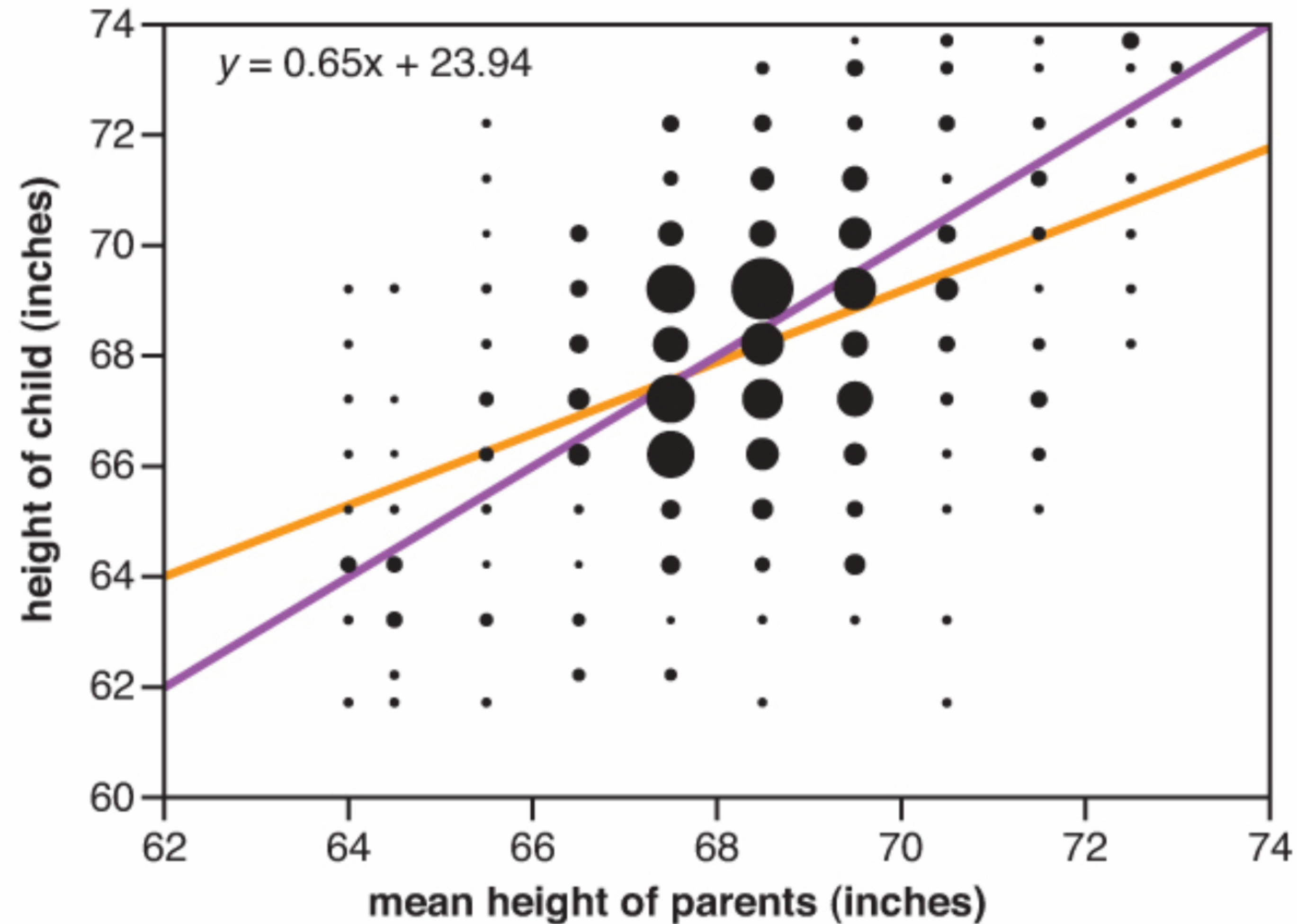


The barnacle Chthamalus attaches to a hard substrate of these are, and one shape environment triggers the g Lively, Bloomington, IN.

you are here		Big Ideas of biology				
		Information	Evolution	Cells	Homeostasis	Emergent Properties
levels of the biological hierarchy	molecules	1	4	7	10	13
	cells	2	5	8	11	14
	organisms I	3	6	9	12	15
	organisms II	16	19	22	28	25
	populations	17	20	23	29	26
	ecological systems	18	21	24	30	27



## POP Trifecta



**Figure 16.1** Data showing the relationship between height of parents and offspring. The purple line indicates a slope of one and the orange line indicates the best-fit line indicated by the equation. Size of circles is proportional to the number of comparisons. Total sample size = 928 offspring and 205 sets of parents. Redrawn with data from Galton, 1889.



## Bio-Math Exploration 16.1: How does linear regression work?

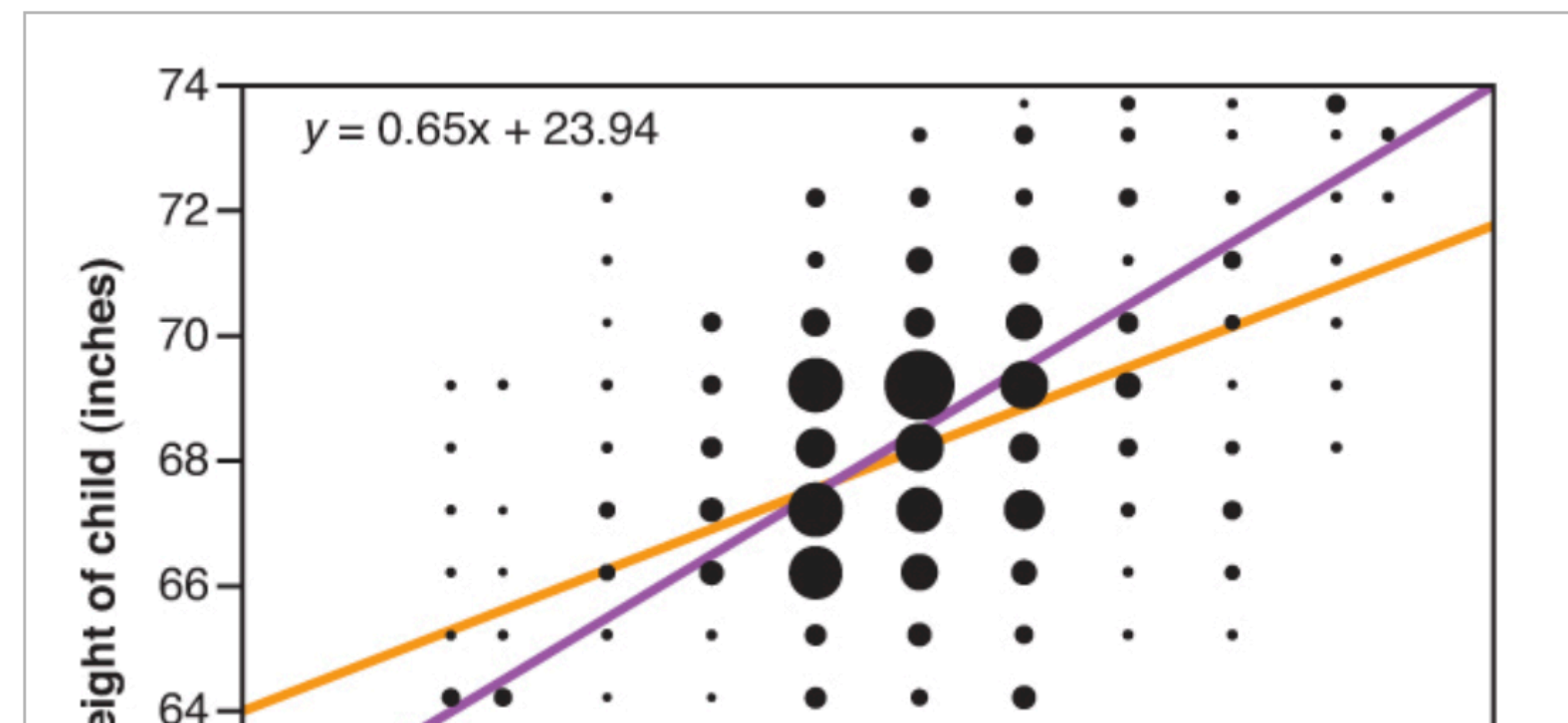
*The goal of this Bio-Math Exploration is to show you how to find the best-fit line for data like that in Figure 16.1, which can be used to represent the data in a compact way and predict future observations. You need to understand the concepts of slope and intercept. You will learn how to find the best-fit line with Excel.*

### Bio-Math Learning Objective

- Discover how linear regression works and apply it to an evolutionary question.

The relationship between variables is at the very heart of science. A scientist makes and tests hypotheses about the effect of one variable on another, and good scientists try to quantify the effects that they are studying. Sir Francis Galton wanted to quantify the effect of parental height on the height of their offspring. He invented the concept of linear **regression** to help quantify this relationship. Today, you can simply enter the data in a spreadsheet, plot it, and ask the software to find the best-fit line. Many scientists do linear regression this way without understanding how or why it works, but good scientists understand their methods before they use them. This Bio-Math Exploration discusses the mathematical principles that are behind the method of linear regression.

The [interactive model](#) below shows all 928 data points in Galton's study. On the x-axis is the "midparent" height, which is the average of the father's height and 1.08 times the mother's height. The factor of 1.08 is used to put male and female heights on the same scale, so that it makes sense to average the two numbers. *{Connections: BME 17.1 uses a similar idea of scaling for firefly signaling times.}* On the y-axis are the heights of their fully-grown adult offspring. There are only 205 different sets of parents represented in the data, so most parents have more than



# Relationship between height of parents and offspring

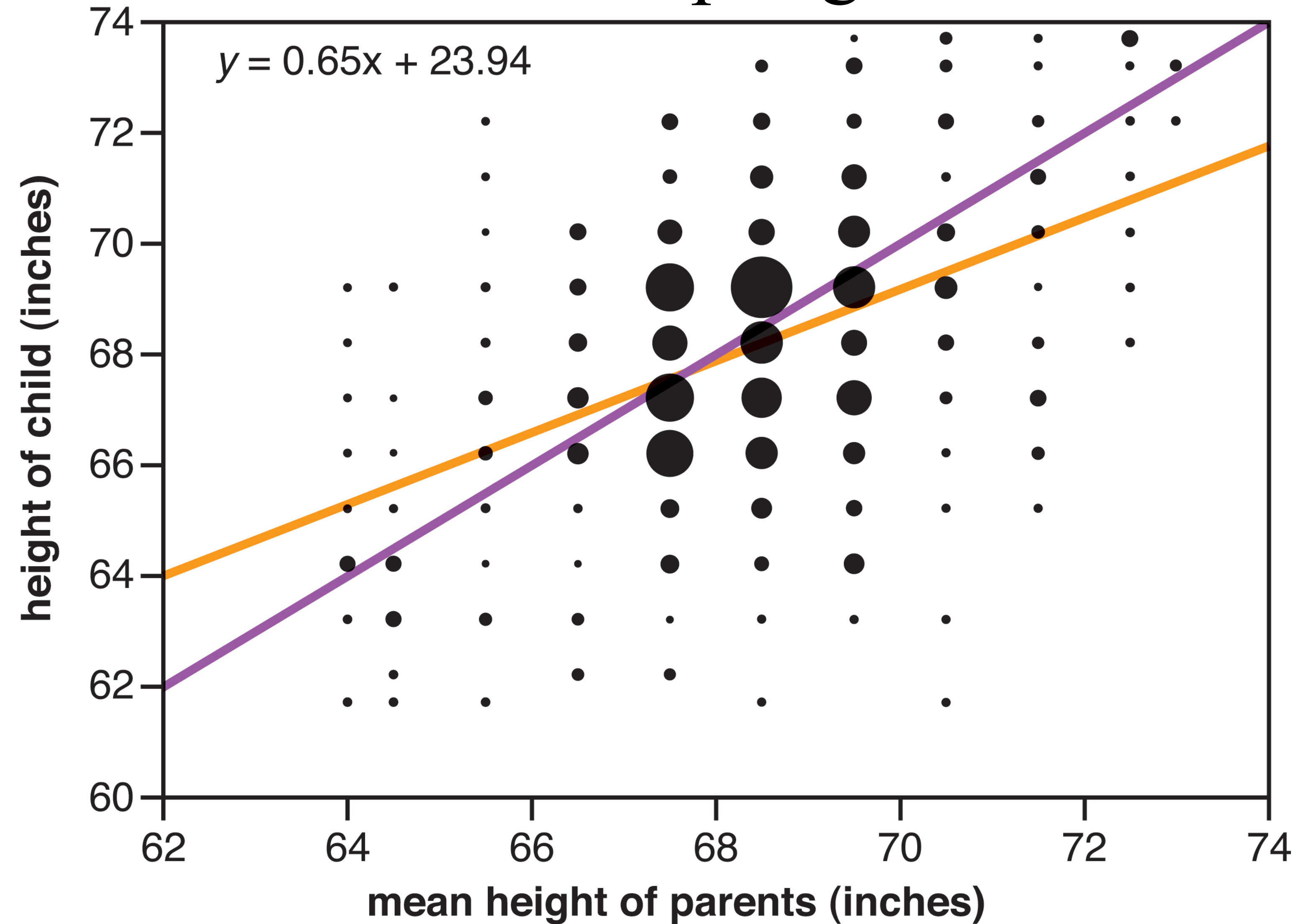


Figure 16.1

Redrawn with data from Galton, 1889.



# Relationship between height of parents and offspring

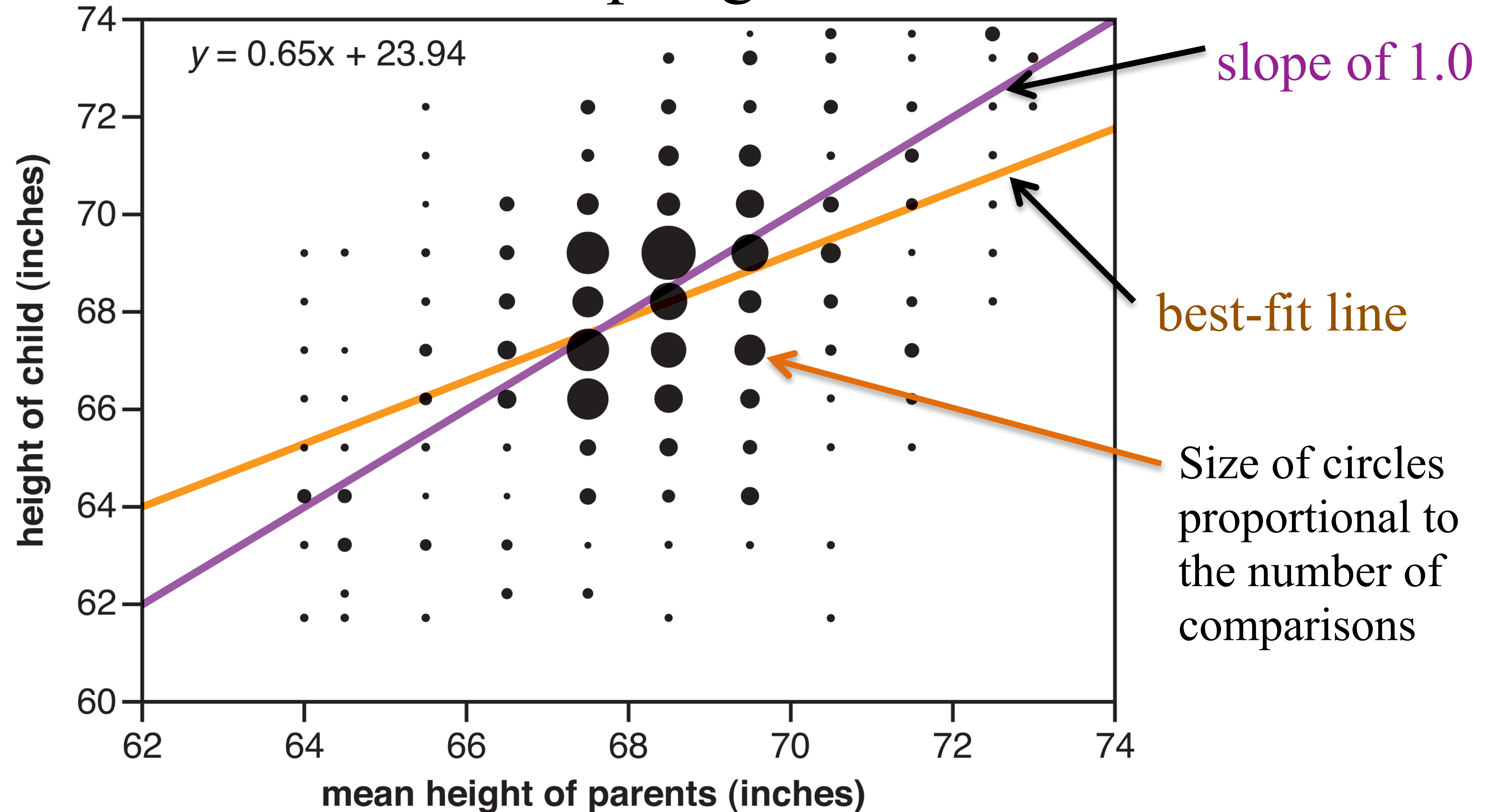


Figure 16.1

Redrawn with data from Galton, 1889.

# NATURAL INHERITANCE

BY

FRANCIS GALTON, F.R.S.

AUTHOR OF

"HEREDITARY GENIUS," "INQUIRIES INTO HUMAN FACULTY," ETC.

8 of 266

## NATURAL INHERITANCE.

### CHAPTER I.

#### INTRODUCTORY.

I HAVE long been engaged upon certain problems that lie at the base of the science of heredity, and during several years have published technical memoirs concerning them, a list of which is given in Appendix A. This volume contains the more important of the results, set forth in an orderly way, with more completeness than has hitherto been possible, together with a large amount of new matter.

The inquiry relates to the inheritance of moderately exceptional qualities by brotherhoods and multitudes rather than by individuals, and it is carried on by more refined and searching methods than those usually employed in hereditary inquiries.

One of the problems to be dealt with refers to the curious regularity commonly observed in the statistical peculiarities of great populations during a long series of

B

generations. The large do not always beget the large, nor the small the small, and yet the observed proportions between the large and the small in each degree of



TABLE 9B.  
MARRIAGES OF THE ARTISTIC AND THE NOT ARTISTIC.

Rank in Pedigrees.	No. of persons.	Percentages.									
		Males.		Females.		Pairs of artistic and not artistic persons.					
						Marriages observed.			Chance combinations.		
		art.	not.	art.	not.	both art.	1 art. 1 not.	both not.	both art.	1 art. 1 not.	both not.
Parents .....	326	32	68	39	61	14	31	50	12	46	42
Paternal grandparents..	280	27	73	30	70	12	31	57	8	41	51
Maternal grandparents..	288	24	76	28	72	9	41	50	7	39	54
Totals and means...	894	28	72	33	67	12	36	52	9	42	49
Tastes of Husband and Wife—alike .....						12 + 52 = 64			9 + 49 = 58		
" " " contrasted.....						36			42		

TABLE 10.  
EFFECT UPON ADULT CHILDREN OF DIFFERENCES IN HEIGHT OF THEIR PARENTS.

Difference in inches between the Heights of the Parents.	Proportion per 50 of cases in which the Heights <sup>1</sup> of the Children deviated to various amounts from the Mid-filial Stature of their respective families.					Number of Children whose Heights were observed. (Total 525.)
	Less than 1 inch.	Less than 2 inches.	Less than 3 inches.	Less than 4 inches.	Less than 5 inches.	
Under 1 inch .....	21	35	43	46	48	105
1 and under 2 .....	23	37	46	49	50	122
2 " 3 .....	16	34	41	45	49	112
3 " 5 .....	24	35	41	47	49	108
5 and above.. .....	18	30	40	47	49	78

<sup>1</sup> Every female height has been transmuted to its male equivalent by multiplying it by 1.08, and only those families have been included in which the number of adult children amounted to six, at least.

NOTE.—When these figures are protracted into curves, it will be seen—(1) that they run much alike; (2) that their peculiarities are not in sequence; and (3) that the curve corresponding to the first line occupies a medium position. It is therefore certain that differences in the heights of the Parents have on the whole an inconsiderable effect on the heights of their Offspring.

TABLE 11 (R.F.F. Data).  
NUMBER OF ADULT CHILDREN OF VARIOUS STATURES BORN OF 205 MID-PARENTS OF VARIOUS STATURES.  
(All Female Heights have been multiplied by 1.08.)

Height of the mid-parents in inches.	Heights of the adult children.														Total number of		Medians or Values of M.
	Below	62.2	63.2	64.2	65.2	66.2	67.2	68.2	69.2	70.2	71.2	72.2	73.2	Above.	Adult children.	Mid-parents.	
Above 72.5...	...	...	...	...	...	...	...	...	...	...	...	1	3	...	4 <sup>1</sup>	5 <sup>1</sup>	72.2 69.9 69.5 68.9 68.2 67.6 67.2 66.7 65.8
72.5...	...	...	...	...	...	...	...	1	2	1	2	7	2	4	19	6	
71.5...	...	...	...	...	1	3	4	3	5	10	4	9	2	2	43	11	
70.5...	1	...	1	...	1	1	3	12	18	14	7	4	3	3	68	22	
69.5...	...	...	1	16	4	17	27	20	33	25	20	11	4	5	183	41	
68.5...	1	...	7	11	16	25	31	34	48	21	18	4	3	...	219	49	
67.5...	...	3	5	14	15	36	38	28	38	19	11	4	...	...	211	33	
66.5...	...	3	3	5	2	17	17	14	13	4	...	...	...	...	78	20	
65.5...	1	...	9	5	7	11	11	7	7	5	2	1	...	...	66	12	
64.5...	1	1	4	4	1	5	5	...	2	...	...	...	...	...	23	5	
Below .....	1	...	2	4	1	2	2	1	1	...	...	...	...	...	14	1	
Totals .....	5	7	32	59	48	117	138	120	167	99	64	41	17	14	928	205	
Medians .....	...	...	66.3	67.8	67.9	67.7	67.9	68.3	68.5	69.0	69.0	70.0					

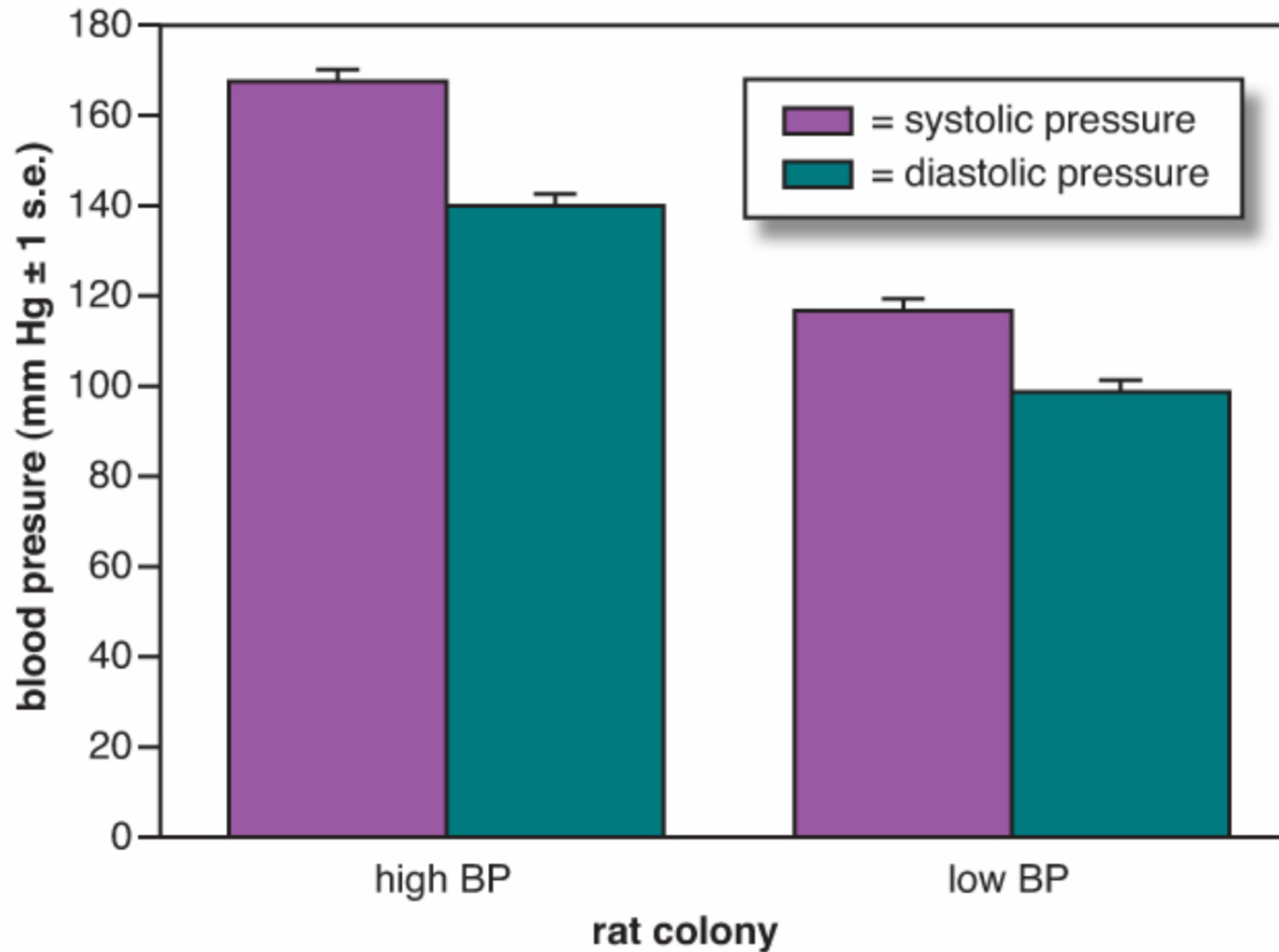
Note.—In calculating the medians, the entries have been taken as referring to the middle of the squares in which they stand. The reason why the headings run 62.2, 63.2, &c., instead of 62.5, 63.5, &c., is that the observations are unequally distributed between 62 and 63, 63 and 64, &c., there being a strong bias in favour of integral inches. After careful consideration, I concluded that the headings, as adopted, best satisfied the conditions. This inequality was not apparent in the case of the mid-parents.

<sup>1</sup> I have reprinted this Table without alteration from that published in the *Proc. Roy. Soc.*, notwithstanding a small blunder since discovered in sorting the entries between the first and second lines. It is obvious that 4 children cannot have 5 Mid-Parents. The first line is not considered at all, on account of the paucity of the numbers it contains. The bottom line, which looks suspicious, is correct.

# Eugenics Movie



## Trifecta



**Figure 16.2** Mean blood pressures for rats in the two colonies, measured in millimeters of mercury. All high blood pressure rats were homozygous for the adducin genes ( $\alpha^Y$  and  $\beta^R$ ). Low blood pressure rats were all homozygous for the  $\alpha^F$  gene only. Data from Bianchi et al., 1994.

# Mean blood pressures for rats in two colonies

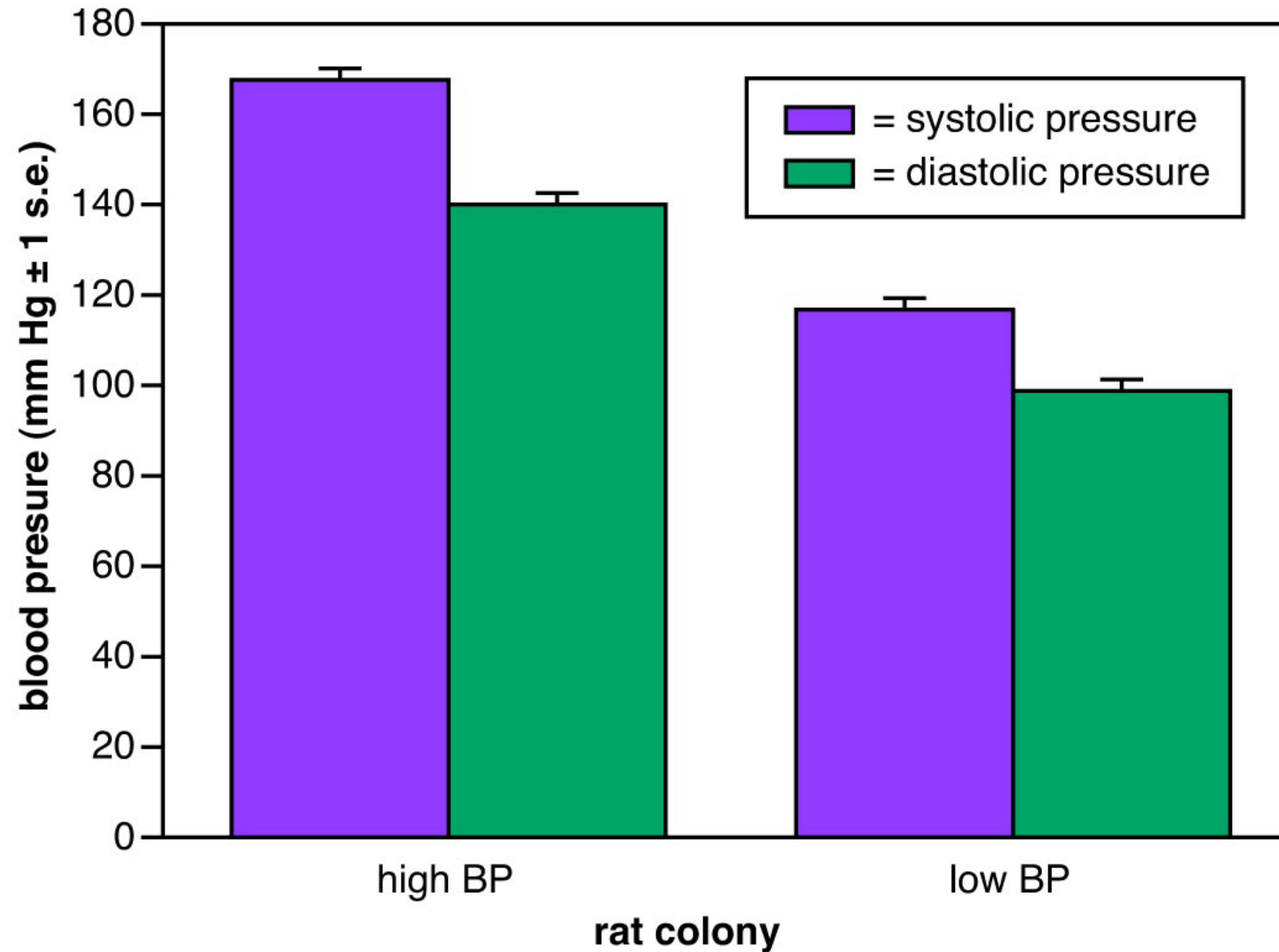


Figure 16.2

Data from Bianchi et al., 1994.



# Mean blood pressures for rats in two colonies

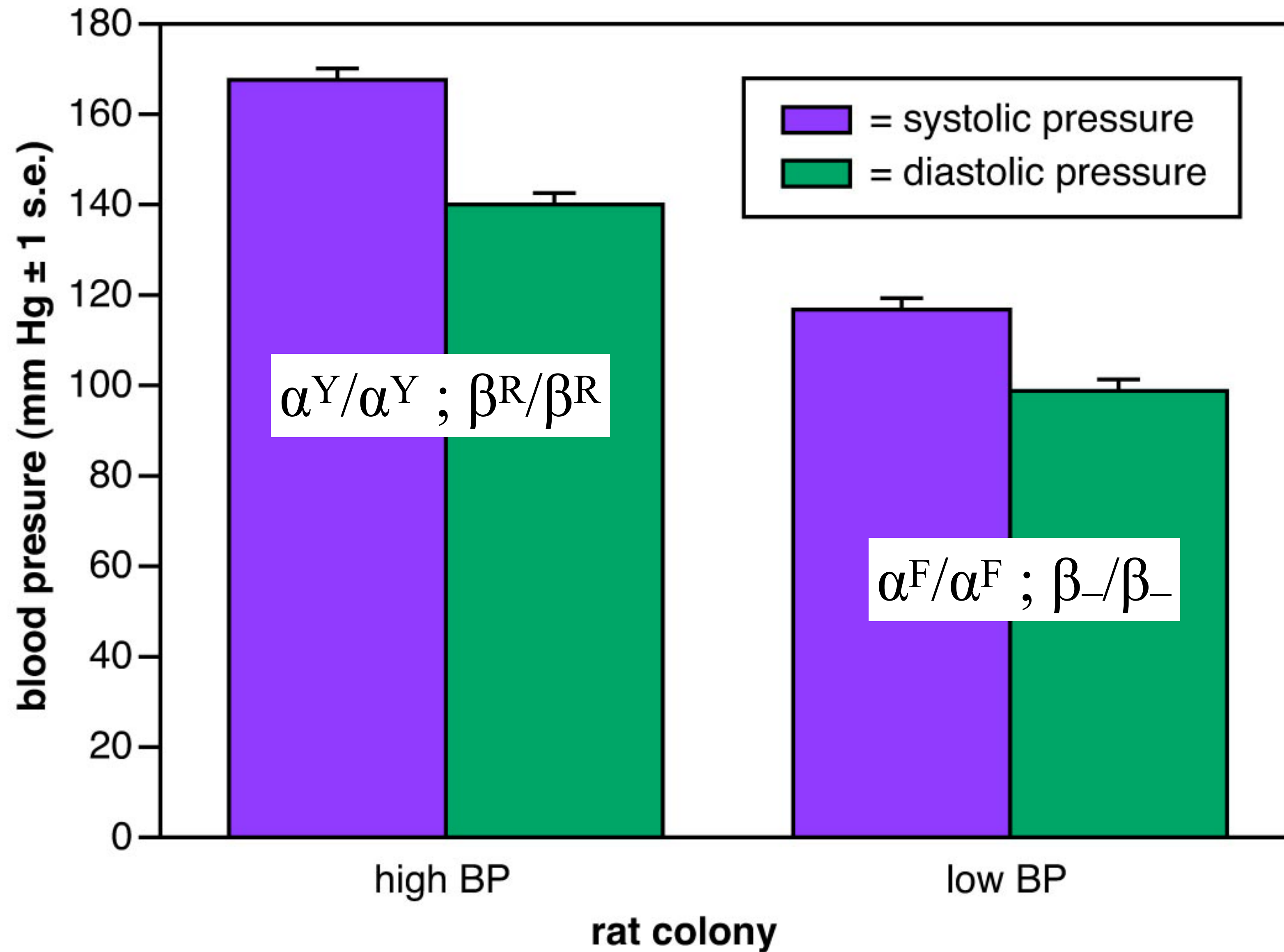
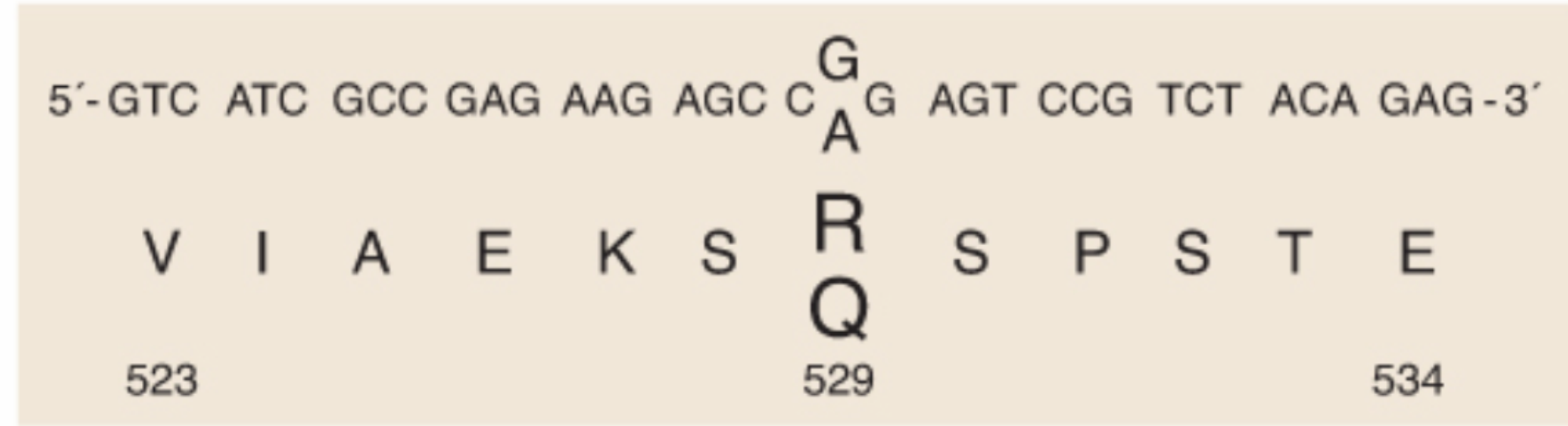


Figure 16.2

Data from Bianchi et al., 1994.

# Trifecta

## A $\beta$ -adducin



## B $\alpha$ -adducin



**Figure 16.3** Portions of the  $\beta$ -adducin (**A**) and  $\alpha$ -adducin (**B**) subunit DNA sequences (top row in each panel) and corresponding amino acid sequence. Numbers below the amino acid sequence indicate the position along the protein, and the letters correspond to different amino acids. At position 529 and 316 of  $\beta$  and  $\alpha$  subunits, respectively, there is a mutation. The top letter in each sequence corresponds to the allele associated with high blood pressure. From Bianchi et al., 1994, Figure 1, copyright (1994) National Academy of Sciences, U.S.A.



# Portions of the $\beta$ and $\alpha$ adducin subunit DNA sequences and corresponding amino acid sequence

letters  
correspond  
to different  
DNA bases

## A $\beta$ -adducin

5'-GTC ATC GCC GAG AAG AGC C<sup>G</sup><sub>A</sub>G AGT CCG TCT ACA GAG-3'

V I A E K S R S P S T E

523 529 534

letters  
correspond  
to different  
amino acids

## B $\alpha$ -adducin

5'-GTG GAG GAG GCC TTC T<sup>A</sup><sub>T</sub>T TAT ATC CAC AAC CTT GTG-3'

V E E A F Y Y I H N L V

311 316 322

position  
along the  
protein

Figure 16.3

From Bianchi et al., 1994, Figure 1, copyright (1994) National Academy of Sciences, U.S.A.



# Portions of the $\beta$ and $\alpha$ adducin subunit DNA sequences and corresponding amino acid sequence

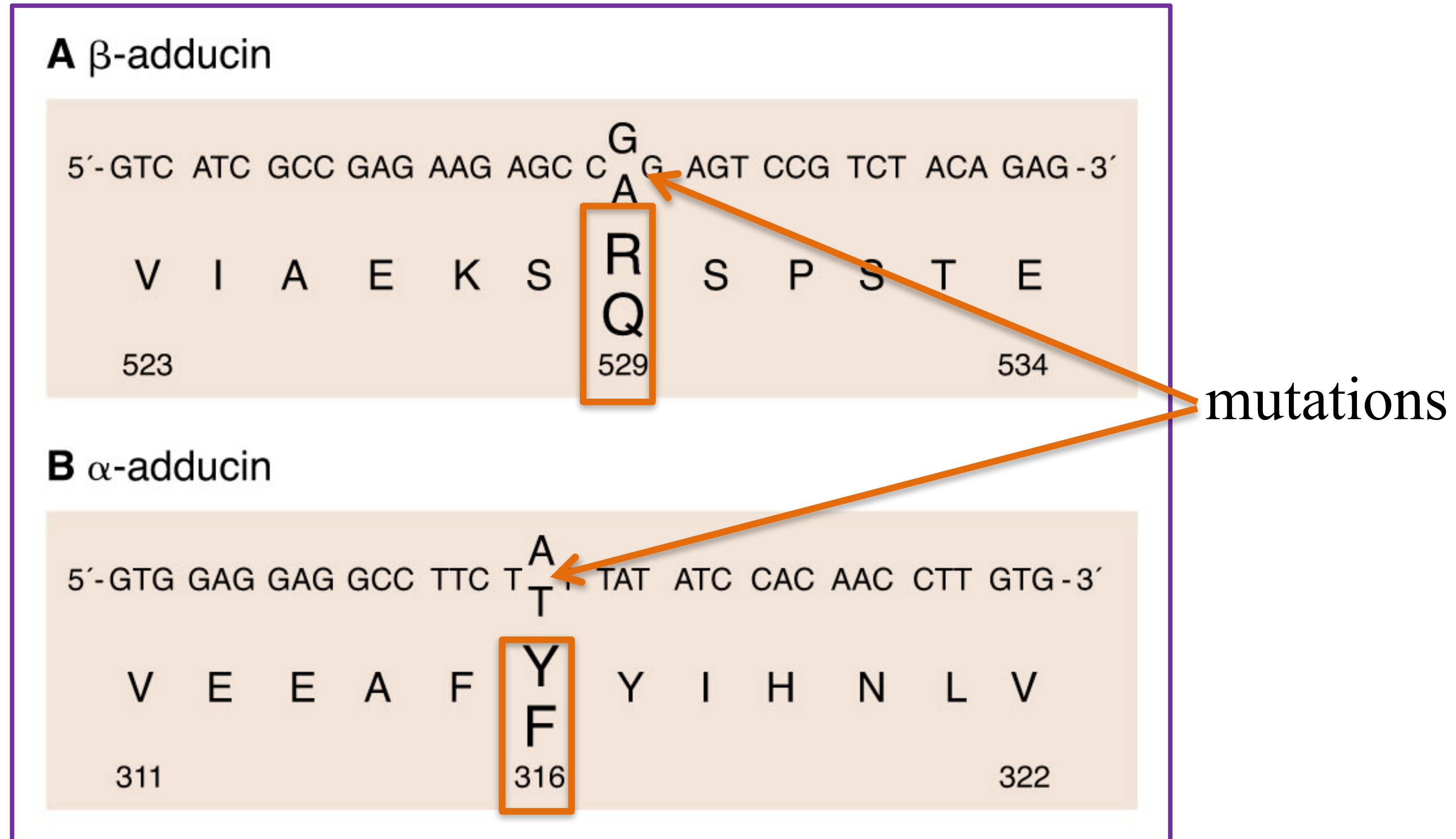


Figure 16.3

From Bianchi et al., 1994, Figure 1, copyright (1994) National Academy of Sciences, U.S.A.



# Portions of the $\beta$ and $\alpha$ adducin subunit DNA sequences and corresponding amino acid sequence

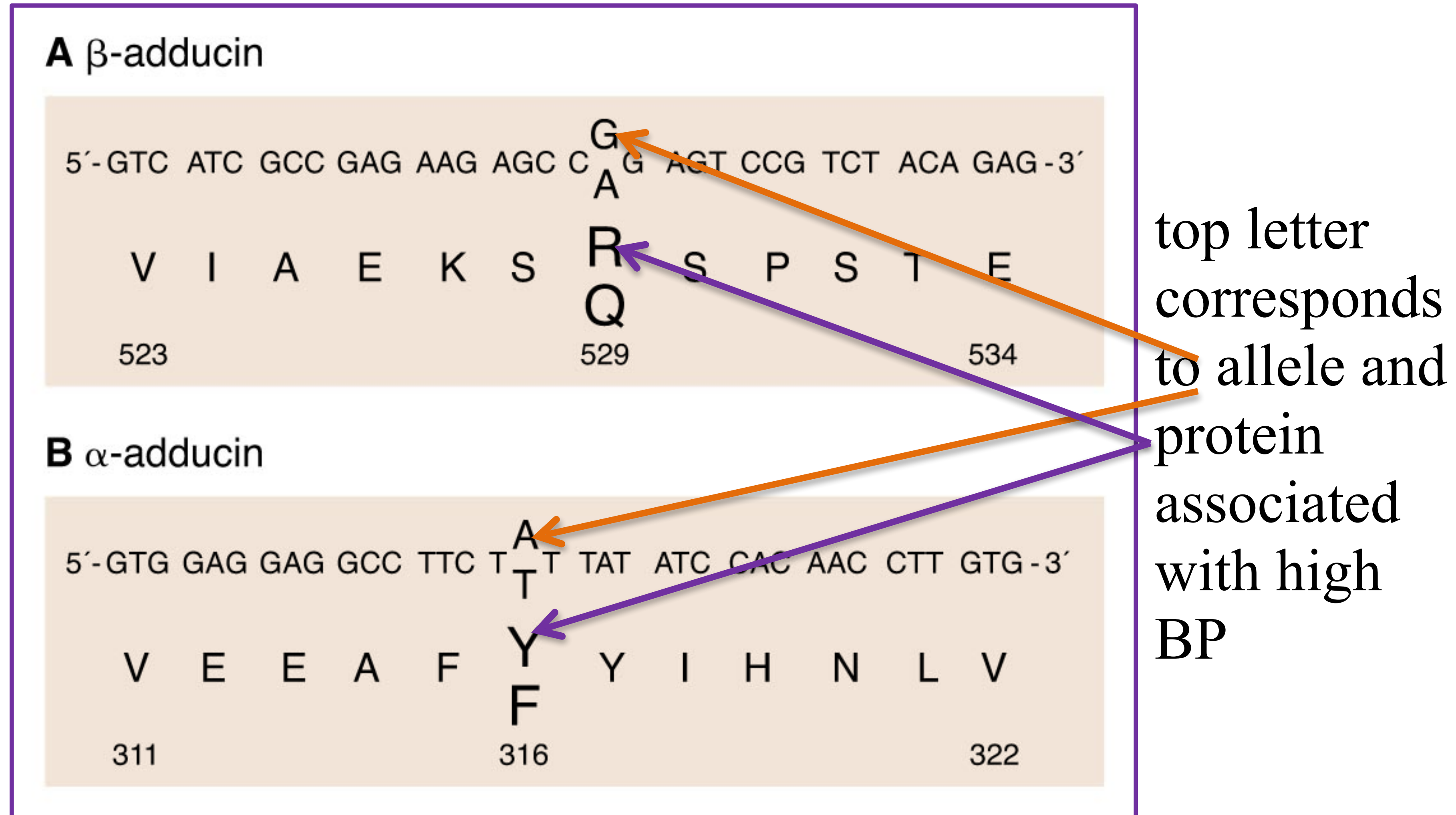


Figure 16.3

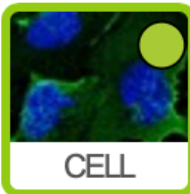
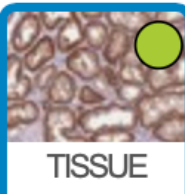
From Bianchi et al., 1994, Figure 1, copyright (1994) National Academy of Sciences, U.S.A.



Search

Fields »

# ADD1



TISSUE ATLAS

PRIMARY DATA

GENE/PROTEIN

ANTIBODIES  
AND  
VALIDATION



Dictionary



Tissue proteome



GENERAL INFORMATION<sup>i</sup>

Gene name <sup>i</sup>	ADD1
Gene description	Adducin 1
Protein class <sup>i</sup>	Plasma proteins
Predicted location <sup>i</sup>	Intracellular
Number of transcripts <sup>i</sup>	13

HUMAN PROTEIN ATLAS INFORMATION<sup>i</sup>

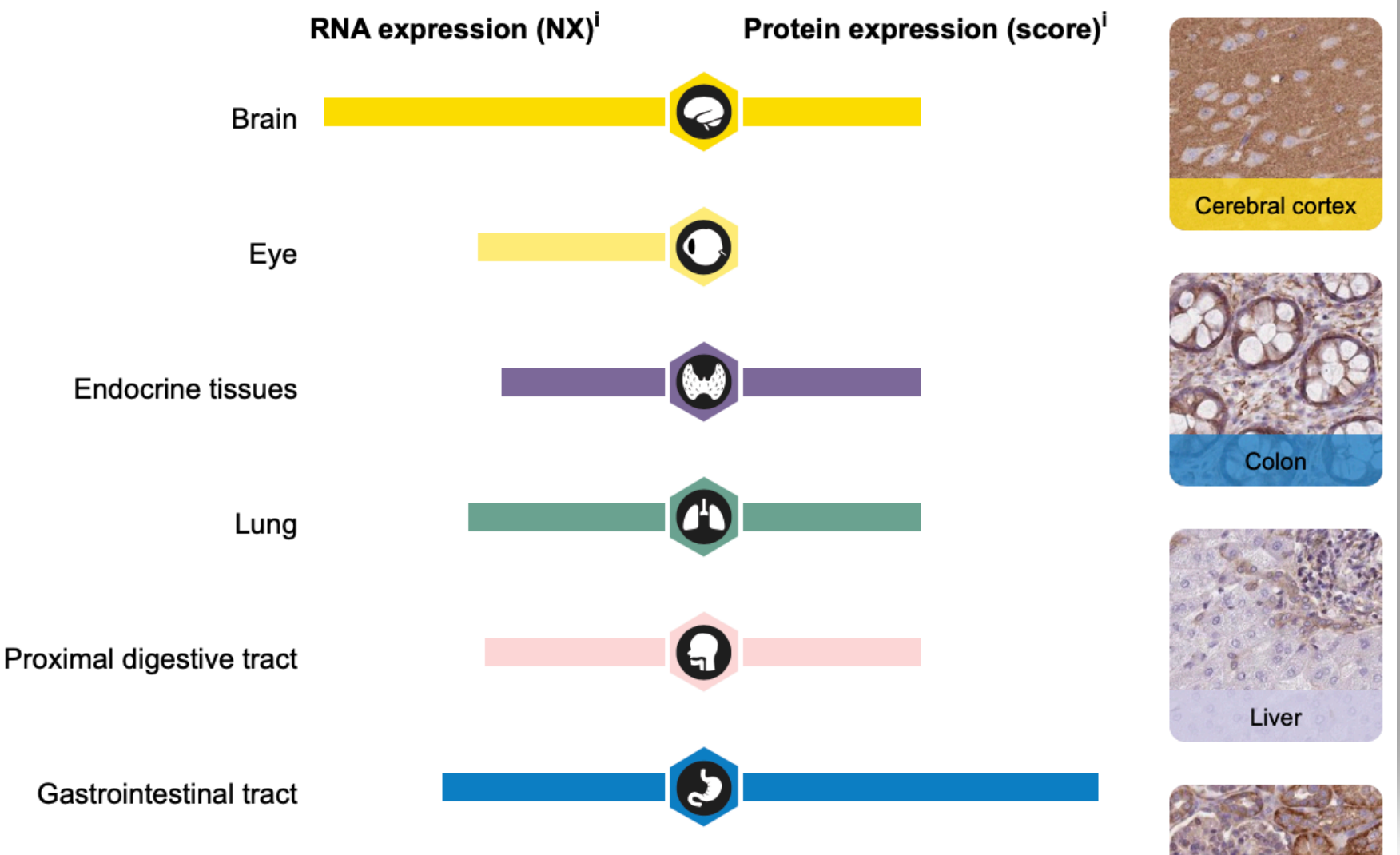
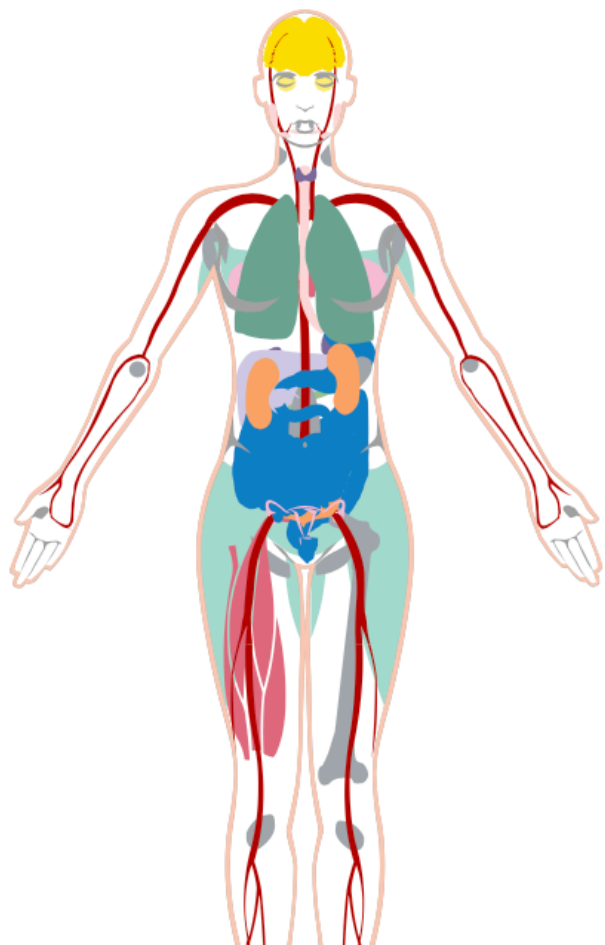
RNA tissue specificity <sup>i</sup>	Low tissue specificity
RNA tissue distribution <sup>i</sup>	Detected in all
Protein evidence <sup>i</sup>	Evidence at protein level
Protein expression <sup>i</sup>	Cytoplasmic and membranous expression in most tissues.

IMMUNOHISTOCHEMISTRY DATA RELIABILITY

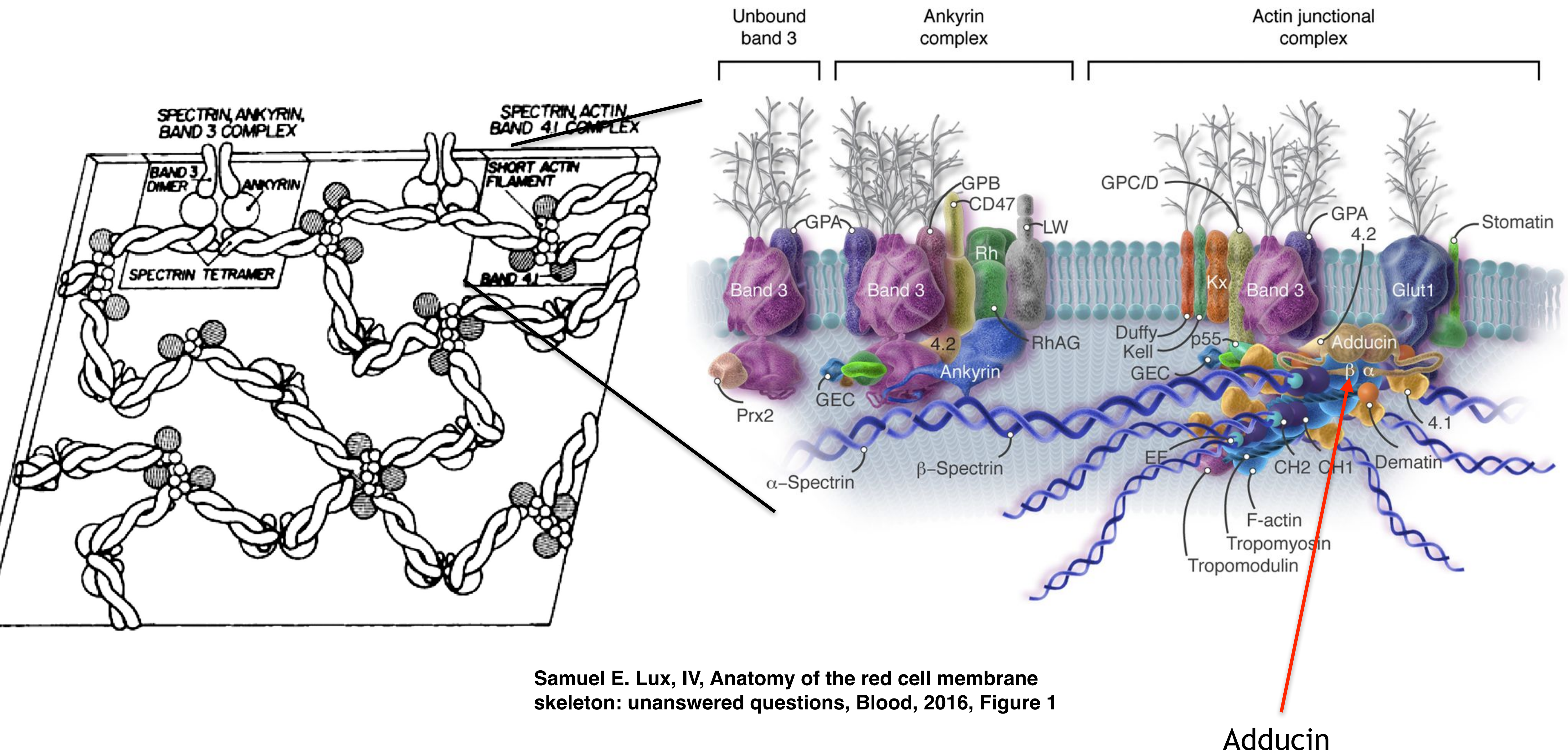
Data reliability description <sup>i</sup>	Antibody staining mainly consistent with RNA expression data.
Reliability score <sup>i</sup>	Enhanced
Antibodies <sup>i</sup>	<a href="#">HPA035873</a> , <a href="#">HPA035874</a> , <a href="#">CAB009794</a>

SHOW MORE

RNA AND PROTEIN EXPRESSION SUMMARY<sup>i</sup>





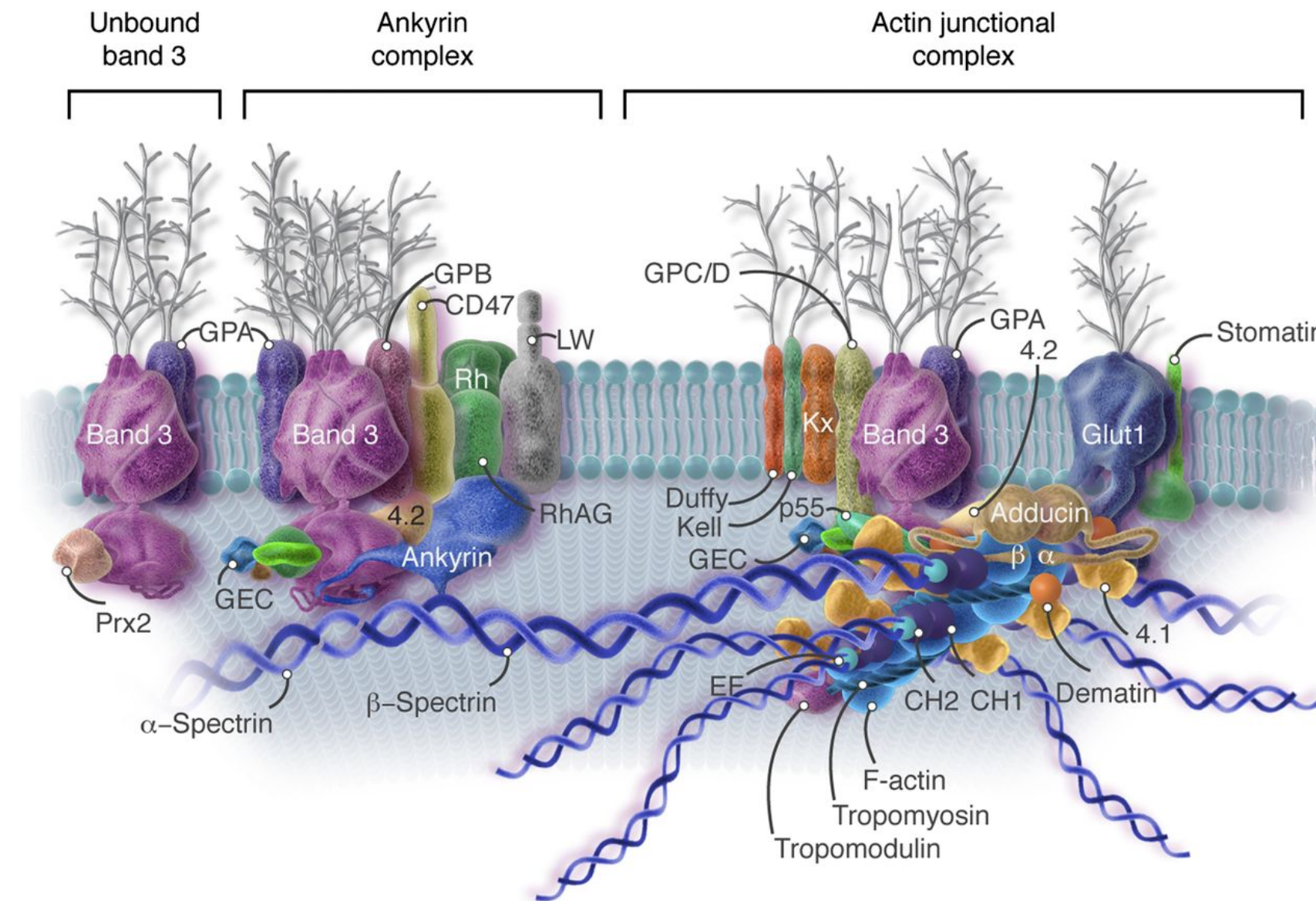


Samuel E. Lux, IV, Anatomy of the red cell membrane skeleton: unanswered questions, Blood, 2016, Figure 1



questions

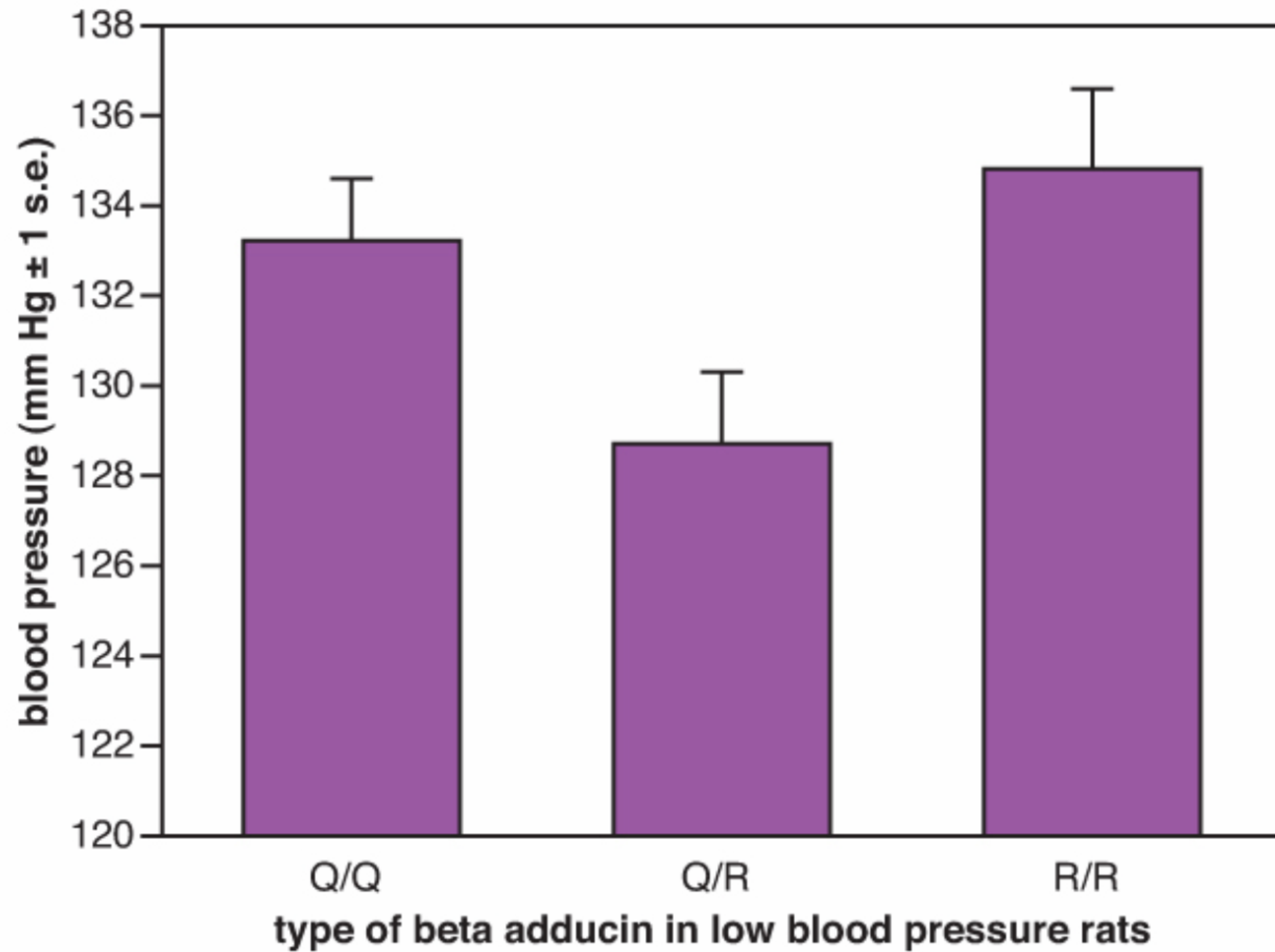
## Anatomy of the red cell membrane skeleton: unanswered



Samuel E. Lux, IV, Anatomy of the red cell membrane skeleton: unanswered questions, Blood, 2016, Figure 2



## Trifecta



**Figure 16.4** Mean systolic blood pressures of the three combinations of two versions of the  $\beta$  adducin gene in rats from the low blood pressure colony. Q and R refer to the amino acid present at position 529 on the  $\beta$  protein subunit of adducin. Error bars = 1 standard error (SE). Data from Bianchi et al., 1994.



Systolic BPs of the 3 combinations of 2 versions of the  $\beta$  adducin gene in rats from the low BP colony

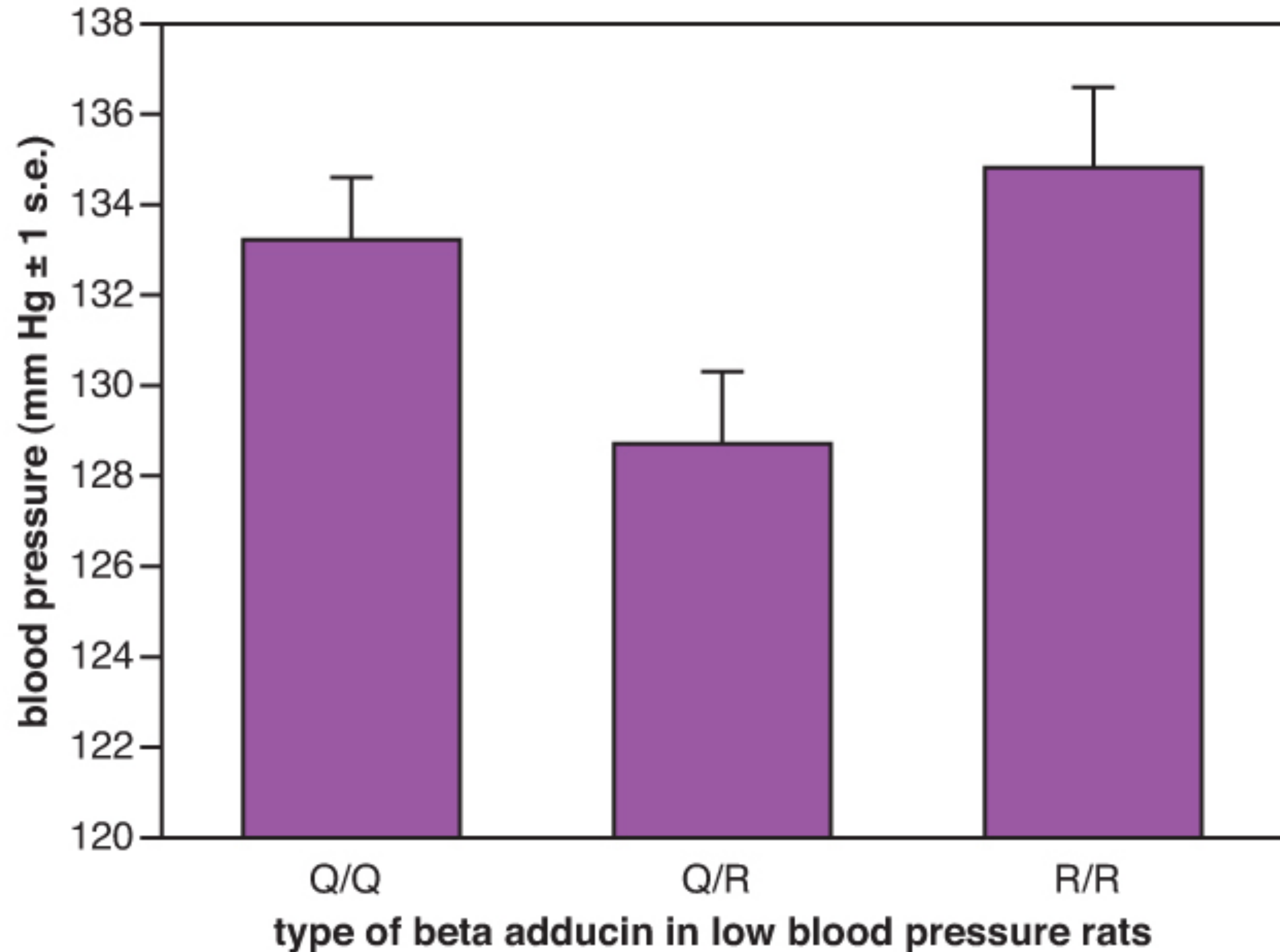


Figure 16.4

Data from Bianchi et al., 1994.



# Systolic BPs of the 3 combinations of 2 versions of the $\beta$ adducin gene in rats from the low BP colony

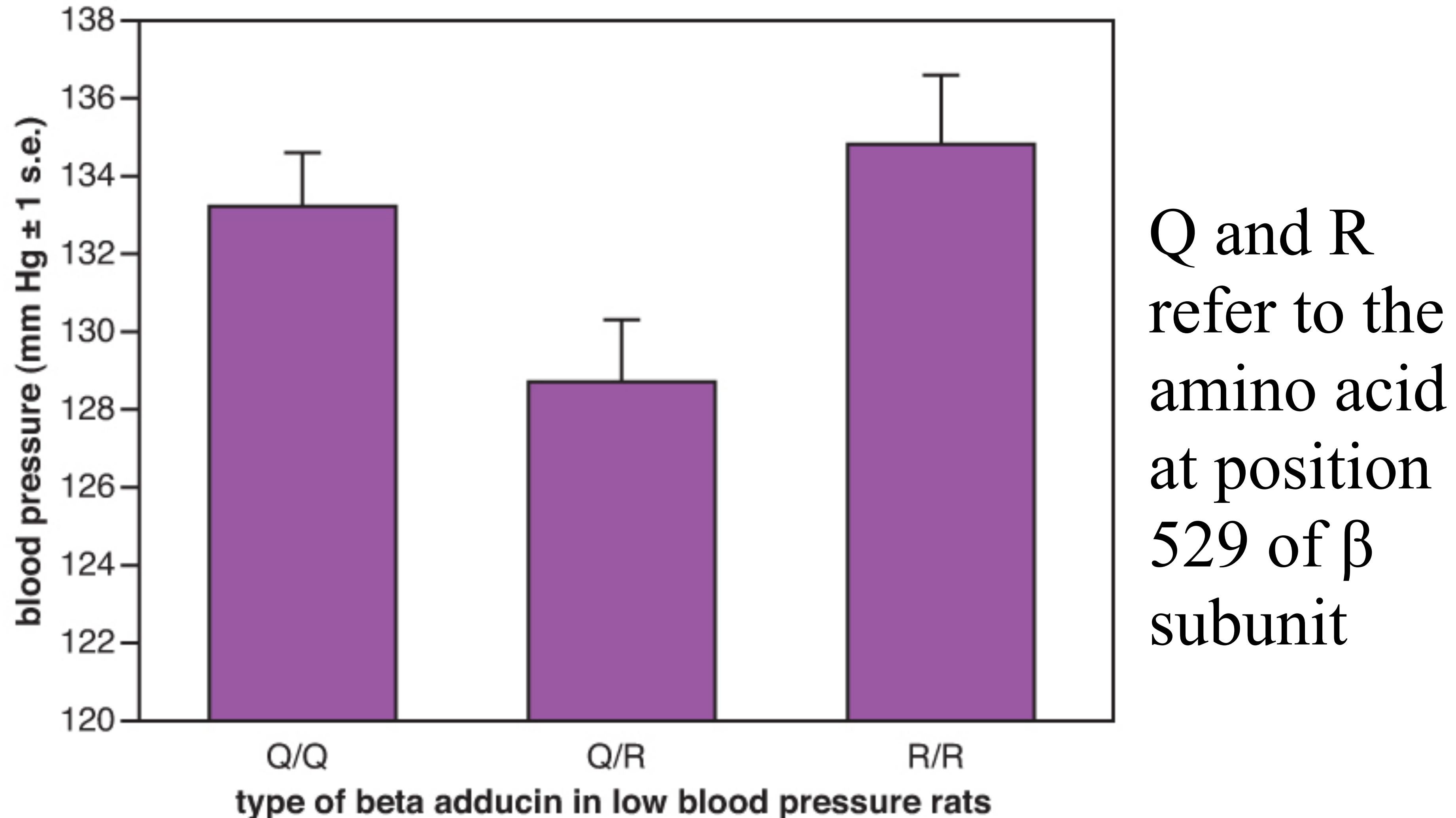
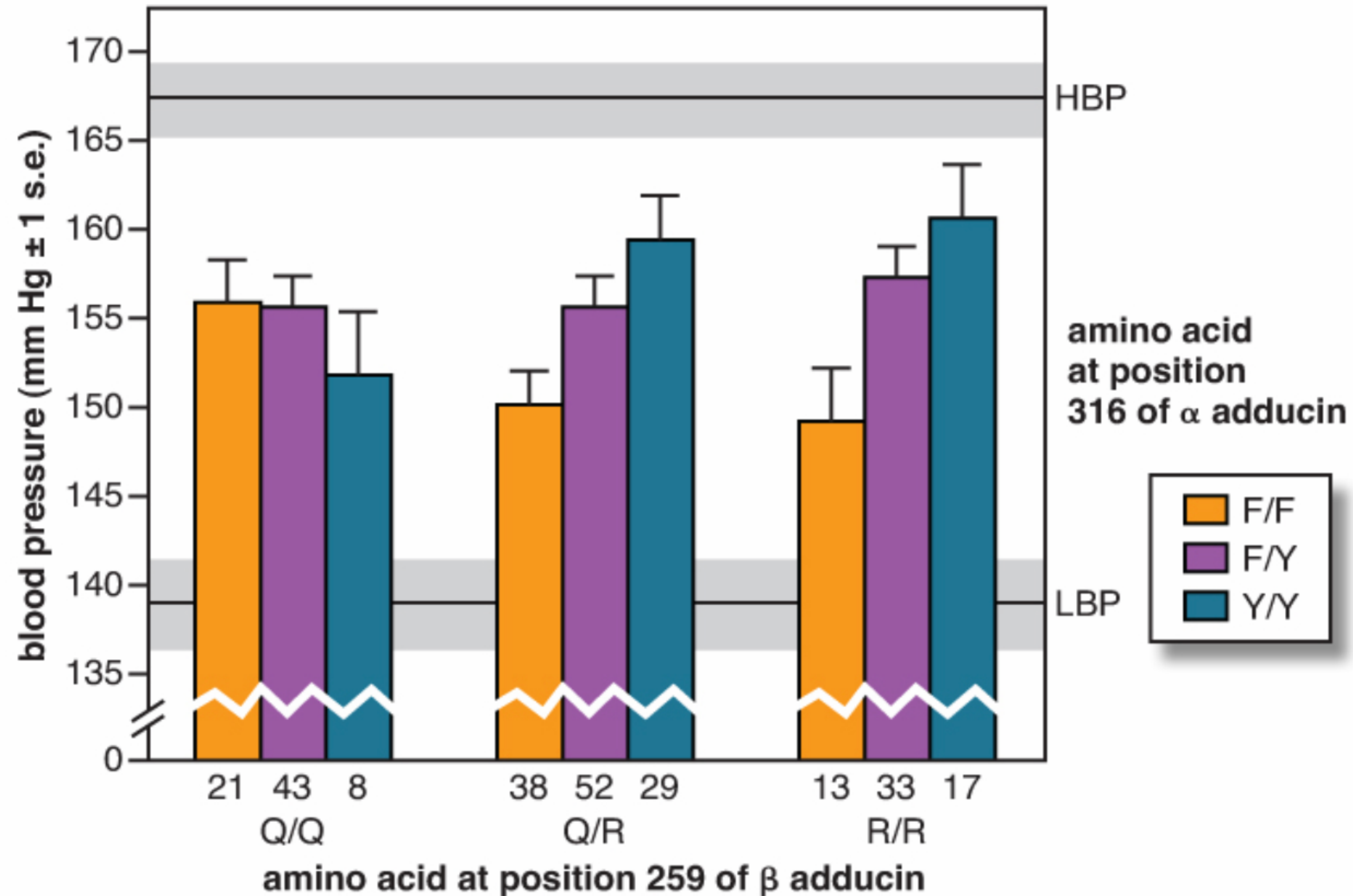


Figure 16.4

Data from Bianchi et al., 1994.



## Trifecta



**Figure 16.5** Mean blood pressures of the nine combinations of two versions of the  $\alpha$  and  $\beta$  adducin genes in rats after two generations of breeding low and high blood pressure rats together. Error bars = 1 SE. Mean blood pressure (horizontal solid lines)  $\pm$  1 SE (stippled areas) of 10 rats of each parental strain are included for comparison. HBP, High blood pressure; LBP, low blood pressure. From Bianchi et al., 1994, Figure 3, copyright (1994) National Academy of Sciences, U.S.A.



BP of 9 combinations of two versions of the  $\alpha$  and  $\beta$  adducin genes in rats after two generations of breeding low and high blood pressure rats together.

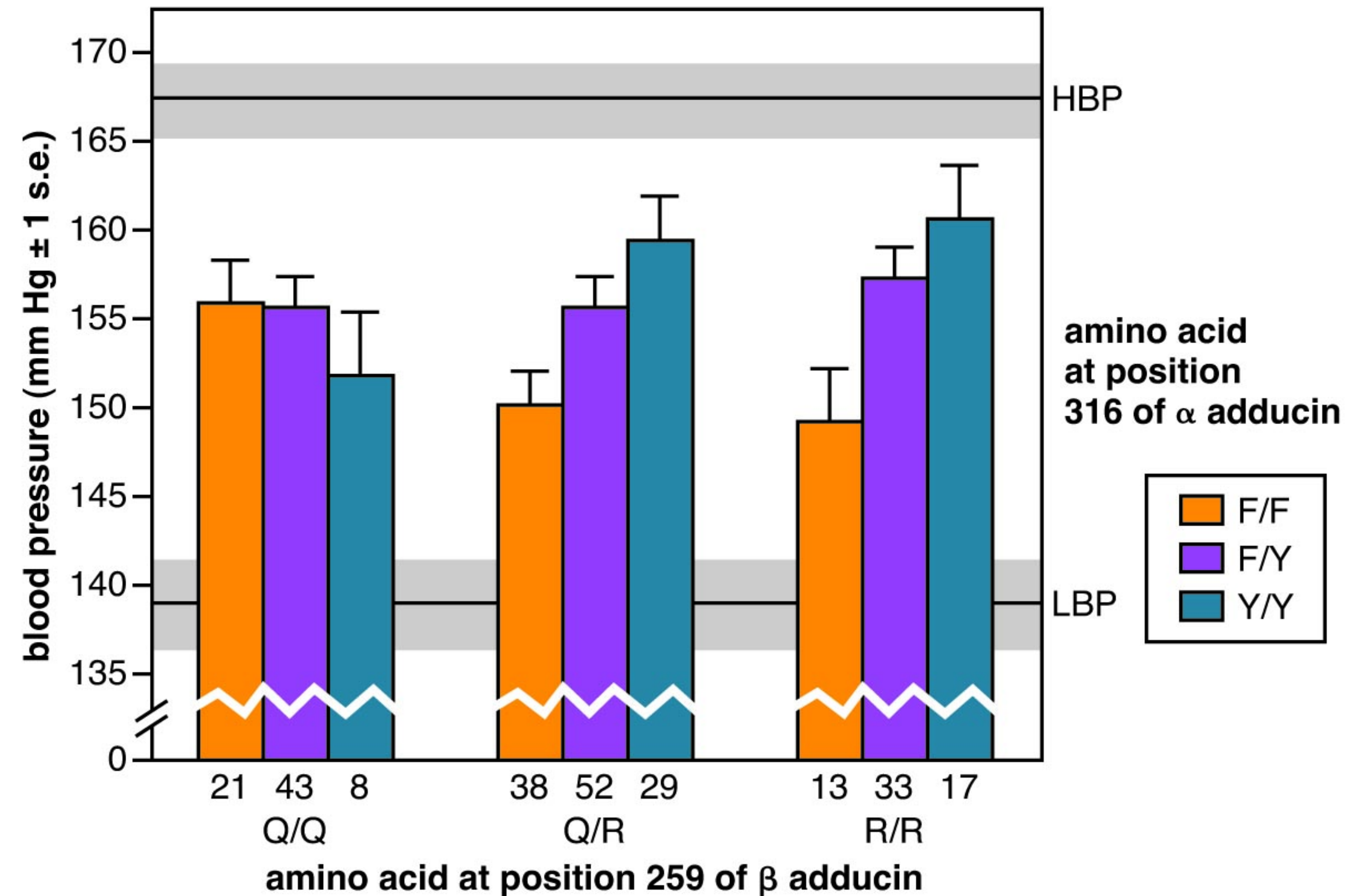


Figure 16.5

From Bianchi et al., 1994, Figure 3, copyright (1994) National Academy of Sciences, U.S.A.



BP of 9 combinations of two versions of the  $\alpha$  and  $\beta$  adducin genes in rats after two generations of breeding low and high blood pressure rats together.

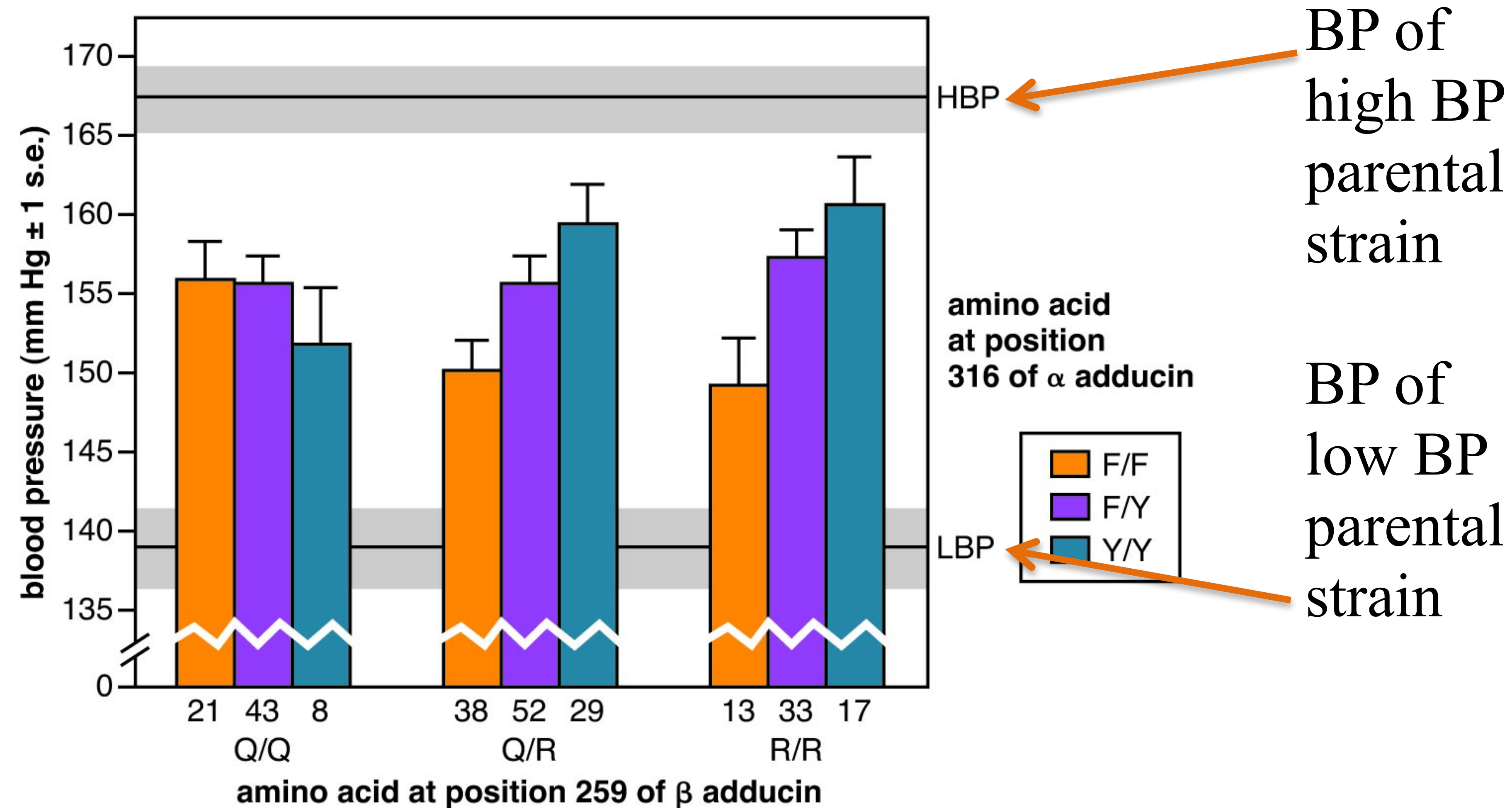


Figure 16.5

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## Two point mutations within the adducin genes are involved in blood pressure variation

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**ABSTRACT** The Milan hypertensive strain of rats (MHS) develops a genetic form of renal hypertension that, when compared to its normotensive control (MNS), shows renal dysfunction similar to that of a subset of human patients with primary hypertension. MHS and MNS were shown to be homozygous by multilocus minisatellite analysis and monolocus microsatellite markers. We show here that one point mutation in each of two genes coding for the membrane skeleton protein adducin is associated with blood pressure in the Milan strain of rats. Adducin is a heterodimer formed by  $\alpha$  and  $\beta$  subunits that promotes the assembly of actin with spectrin. MHS and MNS differ, respectively, by the amino acids Y and F at position 316 of the  $\alpha$  subunit. In the  $\beta$ -adducin locus, MHS is always homozygous for R at position 529 while in MNS either R or Q occurs in that position. The R/Q heterozygotes showed

seen in these rats have also been found in a subset ( $\approx 25\%$ ) of human patients with primary hypertension (10, 20–22). In the rat model, the difference in membrane ion transport disappeared after elimination of the membrane skeleton, which indicated the involvement of some of its components (23, 24). Cross-immunizations between MHS and MNS raised an antibody against a membrane skeleton protein subsequently identified as adducin (25). As this was the only cytoskeletal difference found that could be associated with membrane ion transport differences, adducin was considered a candidate for genetic studies in hypertension.

Adducin is an  $\alpha\beta$  heterodimer with subunits of  $M_r$  103,000 ( $\alpha$ ) and 97,000 ( $\beta$ ). It promotes the organization of a spectrin-actin lattice, a function regulated by phosphorylation and Ca-calmodulin interactions (26–28). Furthermore,  $\alpha$  and

...een a DNA polymorphism  
ested (3–8). The Milan hy-  
was developed by selection  
vergence to its normotensive  
ted for low blood pressure.  
breeding have been reached  
IS shows a greater pressor  
plantation (9, 11), a faster  
ular reabsorption (9, 12), a  
na renin activity (13), and a  
) and tubular cells (15), both  
sport across their plasma  
cyte functional differences  
in the stem cells and are  
rtension in F<sub>2</sub> hybrids (18).  
ally determined cellular de-  
t across the renal cell mem-  
bable cause of hypertension  
te and kidney dysfunctions

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## MATERIALS AND METHODS

**Animal Procurement and Housing.** All MNS and MHS rats were bred in our own facilities and maintained in conditions described elsewhere (9), in agreement with Directive 86/609/CEE of the Council of the European Community and Italian Law no. 116, 22/1/1992. The experimental F<sub>2</sub> population was produced as described in the text.

**Blood Pressure Measurements.** In the foundation colonies, selection for blood pressure levels was carried out each generation on awake animals restrained by wrapping lightly in a small cloth (33), using an indirect tail-cuff method. Measurements were made on a W + W BP recorder (Ugo Basile, Varese, Italy) with piezoelectric pickup.

For genetic analysis of the F<sub>2</sub> population, a cannula was inserted in the carotid artery of the rat under light halothane anesthesia and externalized at the back of the neck throughout a subcutaneous tunnel. The animals recovered within 3–5 min. Four hours later, the rats were connected by catheter to

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Abbreviations: MHS, Milan hypertensive strain; MNS, normoten-  
sive control.

†To whom reprint requests should be addressed.



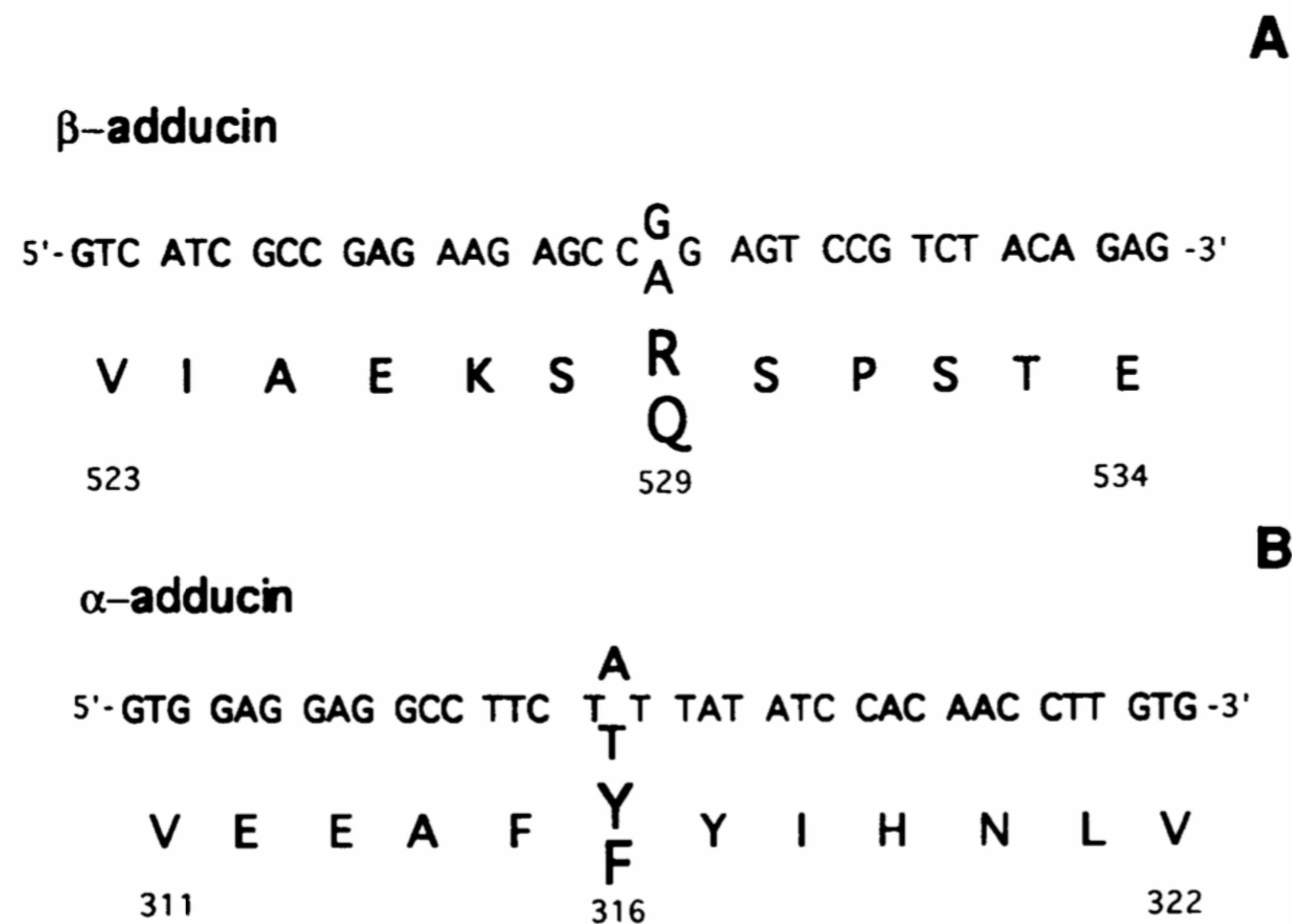


FIG. 1. Nucleotide and predicted amino acid sequences surrounding the mutation sites of adducin subunits in MHS and MNS rats. (A) Nucleotide sequence of  $\beta$ -adducin showed a G to A transition resulting in an arginine (MHS) to glutamine (MNS) substitution (R529Q). (B) Nucleotide sequence of  $\alpha$ -adducin showed an A to T transversion resulting in a tyrosine (MHS) to phenylalanine (MNS) substitution (Y316F).

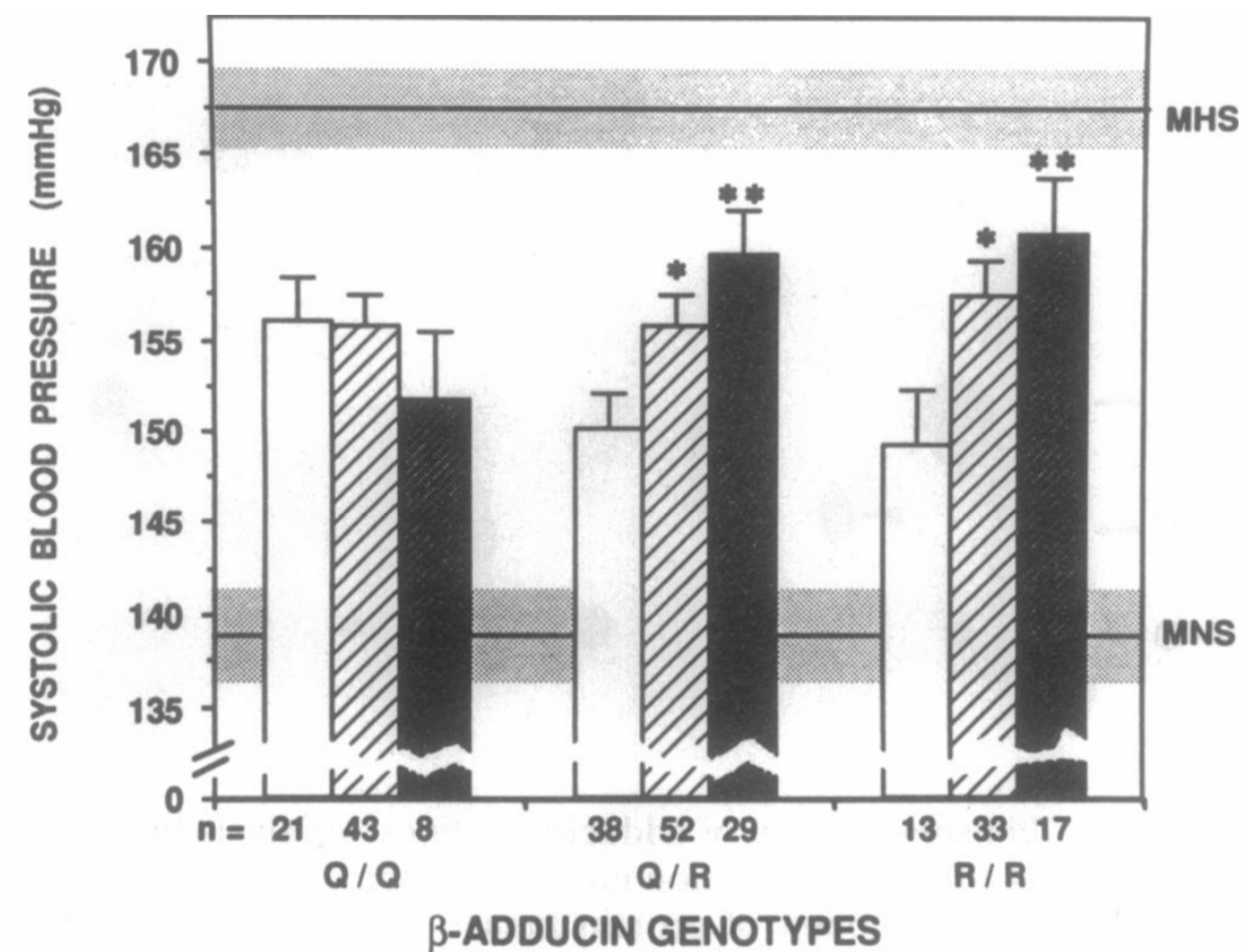
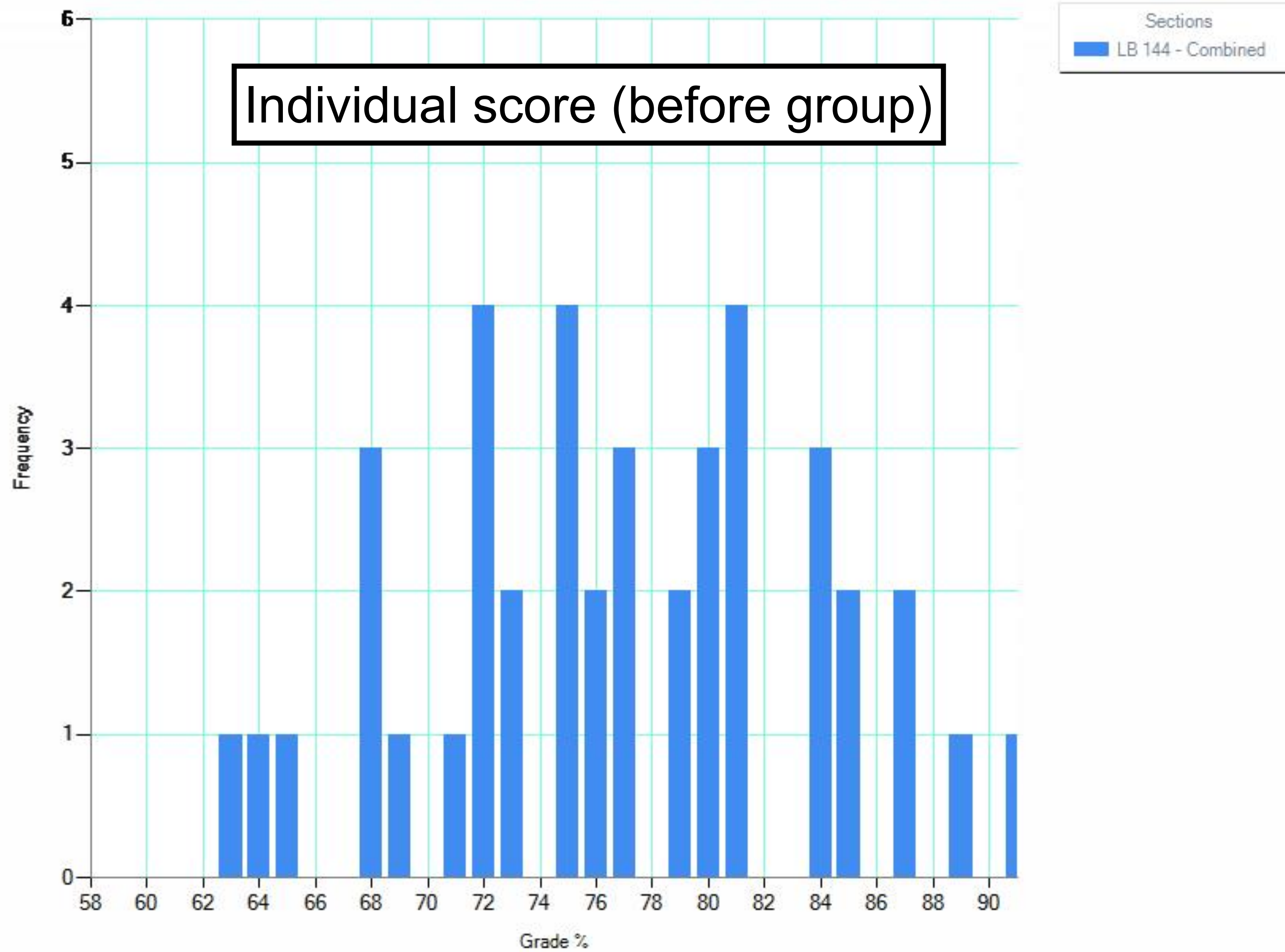


FIG. 3. Systolic blood pressure of  $F_2$  population in different adducin genotype cohorts ( $\alpha^F\beta^Q/\alpha^F\beta^Q$ , MNS genotype;  $\alpha^Y\beta^R/\alpha^Y\beta^R$ , MHS genotype). Systolic blood pressure (mmHg) was measured by a catheter inserted in the carotid artery. Results are expressed as means  $\pm$  SEM. *A posteriori* multiple comparison tests for a  $P$  value of 0.05 or better were performed. \*, Significantly different ( $P < 0.05$ ) from  $\alpha$ -adducin genotype  $F/F$ ; \*\*, significantly different ( $P < 0.01$ ) from  $\alpha$ -adducin genotype  $F/F$ . Mean systolic blood pressure (horizontal solid lines)  $\pm$  SEM (stippled areas) of 10 rats of each parental strain was also included for appropriate comparison. Open bars,  $\alpha$ -adducin genotype  $F/F$ ; light hatched bars,  $\alpha$ -adducin genotype  $F/Y$ ; dark hatched bars,  $\alpha$ -adducin genotype  $Y/Y$ .

## Announcements

1. **Exam I was a pyramid exam (individual then group)** enabled engaged review/revision, *multiple-True False WHY questions test depth of understanding*. Gained partial credit for anything correct.
2. Anyone who wishes to will be able to retake a newly fashioned Exam I again on Monday in E-26A Holmes Hall (no TARDIS pass required).
3. **Results were strong scores**. Average individual score (before group) was 77% which is high. Average Group bonus was +10% thus -> **87%!**
4. **Note CATME GEA#1 survey** has begun, reward people who did amazing things for your group. Note the box that says “what you say to your group mate will be seen by them” (means will be seen).
5. ReDo also available for “4-slide Proposal Talk” if want higher score. Group would give a “Progress Report Talk” in week 7 of semester. Must alert Luckie by Friday, 5pm if wish to do either *Talk* or *Exam* ReDo.







# TARDIS pass

ONE Time-Travel ReDo  
(must invoke within 7days of posted grade)





## Group Exam on Tuesday (last 30 minutes)

### Pyramid Exam

Now: Took the exam as a group  
(can increase your individual score by up to **20%**)

#### Rewards ->:

If **all** members **increased** their exam score -> **+5%**

If all members have group score **> 80%** -> **+5%**

If all members have group score **> 90%** -> **+5%**

If all members have group score **> Jillian\*** -> **+5%**

\*“Jillian” is a nickname for smartest student in a group

# LB144-Pandemic *2022 edition*

